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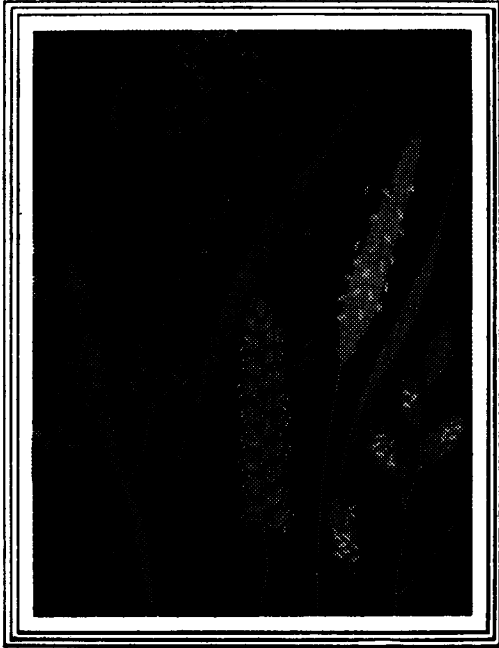
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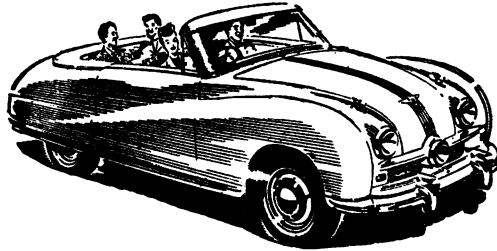
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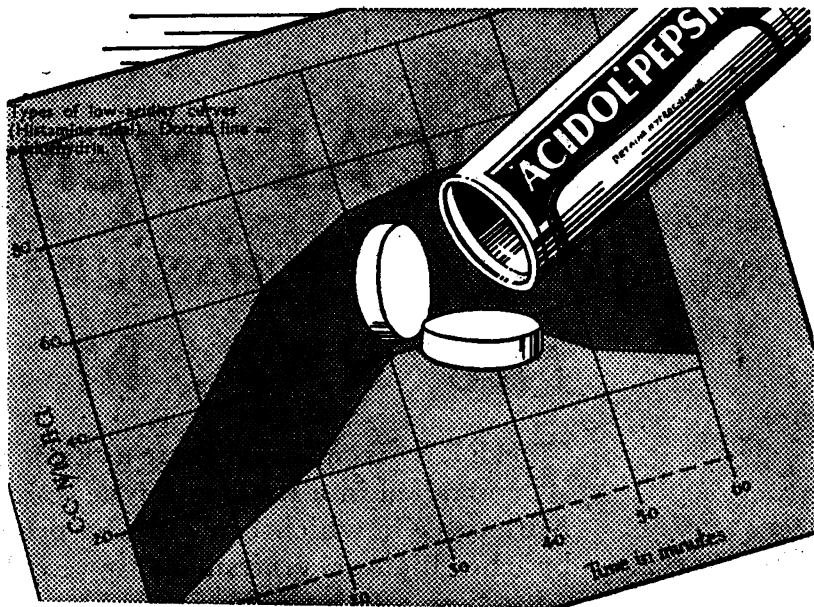
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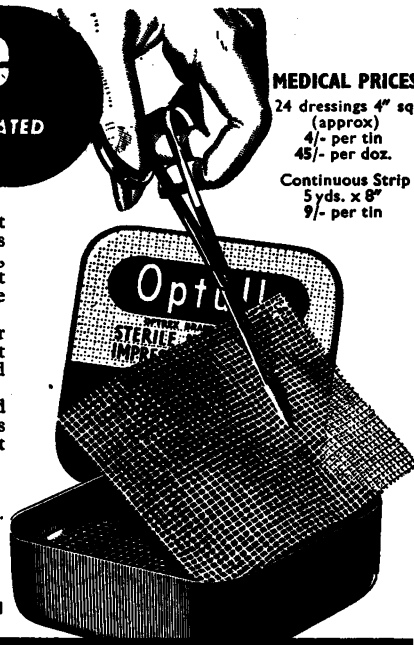
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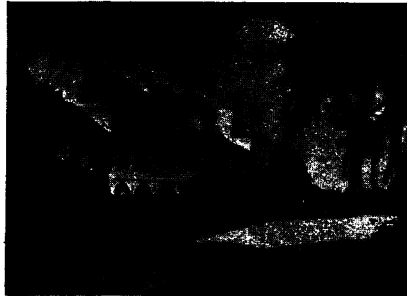
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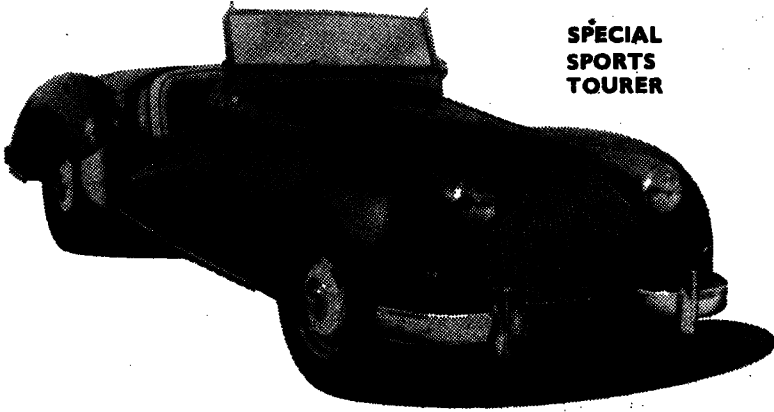
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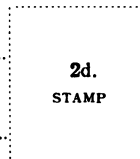
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# THE ULSTER MEDICAL JOURNAL

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## The Simulation of Heart Disease

By WILLIAM EVANS, M.D., D.SC., F.R.C.P.

*Abstract of an Address given to the Ulster Medical Society  
on 12th January, 1950*

THE imposition of unwarranted invalidism on healthy subjects, which results from the misinterpretation of physical signs, gives rise to a serious problem. In examination of the heart there are certain physical signs whose significance is obscure, and is, therefore, the more likely to lead to misguided interpretation. Surmise and conjecture, therefore, take the place of accurate observation; dogma is born without data; and fact gives way to fantasy. The heart itself cannot suffer from this miscarriage in diagnosis, but the damage to the patient's happiness and health is often irretrievable. Various classes of the population are likely to be the victims of such doctor-made illness.

(a) The discovery of an incidental or innocent murmur condemns thousands of children each year to unwarranted invalidism, whereby they are denied healthy games and happy pastimes; more seriously still, their education is all too frequently erratic and interrupted; and the provision of special transport, or even of special schools, is a source of personal embarrassment to the children and of financial embarrassment to the tax-payer.

(b) Similarly, many thousands among recruits for military service, able-bodied young men with healthy hearts, have been relegated to Grades 3 or 4, because they have shown such incidental and innocent cardiac murmurs.

(c) Young women with healthy hearts, showing some equivocal cardio-vascular sign, have been advised not to marry, or, if married, not to bear children, and all for utterly insufficient reasons.

(d) Candidates for life insurance are often rejected or made to pay unnecessarily high premiums in cases in which the cardio-vascular system is not the subject of disease.

(e) Elderly subjects, with a trivial murmur or a chest pain of doubtful

significance, may be compulsorily and unnecessarily retired, this unwarranted invalidism being peculiarly cruel in the autumn of life.

*Effort Syndrome.*—The terminology of this condition is extravagant both in the number and variety of its names. The American term “neuro-circulatory asthenia” has at least the merit of drawing attention away from the heart. To call it by its proper name—neurosis—would be better still, in that it might encourage the patient to stimulate his resolve to overcome his symptoms. The exercise tolerance test is a worthless method of assessing the heart, and is no yardstick whereby to measure the capacity of the heart to fulfil its function, so we should forego it.

*Angina.*—The paucity of physical signs of coronary artery disease demands more than ordinary care in the interpretation of the patient’s history, and his own description of the character of the pain is of the utmost importance. Both mannerism and aphorism, therefore, must be closely observed as one elicits the story of the pain. Lancing, shooting, and darting pains are frequently described with verve and picturesque phrase, and are never caused by true angina. Even pain in the middle of the chest and related to exertion is as often caused by some trivial complaint like flatulent dyspepsia as by coronary artery disease. It is, therefore, important that the physical signs of coronary disease must be carefully sought. These include the proper assessment of blood pressure, the recognition of cardiac enlargement, of aortic incompetence, and of triple rhythm as a sign of heart failure. Changes in the electrocardiogram require expert interpretation. The electrocardiograph is a valuable instrument in the diagnosis of cardio-vascular disorders, but, before acquiring one, we must first know how to read the record which it traces. Passing the “driving test” is a prerequisite to using the automobile. A “decipher test” should be a preliminary condition to possessing an electrocardiograph.

*Hypertension.*—Many patients tell their doctors that they have blood pressure: of these, perhaps one-half have hypertension. Hypertonia is a physiological state, to be distinguished from hypertension, which is a disease-producing condition. Hypertension is present when the systolic blood pressure is 180 millimetres of mercury or over, and/or the diastolic blood pressure is 110 millimetres or over on three consecutive occasions, and when there is present clinical, cardiographic, and cardioscopic evidence of cardio-vascular hypertrophy. Headache, tinnitus, giddiness, loss of concentration, palpitation, and pain under the left breast are characteristic complaints of the neurotic. The symptoms of hypertension, on the other hand, are substantial ones, like paroxysmal dyspnoea from left ventricular failure, cardiac pain from ischæmia and infarction, loss of vision from retinal changes, and locomotor disturbances from cerebral arterial rupture.

*Cardiac Enlargement.*—A statement that the heart is enlarged should not be given lightly, and never with the false reassurance that “there is nothing serious; the heart is only slightly enlarged.” Displacement of the apex beat is due to enlargement of the heart in only the minority of cases. Causes of displacement of an innocent kind include a shallow thorax, scoliosis, and a depressed sternum or other thoracic asymmetry.

*Heart Sounds and Murmurs.*—It is from heart sounds and murmurs that

unwarranted cardiac invalidism springs most readily, and the misinterpretation of certain auscultatory signs is a common fault in clinical medicine. For example, a split first sound which may occur in a healthy subject, may be mistaken for the pre-systolic murmur of mitral stenosis, and this error may be reinforced by finding accentuation of the second sound in the pulmonary area, although this is, indeed, of no value as a supporting sign of heart disease, seeing that it is so common a finding in health.

Three main clinical varieties of innocent murmur heard near the mitral area may be segregated by considering the character of the murmur, the effects upon it of deep inspiration and posture, and its place in systole.

(a) The innocent systolic murmur of reclining posture is blowing in character and is not loud, so that it often disappears on deep inbreathing. It is louder in the reclining posture when a similar murmur appears in the pulmonary area. It is placed in mid-systole. It is common in children and in young subjects; uncommon after thirty years; and is never met with after forty years of age.

(b) The innocent parasternal murmur is blowing or whiffy, and it is loud enough to persist on deep inbreathing. It is loudest in the fourth intercostal space at the left border of the sternum, but, unlike the murmur of ventricular septal defect, it is never accompanied by a thrill. It is placed in mid-systole. Although commoner in young people, it is often met with in older subjects.

(c) The innocent murmur in late systole is blowing in character and is loud, so that it persists during deep inbreathing. It occurs at all ages. The murmur is nearer to the second than the first heart sound, and, for this reason, it is easily recognised on clinical auscultation.

The innocent systolic murmur in the pulmonary area assumes importance because it is commonly mistaken for the murmur of pulmonary stenosis, which it closely resembles. It can be as loud as the murmur of pulmonary stenosis, but it is less loud in the upright posture, especially if deep inbreathing is an added manœuvre. The murmur is placed in mid-systole and is not associated with a thrill.

To allow a diagnosis of *mitral incompetence* to stand as our interpretation of a mitral systolic murmur is to invite an inaccurate diagnosis at any time; while to apply it to the murmurs just described is to commit a wrong limitless in its injurious effects. It is because of the complacency engendered by this mischievous diagnosis of mitral incompetence that we must quickly forego the term; as long as it is allowed to stay, so long will it prove a disservice to medicine and a travesty of diagnosis by clinical auscultation.

*Past History of Rheumatic Fever.*—A child with a heart murmur, even though of the innocent kind, easily falls victim to unwarranted invalidism, but when it becomes known that the child has suffered at one time from frank rheumatic fever or even a painful joint, its escape from such enforced invalidism is very unlikely. Experience in the examination of school children has so convinced me of the malinfluence which a past history of rheumatic fever exerts in a case in which a child with a murmur is presented to a doctor for diagnosis that I am led to make this serious proposal, that we must arrive at a diagnosis of the presence or absence

of valvular heart disease from a regard of the clinical findings by themselves, and without any knowledge of the past history of rheumatic fever. We must have confidence in our objective physical signs.

What has been described here does not tell of startling discoveries in medicine, but it does invite us to think seriously of the several ways in which our recognition of the sham signs of disease has failed in the past. A sober reflection on the poignant ills associated with feigned heart disease should compel us to care for the proper interpretation of the physical signs which have been mentioned, and which simulate those found in heart disease. While we continue to insist on healing the afflicted, let us desist from afflicting the healthy. We should not turn from this firm resolve.

I suggest that adherence to "Ten Commandments" might keep us from committing, within the speciality of cardiology, the blackest of all sins, namely, the sin of subjecting a healthy individual to a life of unwarranted cardiac invalidism. They are:

1. Avoid fanciful diagnosis like "athlete's heart," "tired heart," etc.
2. Do not lightly diagnose cardiac pain in the presence of a normal electrocardiogram.
3. Know how to read the electrocardiogram before acquiring an electrocardiograph.
4. Resist a diagnosis of "high blood pressure" in the absence of cardio-vascular hypertrophy.
5. Make certain that a shifted apex beat is not the outcome of simple cardiac displacement, before attributing it to cardiac enlargement.
6. Don't mistake a "thrill" for a thrill.
7. Don't mistake a healthy splitting of the first heart sound for a pre-systolic murmur.
8. Know the marks of an innocent murmur.
9. Never seek a past history of rheumatic fever before deciding on the diagnosis obtained from the physical signs.
10. Relinquish the term "mitral incompetence" in diagnosis.

## REVIEW

DISEASES OF THE EYE. By May and Worth. Pp. 548. Baillière, Tindall & Cox. 1949. 22s. 6d.

It is always a difficult matter to provide a text-book on a special subject for students and general practitioners—to be comprehensive is to be too elaborate—to be concise is to omit essentials. This book, which has long been popular, now brought up to date avoids both pitfalls.

It contains all the purely eye diseases which students should know, and which general practitioners may meet, set out in a manner which they can assess the points of diagnosis and treatment and, above all, contains a number of plates and photographs which probably mean more than the written word to students.

My only criticism is that more space has not been devoted to Medical Ophthalmology to bring home even more emphatically that Ophthalmology is not purely a local subject but has wide bearings on diseases apparently remote from the eye.

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S. R. S.



# The Problem of the Aged in Mental Hospitals

By C. B. ROBINSON, M.D., F.R.C.P.I., D.P.M.,

Purdysburn Hospital, Saintfield Road, Belfast

THE problem of the aged in Mental Hospitals is universal, and is becoming more acute as time goes by. Mental disease is a tragedy at any time of life, but coming on in the later years it presents a particularly saddening picture. The majority of these patients have led a busy and useful existence, and those who were once the props and pillars of their age and generation are now a burden on the backs of their younger brethren. The aged patient is considerably different from the average inmate of a mental hospital. No other type of patient, with the exception of the acutely ill, requires so prolonged and continuous supervision. Uncontrollable factors, such as incontinence, confusion, and memory impairment, make it impossible for the aged to get along without considerable physical help from the nursing staff. Occupational therapy is limited, especially for those suffering from senile and arteriosclerotic dementia, and the useful output from such a department is so small that it presents a serious drain on the resources of the mental hospital. Looking after such patients constitutes the most monotonous and least glamorous type of nursing, and a year of care of the aged patient is probably equivalent to at least two years care of any other—the acutely mentally ill excepted.

This communication deals briefly with patients of 60 years and over in the six mental hospitals of Northern Ireland in general, and Purdysburn Hospital in particular. The basic age of 60 years rather than 65 years was taken to conform with the statistical requirements of the Geriatric Service. To give some idea of the amount of accommodation required for these patients, the following table gives the return from each of the mental hospitals.

TABLE I

|              | Total No.<br>of Patients | 60 Years<br>and Over | Percentage  | Number Fit<br>for Transfer | Percentage<br>of Over<br>Sixties |
|--------------|--------------------------|----------------------|-------------|----------------------------|----------------------------------|
| Antrim       | 694                      | 236                  | 34          | 99                         | 42                               |
| Armagh       | 562                      | 223                  | 39.7        | 82                         | 36.8                             |
| Down         | 909                      | 305                  | 33.5        | 81                         | 26.5                             |
| Londonderry  | 570                      | 182                  | 31.9        | 96                         | 52.7                             |
| Omagh        | 839                      | 302                  | 36          | 106                        | 35.1                             |
| Purdysburn   | 1563                     | 468                  | 30          | 154                        | 32.9                             |
| <b>TOTAL</b> | <b>5137</b>              | <b>1716</b>          | <b>33.4</b> | <b>618</b>                 | <b>36</b>                        |

It was of interest to find in this survey that if alternative accommodation could be obtained, either in a psychiatric long-stay annexe with simple supervision, resident homes, frail ambulant long-stay annexe, or bed-fast long-stay annexe, 618 patients

in all could be discharged from these six mental hospitals, thereby reducing the number to 998, or 24.3 per cent. of the total resident population.

TABLE II

|              | Total Number in Residence |      |         |      | Sixty Years and Over |      |         |      |
|--------------|---------------------------|------|---------|------|----------------------|------|---------|------|
|              | Males                     | %    | Females | %    | Males                | %    | Females | %    |
| Antrim - -   | 369                       | 53.2 | 325     | 46.8 | 100                  | 42.4 | 136     | 57.6 |
| Armagh - -   | 300                       | 53.4 | 262     | 46.6 | 106                  | 47.5 | 117     | 52.5 |
| Down - -     | 456                       | 50.0 | 453     | 50.0 | 134                  | 43.9 | 171     | 56.1 |
| Derry - -    | 335                       | 58.8 | 235     | 41.2 | 85                   | 46.7 | 97      | 53.3 |
| Omagh - -    | 466                       | 55.5 | 373     | 44.5 | 172                  | 57.0 | 130     | 43.0 |
| Purdysburn - | 829                       | 53.0 | 734     | 47.0 | 204                  | 43.6 | 264     | 56.4 |
| TOTAL        | 2755                      | 53.6 | 2382    | 46.4 | 801                  | 46.7 | 915     | 53.4 |

In all the hospitals in Northern Ireland the total number of male patients exceeds the number of female, the proportion being 53.6 per cent. males to 46.4 per cent. females. Despite this over-all male superiority the scene changes when only those over 60 years are considered—it is then found that the females outnumber the males, the proportion being 46.7 per cent. males to 53.4 per cent. females. In Omagh mental hospital only do the male patients keep a majority, the proportion being 57 per cent. males to 43 per cent. females.

TABLE III—PURDYSBURN HOSPITAL

|            | Total Number in Residence |         |       | Sixty Years and Over |      |         |      | Total | % of Total Pop. |
|------------|---------------------------|---------|-------|----------------------|------|---------|------|-------|-----------------|
|            | Males                     | Females | Total | Males                | %    | Females | %    |       |                 |
| 1/ 1/38 -  | 910                       | 779     | 1689  | 146                  | 38.4 | 234     | 61.6 | 380   | 22.5            |
| 1/ 1/39 -  | 926                       | 815     | 1741  | 169                  | 40.0 | 251     | 60.0 | 420   | 24.1            |
| 1/ 1/40 -  | 928                       | 794     | 1722  | 156                  | 39.0 | 244     | 61.0 | 400   | 23.2            |
| 1/ 1/41 -  | 913                       | 772     | 1685  | 170                  | 41.6 | 239     | 58.4 | 409   | 24.3            |
| *          |                           |         |       |                      |      |         |      |       |                 |
| 1/ 1/46 -  | 802                       | 680     | 1482  | 155                  | 40.3 | 230     | 59.7 | 385   | 26.0            |
| 1/ 1/47 -  | 802                       | 681     | 1483  | 161                  | 41.1 | 231     | 58.9 | 392   | 26.4            |
| 1/ 1/48 -  | 817                       | 684     | 1501  | 164                  | 40.0 | 246     | 60.0 | 410   | 27.3            |
| 1/ 1/49 -  | 836                       | 713     | 1549  | 184                  | 41.7 | 257     | 58.3 | 441   | 28.5            |
| 15/10/49 - | 845                       | 748     | 1593  | 195                  | 41.2 | 278     | 58.8 | 473   | 29.8            |

\* Figures for 1942-45, inclusive, are not given, as over 500 patients of all ages were temporarily transferred to other hospitals during these war years.

The number of older patients in mental hospitals is rising and, unfortunately, is rising quickly. On 1st January, 1938, the proportion of the over sixties to the total number of patients in this hospital was approximately 22.5 per cent. On 1st January, 1941—24.3 per cent., 1st January, 1946—26 per cent., and 1st January, 1949—28.5 per cent. There has been a further increase since the beginning of this year, and the figure now stands at 29.8 per cent., despite a rise of 44 in the total number of patients. If consideration is given to the actual number of over sixties, it will be

found that on 1st January, 1938, there were 380. This figure remained fairly constant up to 1st January, 1947, when there were 392, or an increase of 12 in 9 years, despite a drop in the total number of patients, but since then there has been a sharper rise, the total on the 1st January, 1949 being 441, an increase of 49 in 2 years, and at the present time is 473. It is interesting to note that the proportion of males to females over 60 in this hospital has remained almost constant since 1939, the proportion being at present 41.2 per cent. males to 58.8 per cent. females. This is certainly not the findings of some of the American hospitals who note an increase in the proportion of females. The outlook is not promising, and the time may come shortly when the chronic schizophrenics will lose their long-held honoured position as the biggest single group in the mental hospitals.

Various factors, mainly beyond control, have combined to bring this about. During the present century there has been a steady rise in the number of persons over sixty years of age, and this is still increasing. In Great Britain in the three years 1944-47 the number rose from 6,300,000 to 7,400,000, an increase of over one million. At the beginning of the century the ratio of over sixties to the total population was 1 to 18, but in 1947 it was 1 to 5.5. The figure for the Northern Ireland Mental Hospitals at present is 1 to 3, but if the Geriatric Service could find accommodation for the 618 individuals already mentioned who are fit for discharge to their care, the figure would become 1 to 4.7.

The advance of medical science and the progress of preventive medicine have played a large part in reducing the havoc wrought by physical disease in later life, but this has left the way open for an increase in the mental disorders of old age. As repair of physical injury is slowed down by advancing years, so also is recovery from mental breakdown retarded, especially where organic illnesses, such as arteriosclerosis, have an influence on the condition. Two thousand years ago the average span of life was 25 years, in 1900 it was 49, but to-day it is 66.5.

The mobilization of women during the war years for the services and for work in the factories has greatly reduced the number available to look after the aged sick as, having once tasted independence, they are loath to return to their homes to care for aged relatives. Families are smaller, and it is becoming more difficult to find the dutiful daughter, who, willingly or unwillingly, was expected to stay at home to look after the parents from their retirement from active work until their death, anytime within the next twenty to thirty years. Through pension schemes, many old people have become more or less financially independent and live alone, so that when they fall ill, either mentally or physically, they require to be nursed in hospital. Domestic and nursing help have been more difficult to find of recent years, at least at reasonable prices, and difficulties in housing conditions, as well as the lessening of filial responsibility, have all played their part in adding to the problem.

A comprehensive health service which is to look after everyone from the cradle to the grave has provided an excuse for those who wish to shelve any responsibility in this matter, and who expect the State to step in and provide the necessary accommodation and care. In addition to a more enlightened public attitude towards earlier use of the mental hospitals, many other factors have played their part, but

the net result has been to put an unprecedented demand for care and treatment of the old people on the existing hospitals and institutions.

The general hospitals in their turn, have their own problems, especially since July 1948, and they are extremely reluctant to receive any but acute cases whose stay in their beds will be of short duration.

This is the age of specialisation, and it has been carried further than could have been foreseen many years ago. As well as general hospitals there are now chest, fever, children's, neuro-surgical, and orthopædic hospitals, to name but a few, and each of these has no place in it for anyone who falls into a different category. The medical wards of the general hospitals have no place in them for the aged chronic sick, so again specialisation has been resorted to, and the geriatric service has come into being. Here the new broom is desirous of making a very clean sweep, and any long-term sick patient who shows outward manifestation of being a little odd, of being troublesome at times, or even shows mild resentment at being firmly handled by the nurses, is promptly despatched to the nearest mental hospital. Some Poor Law Institutions have been converted into hospitals by a stroke of the pen, and are now anxious to shift all patients to mental hospitals who show signs of mental derangement, irrespective of the bodily condition from which they may be suffering—this factor is causing intense concern to some of the county mental hospitals in particular. The result has been to increase the numbers of the over sixties in the mental hospitals during the past year. It is interesting to point out, in passing, that in 1941, owing to the great upheaval caused by the air raids on Belfast, 156 over sixties were admitted to Purdysburn as against 114 for 1940 and 79 for 1942. In 1947, 168 were admitted, 203 in 1948, and 117 *in the first six months alone* of 1949. It has been our experience that the female admissions over sixty have always outnumbered the males, sometimes by as much as 2 : 1. In some places in England and America the contrary has been found. A survey was carried out in this hospital to see the fate of those patients of sixty and over.

TABLE IV—PURDYSBURN HOSPITAL

|      |   | Admissions |     | Discharged |     | Death within |     | Death within |     | Remaining      |
|------|---|------------|-----|------------|-----|--------------|-----|--------------|-----|----------------|
|      |   | over Sixty |     |            |     | 6 Months     |     | 1 Month      |     | After 6 Months |
| 1928 | - | 111        | ... | 24         | ... | 40           | ... | 16           | ... | 47             |
| 1933 | - | 148        | ... | 58         | ... | 61           | ... | 29           | ... | 29             |
| 1938 | - | 143        | ... | 53         | ... | 50           | ... | 25           | ... | 40             |
| 1943 | - | 74         | ... | 27         | ... | 16           | ... | 6            | ... | 31             |
| 1948 | - | 203        | ... | 64         | ... | 71           | ... | 38           | ... | 68             |
| 1949 | - | 117        | ... | 31         | ... | 42           | ... | 30           | ... | 44             |

(First six months only)

Some of these patients lived for a few days only, and in one case this year death occurred within 12 hours of admission. In the majority of cases of early death the underlying cause was arteriosclerosis. After studying the case records of these patients, one is forced to the conclusion that some of them could have been dealt with best by being allowed to remain in the hospital from which they came, rather than transfer them to die in a mental hospital.

TABLE V—PURDYSBURN HOSPITAL

|              | Admitted<br>when Sixty + | Arterio-<br>sclerosis | Senile     | Other     | Long-standing<br>Mental Disease | Total      |
|--------------|--------------------------|-----------------------|------------|-----------|---------------------------------|------------|
| Males -      | 57 ...                   | 13 ...                | 26 ...     | 18 ...    | 138 ...                         | 195        |
| Females -    | 128 ...                  | 27 ...                | 76 ...     | 25 ...    | 150 ...                         | 278        |
| <b>TOTAL</b> | <b>185</b>               | <b>40</b>             | <b>102</b> | <b>43</b> | <b>288</b>                      | <b>473</b> |

Of the 473 (195 males, 278 females) patients at present resident in this hospital 288 are cases of long standing mental disease and of the remaining 185 admitted at the age of sixty or over 21.6 per cent. are arteriosclerotics, 55.1 per cent. are seniles, and 23.3 per cent. are other types of mental disorder.

The age distribution of the 473 patients is as follows :—

TABLE VI—PURDYSBURN HOSPITAL

|             | 60-64  | 65-69  | 70-74  | 75-79  | 80-84  | 85 + |
|-------------|--------|--------|--------|--------|--------|------|
| Males -     | 57 ... | 50 ... | 52 ... | 22 ... | 9 ...  | 5    |
| (Per Cent.) | 29.2   | 25.7   | 26.6   | 11.3   | 4.6    | 2.6  |
| Females -   | 67 ... | 54 ... | 67 ... | 45 ... | 30 ... | 15   |
| (Per Cent.) | 24.0   | 19.1   | 24.1   | 16.3   | 10.9   | 5.5  |

It is obvious from these figures that the average mean age of the females over 60 is much greater than that of the males.

The use of penicillin and the sulphonamides has helped to prolong the lives of many of these patients who would otherwise have succumbed to acute lung conditions, and it is obvious that this will give rise to the need of more nursing care from an already overworked and understaffed personnel.

What then can be done to help towards a solution to this grave problem? The exclusion of the aged from the medical wards of the teaching hospitals has resulted in a gross defect in the training of both doctors and nurses. In general practice the care of such patients constitutes a considerable proportion of the work so that newly qualified doctors have seen little or nothing of the special problems which arise in dealing with these patients, and, consequently, the only solution they have to offer is to call for help from the geriatric service. Here again, the doctors and nurses require training in mental abnormalities which may occur in the aged. Unless such training is given the eccentricities and behaviour disorders of older people are taken far too seriously, as they are not properly understood and no allowance is made for them, with the inevitable result that there will be confinement in a mental hospital for the majority.

It would be retrograde in these days to suggest that admission to mental hospitals should be made more difficult, but, in the case of the over sixties, with the exception of the acutely mentally ill, some screening appears to be necessary. This could be brought about by greater co-operation between psychiatrists and physicians in charge of the geriatric wards, when each case should be approached as a problem to be worked out together with equal responsibility. At present the number of

psychiatrists available is not adequate for much time to be given in this direction, but when this defect has been remedied, it should be part of the extra-mural duties of the local mental hospital staff to give their advice in the border-line cases, thereby establishing a much-needed liaison between the mental and general hospitals.

A suggestion has been made that a geriatric unit should be established at the mental hospital, as there already exists in each hospital a great number of these unfortunates who, for the most part, lead a vegetative existence. There is no doubt that such a unit, comprising occupational therapy and specialised services for the aged, would be of great benefit, but there are many factors against such a solution. Economics, difficulty in staffing, and, above all, the situation of the unit, would be immense handicaps. It has been found that psychiatric out-patient clinics are more successful when run in conjunction with general hospitals, and the same factors apply in this case.

Another suggestion has been the establishment of a special hospital, with out-patient clinic and social services, where all patients of 60 and over would be admitted when ill. After admission, disposal would be made to the medical, surgical, geriatric or psychiatric divisions with full co-operation and facility of transfer between the various units. The psychiatric division would be under the control of the local mental hospital, whose staff would be responsible for running it. One advantage of this would be that it would give rise to easy inter-change of patients from the mental hospital who no longer needed such control, and from this special hospital disposal could be made to a suitable position in the outside world without the stigma of the mental hospital resting upon them.

In conclusion, I would like to emphasise the following points :—

1. This is an urgent problem requiring solution at an early date to avoid the mental hospitals becoming the dumping ground for any of the aged chronic sick who are not quite up to standard from a mental point of view.
2. Nothing much can be done for the apathetic type of senile already in the mental hospital, in view of overcrowding, shortage of staff and lack of equipment— for this type of patient the solution appears to be the reduction of further admissions by the provision of some organisation prepared to take an interest in these old people.
3. The setting up of a geriatric unit away from the mental hospital is probably the best way of dealing with new cases, where the psychiatric division would be under the supervision of the local mental hospital medical staff.
4. The more efficient screening of the chronic sick showing evidence of mental disorder seems necessary, and this could be brought about by more co-operation between the psychiatrists and the geriatric service.
5. In view of the short duration of life of some of the patients admitted from general hospitals, a few beds should be available there for treatment of cases who break down owing to their physical illness.
6. The Mental Health Service has long been regarded as the Cinderella of medicine, but the time has now come for it to take a lead in dealing with this problem. This can only be done by active co-operation with our colleagues in

other branches of medicine. We can no longer afford to lead an isolated existence, but must recognise that there is as great a job to be done extramurally, by publicity and by action, as well as within the walls of the mental hospitals.

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## REVIEWS

### THE POCKET PRESCRIBER AND GUIDE TO PRESCRIPTION WRITING.

By D. M. Macdonald, M.D., D.P.H., F.R.C.P.E. Revised by A. G. Cruikshank, F.R.C.P.E. Pp. 275. Edinburgh : E. & S. Livingstone Ltd. 4s. 6d.

THIS is one of the most useful little reference books that the G.P. could carry with him. It is truly pocket size, measuring  $4\frac{1}{8}$  inches by  $2\frac{1}{8}$  inches.

The fourteenth edition of this book, which was first published in 1882, is brought well up to date with information on penicillin, sulphonamides, thiouracil, and proprietary medicines, as well as many of the old, well-tried prescriptions. The book is eminently practical and gives concise alternative treatments for the different diseases under separate indexed headings, e.g., Alimentary, Respiratory, Blood, etc. There are also notes on prescription writing, dosage tables (adults and children), diets, average heights and weights, incubation periods, D.D.A. regulations, etc.

The Pocket Prescriber should be of service to the recently qualified doctor, as well as a memory refresher to the older doctor.

R. G. K.

### A PSYCHIATRIST LOOKS AT TUBERCULOSIS. By Eric Wittkower, M.D.

The National Association for the Prevention of Tuberculosis, Tavistock Square, London, W.C.1.

IN this well-written book the author gives a psychopathological hypothesis of tuberculosis and a description of the various reactions between the illness and the different types of personalities.

Every chronic condition constitutes a psychological problem for the patient, and we may as well admit, for the doctor too. The physical factors, like the toxæmia, which lowers the threshold of inhibition, and the lack of muscular activity, which leads to tension and anxiety, are mentioned and very well described.

A need for affection as an outstanding common feature of the premorbid personality of the tuberculous patient is demonstrated in this book.

External events as precipitating or "reactivating" factors are discussed.

The masterly description of the psychological state of a tuberculous patient in Thomas Mann's "Magic Mountain" is not mentioned.

Before concluding the review of this very interesting and readable book, the reviewer wants to quote another writer, G. B. Shaw, who says in "Doctor's Dilemma," "Now-a-days the troubles of consumptive patients are greatly increased by the growing disposition to treat them as lepers."

K. S.

other branches of medicine. We can no longer afford to lead an isolated existence, but must recognise that there is as great a job to be done extramurally, by publicity and by action, as well as within the walls of the mental hospitals.

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### THE POCKET PRESCRIBER AND GUIDE TO PRESCRIPTION WRITING.

By D. M. Macdonald, M.D., D.P.H., F.R.C.P.E. Revised by A. G. Cruikshank, F.R.C.P.E. Pp. 275. Edinburgh : E. & S. Livingstone Ltd. 4s. 6d.

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The fourteenth edition of this book, which was first published in 1882, is brought well up to date with information on penicillin, sulphonamides, thiouracil, and proprietary medicines, as well as many of the old, well-tried prescriptions. The book is eminently practical and gives concise alternative treatments for the different diseases under separate indexed headings, e.g., Alimentary, Respiratory, Blood, etc. There are also notes on prescription writing, dosage tables (adults and children), diets, average heights and weights, incubation periods, D.D.A. regulations, etc.

The Pocket Prescriber should be of service to the recently qualified doctor, as well as a memory refresher to the older doctor.

R. G. K.

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The National Association for the Prevention of Tuberculosis, Tavistock Square, London, W.C.1.

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Every chronic condition constitutes a psychological problem for the patient, and we may as well admit, for the doctor too. The physical factors, like the toxæmia, which lowers the threshold of inhibition, and the lack of muscular activity, which leads to tension and anxiety, are mentioned and very well described.

A need for affection as an outstanding common feature of the premorbid personality of the tuberculous patient is demonstrated in this book.

External events as precipitating or "reactivating" factors are discussed.

The masterly description of the psychological state of a tuberculous patient in Thomas Mann's "Magic Mountain" is not mentioned.

Before concluding the review of this very interesting and readable book, the reviewer wants to quote another writer, G. B. Shaw, who says in "Doctor's Dilemma," "Now-a-days the troubles of consumptive patients are greatly increased by the growing disposition to treat them as lepers."

K. S.



other branches of medicine. We can no longer afford to lead an isolated existence, but must recognise that there is as great a job to be done extramurally, by publicity and by action, as well as within the walls of the mental hospitals.

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## REVIEWS

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K. S.

# The Obstetrical Aspects of Tristram Shandy

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

*Paper read at the request of Professor F. W. Baxter to the  
Honours English Class at the Queen's University.*

My terms of reference, according to Professor Baxter's letter, are "the obstetrical aspects and any others you choose." The last four words are my excuse for copying Laurence Sterne and digressing at the very beginning from "Tristram Shandy" to consider for a brief period the author himself from a medical aspect.

Powys, in an introduction to one edition of "Tristram Shandy," writes: "It would be interesting to get hold of some sophisticated young writer, of the present generation of either sex, though perhaps preferably male, some writer, I mean, who has never in his life opened a volume of Sterne, and who is shamelessly free from all literary snobbishness, and to persuade such an one to record his precise and exact reactions to his first reading of 'Tristram Shandy.'"

I had read this book so long ago that I had forgotten all the details and the occasion of this lecture forced me to refresh my memory. Therefore I feel that I can, from a medical aspect, express some of my reactions to what might be described as my first intelligent reading of the book.

My first thought was that no man, who had not steeped himself to the full in the vices of the period, could have written the book. Yet compared with a modern novel, such as "East Side, West Side," which has a pornographic flavour, the plain speaking of "Tristram Shandy" does not leave the same bad taste.

My second reaction, following the reading of the first four chapters of Volume I, was that he must have been unhappily married. To a gynaecologist it is obvious in these chapters that the relations between Mr. and Mrs. Shandy were those of an oversexed husband with a frigid wife. Such chapters could only have been written by a man with personal experience.

His knowledge and description of obstetric problems suggests that he must have had close contact with some obstetrician of the period. Doran states that Sterne studied current works on obstetrics, and in Book II, Chapter XIX, he mentions "Lithopædus Senonesis de Partu Difficili." This means that he had at least read the famous letter of Dr. Burton to William Smellie, where he takes him to task for his mistake. The mistake referred to is explained by the following passage from Dr. Burton's letter:

"If anything can be added to shock human Faith, or prejudice your Character as an Historian or Translator, it is your having converted Lithopædii Senonensis Icon (which you call Lithopædus Senonensis) an inanimate petrified substance, into an Author."

Sterne has aggravated the mistake by describing it as Lithopædus Senonesis. The name "Smelvgot" really refers to Smellie, and there is good reason for concluding that he may not only have read Dr. Burton's five shilling "Midwifery," but also Smellie's current book as well.

It is unlikely that he obtained his information direct from Dr. Burton of York, in whose persecution in 1745 he took part, and whom he has savagely caricatured as Dr. Slop.

My further rapid and, I am afraid, cursory researches, have proved the correctness of my first two conclusions.

His relations with Mrs. Sterne were far from ideal, although following their marriage in 1741, the "Parson who once delighted in debauchery" (to quote his wife's cousin), led the life of a contemporary parish priest for nearly twenty years.

Mrs. Sterne was not blameless, as Quennell describes her as having "proved a prickly and sharp-tongued woman who displayed great energy in housekeeping but very little skill."

Sterne's affairs with Catherine Fourmantelle, Mrs. Elizabeth Draper, and others were probably reactions, however unjustifiable in a parson, to the attitude of his wife.

As you know, Sterne had pulmonary tuberculosis, but it is possible that he also suffered from syphilis. At one stage of his terminal illness in May, 1767, the diagnosis of venereal disease was made, but he protested that this could not be right, as he had had "no commerce with the fair sex for fifteen years." Judging by what ultimately happened, the supposed venereal infection was almost certainly tuberculous.

He illustrates well the euphoric outlook of the patient suffering from phthisis. Even when he was within a few months of death he was writing to Mrs. Draper expressing the desire to marry her when her husband and his wife would die.

Another important factor in Sterne's outlook and future was the relationship between his own father and mother. His father had married his mother to settle a debt with her stepfather, who was anxious to dispose of her. The marriage was unhappy and from the age of ten Sterne was separated from his parents, being brought up and provided for by a cousin of his father. His mother has been described as a "vulgar, tactless, grasping woman, whom it would have been hard to love" (Quennell), and she certainly appears to have been a "limpet" in her later years. Sterne's conduct, however, towards his mother was most unfilial and it is stated that she was a prisoner in "the common gaol at York" for a time before her death in 1759.

He died on March 18, 1768, and was buried in St. George's, Hanover Square. It is said that only two men attended his funeral, one of them being his publisher. Some days later his body appeared in the dissecting rooms at Cambridge. It had been sold to the University by the resurrectionists—a macabre revenge for his attack on the medical profession (Radcliffe).

Having digressed to consider the author, one must return to the novel.

Having taken four chapters to discuss his conception and intrauterine life, the effect of prenatal influences, and to prove that he is his father's son, the first sentence in Chapter V either casts some doubt on his paternity or proves that Tristram Shandy was a premature baby.

In Chapter IV it is stated: "I was begot in the night betwixt the first Sunday

and the first Monday in the month of March in the year of our Lord one thousand seven hundred and eighteen.”

The first sentence of Chapter V to which I have referred reads: “On the fifth day of November, 1718, which to the aera fixed on, was as near nine kalendar months as any husband could in reason have expected.”

In this year (1950) the first Sunday of March was the 5th, and 280 days from that date would bring us to December 10th.

According to this calculation Tristram was born at the thirty-fifth instead of the fortieth week. He takes the trouble to explain in the last paragraph of Chapter IV that he could not have been conceived in December, January, or February, so that without casting doubts on his mother’s chastity, we must conclude that he was premature. I think this has an important bearing on a thesis I intend to develop at a later stage.

In building up the details of his arrival into “This scurvy and disastrous world of ours” he gives a most interesting picture of contemporary obstetrics.

Look at his description of the local midwife and you will get a vivid picture of the type of woman who practised the art of midwifery in that period:

“She had been left, it seems, a widow in great distress, with three or four small children, in her forty-seventh year; and as she was at that time a person of decent carriage—grave deportment—a woman moreover of few words, and withal an object of compassion.”

Compare this with the qualifications recommended by William Smellie, the famous contemporary London obstetrician. “Nurses,” he says, “as well as midwives, ought to be of middle age, sober, patient and discreet, able to bear fatigue and watching, free from external deformity, cutaneous eruptions and inward complaints, that may be troublesome or infectious.”

In the same chapter, in describing this midwife, he gives what might be the definition of the ideal obstetrician: “with the help of a little plain good sense, and some years’ full employment in her business, in which she had all along trusted little to her own efforts, and great deal to those of dame nature.”

He makes a note that the parson, Yorick, “paid the fees” for the midwives licence “amounting in the whole, to the sum of eighteen shillings and four pence.” At this period it was still the practice for midwives to be licensed by the bishops, and a godly, righteous, and sober life was of more importance than a knowledge of the art of obstetrics. These women were usually qualified to be midwives solely on account of the fact that they were married women who had had children themselves and had supervised the confinements of many of their neighbours.

They were often destitute widows who were prepared to submit to the drudgery and long hours associated with this branch of medicine, so as to keep body and soul together. They knew nothing about the physiology or pathology of labour, apart from that which they had acquired as the result of years of experience and in many cases good common sense.

This type of midwife existed in these islands until well on in the 1920’s.

The fact that the midwife was secured for the area in which Tristram was born, so as to allow Yorick to keep a good horse, brings to light the fact that many

women in this period must have had babies without any assistance, apart from that given by neighbouring women.

The reason why Tristram Shandy was born in Shandy Hall under the care of the local midwife and Dr. Slop, instead of in London under the care of Dr. Manningham, is revealed when we read the article in his mother's marriage settlement in Chapter XIV, Book I. According to this article the sum of £120 was to be paid to Mrs. Shandy to provide her with a coach and cover the expenses of her confinement in London. This reveals a consideration for the expectant mother which was remarkable for the period. It means that Sterne, in writing this, realised that a woman attended at her confinement in London might receive more skilled attention than was available in the Provinces. It may, of course, have been a subtle insult to Dr. Burton of York, whom he evidently despised and insulted on every possible occasion. Doran, in his papers, also makes the latter suggestion.

There was, however, a protective clause in the marriage settlement which Tristram describes as follows: "That in case my mother hereafter should, at any time, put my father to the trouble and expense of a London journey upon false cries and tokens; that for every such instance she should forfeit all the right and title which the covenant gave her to the next turn; but to no more."

He then says; "But I was begot and born to misfortune; for my poor mother, whether it was wind or water—or a compound of both—or neither; or whether it was simply the mere swell of imagination and fancy in her; or how far a strong wish and desire to have it so, might mislead her judgment."

The above is a most accurate description of a well-recognised obstetric condition, namely, pseudocyesis. In this condition the patient falsely believes herself to be pregnant, believes that she has all the subjective symptoms and produces the objective signs by abdominal distension.

This incident happened in September, 1717, the year before Tristram was born, with the result that when Mrs. Shandy was found to be genuinely pregnant in the following spring, his father insisted on the observance of the protecting clause.

Chapter XVI reveals the reaction of a cantankerous husband who has been made look a fool. The disappointment at the loss of a probable son and "the foolish figure they should both make at Church the first Sunday" are perhaps understandable. The fact that Mrs. Shandy evidently saw the funny side of the incident cannot have made Mr. Shandy accept the inevitable in any better spirit.

As Tristram says, in Book I, Chapter XV: "so that I was doomed, by marriage articles, to have my nose squeezed as flat to my face, as if the destinies had actually spun me without one."

In Chapter XVIII, Book I, we read: "As the point was that night agreed, or rather determined, that my mother should lye-in of me in the country, she began to cast her eyes upon the midwife, as the famous Dr. Manningham was not to be had."

The famous Dr. Manningham referred to above was later Sir Richard Manningham, who was the leading obstetrician of his day, and had the honour of establishing the first lying-in wards in these Islands in 1739. The house adjoining his own in Jermyn Street, London, accommodated parturient women and was the

forerunner of Queen Charlotte's Hospital. He was also famous for his exposure of Mary Tofts, who pretended to give birth to rabbits.

Mr. Shandy disagreed with his wife regarding the employment of a midwife alone, when, as described in Chapter XVIII, Book I: "there was a scientific operator within so near a call as eight miles of us, and who, moreover, had expressly wrote a five shillings book upon the subject of midwifery, in which he had exposed . . . the blunders of the sisterhood (midwives) itself." This refers to Dr. Burton's small book and also shows the rivalry which existed between the midwives and the obstetricians of the day. At this time Smellie, in London, was being exposed to vicious attacks, not only by his colleagues, but also by midwives, particularly a Mrs. Nihell, who referred to him as "a great horse godmother of a he-midwife."

At a later stage in the novel Mr. Shandy accuses Uncle Toby of distracting the "man midwife" from his duty to Mrs. Shandy. This irritates Slop, who makes the interpellation: "Accoucheur if you please."

Ultimately Mr. Shandy gains his point to a limited extent: "In a word, my mother was to have the old woman, and the operator was to have licence to drink a bottle of wine with my father and my Uncle Toby Shandy in the back parlour, for which he was to be paid five guineas." As events turned out, Mrs. Shandy and the baby would have been safer with the midwife alone, or perhaps the bottle of wine had clouded Dr. Slop's judgment. It is a pity that Dr. Slop was not aware of, and did not follow, the dictum laid down by Mr. Shandy in Chapter VI, Book II: "The doctor must be paid the same for inaction as action—if not better—to keep him in temper."

This follows a remark by Uncle Toby, who had suggested that Mrs. Shandy did not want Dr. Slop so as to save expense, but later suggests (Chapter VI, Book II) that it was modesty on her part. Modesty was probably a big factor at this period, but could not apply in Mrs. Shandy's case, as she had evidently been attended at her first confinement by Dr. Manningham. This, of course, like Dr. Slop, is an anachronism, because Manningham was only twenty-eight years old in 1718 and was younger when Tristram's elder brother was born and therefore relatively unknown.

The exact arrangement between Mr. and Mrs. Shandy is described in Chapter XVIII, Book II: "I have ordered, answered my father, the old midwife to come down to us upon the least difficulty; for you must know, Dr. Slop, that by express treaty, solemnly ratified between me and my wife, you are no more than an auxiliary in this affair— and not so much as that—unless the lean old mother of a midwife above stairs cannot do without you." Yet another example of Sterne making little of Dr. Burton. It has been suggested that Mr. Shandy's object in having Dr. Slop present was to have Tristram delivered by Cæsarean section. In Chapter XIX, Book II, there is a dissertation on the risks of a baby being born by the vertex with some misstatements.

It is stated "that by the force of a woman's efforts, which, in strong labour pains, was equal, upon an average, to the weight of 470 pounds avoirdupois acting perpendicularly upon it; it so happened that, in forty-nine out of fifty, the said

head was compressed and moulded into the shape of a conical piece of dough— Good God, cried my father, what havock and destruction must this make in the infinitely fine and tender texture of the cerebellum.”

The force exerted on the child's head is grossly exaggerated in the above statement. At the most, the force is somewhere between thirty-two to fifty pounds. Moulding of the child's head does occur in labours where there is some disproportion between the size of the head and the maternal pelvis and may cause injury to the brain as the result of small intracranial vessels being torn. Mr. Shandy's anxiety is understandable, as his first son, Bobby, was born head first and developed into what appears to be a mental deficient (“A lad of wonderful slow parts”).

We know that Mr. Shandy did raise the question of Cæsarean section as “he had read and was satisfied . . . that the belly of the mother might be opened extremely well to give a passage to the child. He mentioned the thing one afternoon to my mother—merely as a matter of fact.” It was natural for Mrs. Shandy to be averse to having a Cæsarean section. Her reactions are mentioned in Chapter XIX, Book II: “but seeing her turn as pale as ashes at the very mention of it—he thought it as well to say no more of it—contenting himself with admiring—what he thought was to no purpose to propose.” To have had such an operation without anæsthesia at a period when it was associated with a mortality of approximately seventy per cent. was more than any ordinary woman could face.

The advantages of the baby being born feet first are also discussed. At this period, and for two hundred years after, it was common practice to convert a head presentation into a breach when dealing with obstruction in labour. The risks, of course, were greater than is suggested in *Tristram Shandy*.

Again I must digress to discuss briefly the man Dr. Burton caricatured as “Dr. Slop.” Burton was the son of a London merchant born in 1710 at Colchester and educated at Merchant Taylor's School. Therefore “Dr. Slop” is an anachronism, as Burton was only eight years old when *Tristram Shandy* was born.

Burton graduated from Cambridge in 1733 and later studied under Boerhaave at Leyden. On his return from Holland he settled at York, where he became a well-known obstetrician and antiquary. Doran (1913) states: “According to Percy Fitzgerald, Burton acquired the nickname “Dr. Slop” early in his career, presumably before “*Tristram Shandy*” was written.” His philanthropic work for the poor of York resulted in the foundation of York County Hospital, against the opposition of Sterne's uncle and probably Sterne himself.

Through what appears to have been an accident and the machinations of his enemies, he was arrested and confined in York Gaol for supposedly being involved in the Pretenders Rebellion of 1745. Sterne's uncle went to great pains to try to have him executed, but was unsuccessful, and, after appearing before the Privy Council in London, he returned to practise in York.

This brief outline may explain why Sterne held “Dr. Slop” up to ridicule and contempt. This attitude is obvious from the very first introduction of “Dr. Slop” into “*Tristram Shandy*.” The rather amusing description of Dr. Slop being upset in the mud by Obadiah and that of the entrance of the bespattered doctor into the room in Shandy Hall make him appear a ludicrous figure. He is described as: “a

little squat, uncourtly figure of about four feet and a half perpendicular height, with a breadth of back, and a sesquipedality of belly which might have done honour to a serjeant in the horse guards.”

When we add to this the information in Chapter X, Book III, that he had knocked out three of his best teeth through his forceps slipping at a hard labour, he certainly was an unprepossessing figure.

Is it any wonder that Uncle Toby offered the opinion, “That mayhap this sister might not care to let such a Dr. Slop come so near her . . . .”

In Chapter X, Volume II, we read : “Dr. Slop’s presence, at that time, was no less problematical than the mode of it; though it is certain, one moment’s reflection in my father might have solved it; for he had apprized Dr. Slop but the week before that, and my mother was at her full reckoning; and as the doctor had heard nothing since, ’twas natural and very political too in him, to have taken a ride to Shandy Hall, as he did, to see how matters went.”

Here we see a doctor in 1718 (really after 1733) coming on an antenatal visit, for which one must give him credit. At this time, and indeed until well on in the present century, such a visit was not considered necessary and might, indeed, have been resented. This is probably a charitable explanation. The true explanation may be that through “marriage articles” he had secured an important patient who had previously been attended by a London colleague and he was anxious to ensure that the local midwife did not belittle him by attending the case herself without summoning him. It did mean, however, that he arrived unprepared for a confinement.

As described in the novel : “Thou hast come forth unarmed; thou hast left thy tire-tete, thy new invented forceps, thy crochet, thy squirt, and all thy instruments of salvation and deliverance behind thee.”

The tire-tete is a destructive instrument; the crochet is a hook used in days gone by in difficult breech extractions; and the squirt is a syringe used for baptising the unborn infant.

The “new invented forceps” refers to “Burton’s forceps” invented by himself. It was a brutal and powerful instrument as shown by Uncle Toby’s remark : “upon my honour, sir, you have torn every bit of the skin off the back of both my hands with your forceps and you have crushed all my knuckles into the bargain with them, to a jelly.”

Uncle Toby had evidently been allowing Dr. Slop to demonstrate his forceps, using his two closed fists to represent the foetal head.

The story of the obstetric forceps is one of the most interesting chapters in medical history, and, although not quite relevant to this paper, as Burton’s forceps and the results of their use have figured so prominently in “Tristram Shandy,” I feel justified in mentioning this bit of history at this stage.

The invention of the obstetric forceps was one of the most important epochs in the history of obstetrics. The instrument was invented by Dr. Peter Chamberlen, the senior son (1560-1631) of Wm. Chamberlen, a Huguenot who escaped from France shortly before St. Bartholomew’s Day in 1572. Five generations of this family were eminent members of the medical profession.



Peter Chamberlen (1601-1683) and his successors kept the forceps as a family secret for the purpose of family aggrandisement, and, one regrets to say, family profit. Peter's son endeavoured to sell the family secret to a contemporary Parisian obstetrician, who, before he would buy the instrument, demanded that he should deliver a patient in whom a delivery per vias naturales must have been obviously impossible. He failed to deliver the patient and had to return to England with the family secret.

The first published account of the Chamberlen forceps was given by Dr. Chapman of South Halstead, Essex, in 1733, the year that Dr. Burton qualified in Cambridge.

Burton's forceps were not invented until long after Tristram was supposed to be born; Palfyn did not make public his *mains de fer* until 1720, and, as I have mentioned, the Chapman Essay was not published until 1733. Burton's forceps was a clumsy instrument and certainly should have caused serious injury to a child's head.

We read in Chapter VI, Book II, that, following some commotion in the room above, Uncle Toby advises Mr. Shandy to ring the bell and enquire what is amiss. Obadiah, on answering the bell, states: "my mistress is taken very badly," and, regarding Susannah the maid, "she is running the shortest cut into the town to fetch the old midwife." Obadiah was instructed to saddle a horse and "do you go directly for Dr. Slop, the man midwife . . . and let him know your mistress is fallen into labour and that I desire he will return with you with all speed."

We know that within "two minutes, thirteen seconds and three fifths" Dr. Slop arrived back with Obadiah, having been thrown from his pony after a collision with Obadiah about sixty yards from the stables. At some time in the next two hours and ten minutes (Chapter XVIII, Book III) Susannah arrived downstairs, where Dr. Slop, Mr. Shandy, and Uncle Toby were sitting, saying: "Bless my soul—my poor mistress is ready to faint—and her pains are gone—and the drops are done—and the bottle of julap is broke—and the child is where it was—you had better look at my mistress, but the midwife would gladly first give you an account how things are, so desires you would go upstairs and speak to her this moment."

Dr. Slop, however, having been superseded by the midwife, was now determined to establish his own position. "No," replied Dr. Slop, "'twould be full as proper, if the midwife came down on me." Susannah's description would suggest that Mrs. Shandy had developed uterine inertia. This is a condition where the patient appears to start in labour, but at some stage following this the uterine contractions cease and may not be re-established for hours or days.

There is another possibility, namely, that Mrs. Shandy was not in labour at all. Earlier I drew attention to the fact that Tristram was a premature baby and that on the date in question the gestation was only thirty-five weeks. It is not uncommon to see patients before full term who believe they are in labour, appear to be so, and yet do not commence properly for some weeks.

In Chapter XVII, Book III, we are permitted to hear the interview between the doctor and the midwife. Dr. Slop says: "And pray, good woman, after all will

you take upon you to say, it may not be the child's hip, as well as the child's head? "'Tis most certainly the head," replied the midwife.

This dialogue is followed by a discussion with Mr. Shandy regarding the danger of injury to the male genitals if forceps are applied to a male breech presentation.

The poor father's reaction to this appallingly bad psychological treatment by the doctor was to say: "but when your possibility has taken place at the hip—you may as well take off the head too."

Dr. Slop's departure to the patient is described as follows: "So taking the green bays bag in his hand, with the help of Obadiah's pumps, he tripped pretty nimbly, for a man of his size, across the room to the door, and from the door was shewn the way, by the good old midwife, to my mother's department."

Sterne, who revealed the secrets of the conjugal bed in the first chapter, has kept his readers out of the lying-in room. Allport suggests that Sterne realised how far he might go with his irreverent imagination without being unfrocked. In spite of this we can reach certain conclusions from details given before and after the delivery.

At the beginning of Chapter XVIII, which follows the departure of the doctor to attend Mrs. Shandy, we read: "It is two hours and ten minutes—and no more—cried my father, looking at his watch, since Dr. Slop and Obadiah arrived—but to my imagination it seems almost an age."

If one is to judge from the time that Obadiah was dispatched for the doctor and Susannah ran for the midwife, Mrs. Shandy cannot have been in labour for more than about three hours, and yet Dr. Slop proceeded upstairs with the evident intention of delivering the patient.

Even at a second confinement three hours in most cases would be a very short labour, and for such a patient not to be delivered at the end of this time could not be regarded delay in labour. Neither could it justify any meddlesome midwifery such as Dr. Slop evidently intended. That he was most anxious to display his skill with the forceps is revealed in Chapter IX, Book III, where he is having difficulty in disentangling Obadiah's knots. (It is on this occasion that he misses his front teeth!!) "'Tis God's mercy, quoth he (to himself), that Mrs. Shandy has had so bad a time of it—else she might have been brought to bed seven times told, before one half of these knots could have got untied." And later, following some commotion upstairs, "By all that is unfortunate, quoth Dr. Slop, unless I make haste, the thing will actually befall me as it is."

There is no indication in the text as to the interval between Dr. Slop's departure upstairs and the time that Trim announces that the doctor is in the kitchen making a bridge. Uncle Toby, who had been asleep, takes this to mean a drawbridge until Trim cries "God bless your honour 'tis a bridge for master's nose. In bringing him into the world with his vile instruments he has crushed his nose, Susannah says, as flat as a pancake to his face, and he is making a false bridge with a piece of cotton and a thin piece of whalebone out of Susannah's stays, to raise it up."

Undoubtedly Dr. Slop's forceps could have crushed the child's nose, but if I am correct in stating that Tristram was premature, it is most unlikely that a

premature child whose nose was "crushed flat as a pancake" could have survived. Such an injury would almost certainly have been associated with a serious and fatal fracture of a thin plate of bone at the base of the skull.

It is more likely that the nasal deformity was evidence of congenital syphilis, and this might be supported by what we know about the mental condition of Tristram's elder brother and the possible fact that Tristram was premature.

The reactions of Mr. Shandy and the sympathetic Uncle Toby are vividly portrayed. Uncle Toby, in Chapter IX, Book IV, tries to comfort Mr. Shandy by saying, "It might have been worse." "I don't comprehend," said my father, "suppose the hip had presented" replied my Uncle Toby, "as Dr. Slop foreboded." After reflexion and understanding that he referred to the danger of injury to the boy's genitals he agrees.

Having recovered from his distress and being rather casually treated by Susannah on the stairs, Mr. Shandy makes the following interesting and very true observation. "Of all the riddles of married life—of all the puzzling riddles in a married state—there is not one that has more intricacies in it than this—that from the very moment the mistress of the house is brought to bed, every female in it, from my lady's gentlewoman down to the cinder wench, becomes an inch taller for it; and give themselves more airs upon that single inch, than all the other inches put together."

We can conclude that the delivery was difficult—it was almost certain to be if carried out prematurely, and we can also conclude that the child did suffer, because it is recorded that it had a fit early the next morning. Susannah rushes into her master's bedroom: "Sir, the child's in a fit—the child is as black as my shoe."

This description would fit in with either the cyanotic attacks frequently seen in the twelve to twenty-four hours following delivery of a premature baby, or might be accounted for by cerebral irritation following an intracranial hæmorrhage, the result of injury. At any rate, the confusion and haste to have the child baptised resulted in it being christened Tristram instead of Trismegistus.

In Chapter XX, Book I, the subject of baptism is considered at considerable length. He makes his readers conclude that his mother was not a Catholic, because of the statement, "It was necessary I should be born before I was christened." Following this is the Memoire presented to the Doctors of the Sorbonne on the question of prenatal baptism.

The Catholic ritual directs the baptism of the child in cases of danger before it is born, provided that some part of the child's body can be seen by the baptiser. The Doctors of the Sorbonne in 1733 enlarged the powers of the midwives and doctors by determining that baptism can be administered to the child by injection. Hence the presence of a squirt or syringe in Dr. Slop's equipment.

Like Dr. Slop himself, the Memoire to the Doctors of the Sorbonne is an anachronism, as this incident occurred twenty years after Tristram was born.

In discussing the obstetric aspects of Tristram Shandy, I have so far confined myself to a large extent to the text of the novel, but in conclusion I should like

to take a broader view of these aspects in the light of contemporary medical history.

In this work of fiction Laurence Sterne has given us a relatively clear picture of the obstetrical customs of his time. "Tristram Shandy" was published in 1759 and during the previous twenty years a gradual revolution had been occurring in obstetric practice. Prior to the advent of the Chamberlens and Smellie, and, indeed, during their reign, midwifery had been in the hands of uneducated midwives. The invention of the obstetric forceps and the teaching of Smellie resulted in a slow and gradual change, which was not secured without one of the fiercest discussions that ever took place in medicine. Sterne, in "Tristram Shandy," brought this discussion to the knowledge of the general public of his day and, to a certain extent, threw a beam of light on the prevailing iniquities of both sides taking part in the debate.

He revealed, perhaps unconsciously, the great dangers to which the expectant and parturient woman of his day was exposed. Admittedly, he has done so in an exasperating and irreverent fashion, and, in doing so, he reveals the latitude permitted by the authorities of the Church of England of that period. It is hard to tell if Sterne was trying to be the Dickens of his generation by exposing these prevalent evils.

One is inclined to think that his exposure was unwitting and only resulted from his spiteful caricature of Dr. Burton. If this is so, then at least one can say "out of evil came good."

I wish to acknowledge with thanks my indebtedness to Professor Miles H. Phillips, Laugharne, Emeritus Professor of Obstetrics and Gynæcology, University of Sheffield, for reading the paper and for his helpful criticism.

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#### REVIEW

**HANDBOOK OF ENCEPHALOGRAPHY.** By Robert S. Ogilvie. Addison-Wesley Press Inc., Cambridge 42, Mass.

THIS is a very good little book, of some 129 pages, for anyone interested in this comparatively new subject. The author was technician to Dr. and Mrs. F. A. Gibbs and is thus well equipped to pass on valuable experience. After a brief history of the subject, the author describes the setting up of a laboratory and follows the various steps to the interpretation of the tracing. The book will be particularly interesting to people with a basic knowledge of modern electricity and who wish to take up this fascinating branch. It will also be useful to medical men who do not require too technical a knowledge of the subject. The book is essentially American and no British workers are mentioned in the bibliography. The list of manufacturers of the electro-encephalograph apparatus is useless over here.

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H. H. S.

# A Survey of Hæmoglobin Levels of Children attending the Royal Belfast Hospital for Sick Children

By A. W. DICKIE, M.D., D.C.H.

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## INTRODUCTION

THE aims of the present investigation are set out below:—

- (a) To estimate the level of hæmoglobin in the blood of children between the ages of six and twelve years, inclusive, attending the Royal Belfast Hospital for Sick Children between June, 1947, and September, 1948.
- (b) To examine the possibility of a relationship between hæmoglobin level and social influence.

## PREVIOUS ENQUIRIES

The Committee on Hæmoglobin Surveys of the Medical Research Council (1945) summarised the work done in estimation of hæmoglobin levels in children until 1945. In the section of their report on children over 5 years and under 15 years, they point out that there are no accurate results available previous to those obtained from their observations in 1943. As far as possible, comparable tabulations of the results of previous workers (Davidson *et al.*, 1935, 1942, 1943; Colver, 1938; Wills *et al.*, 1941-42; Fullerton *et al.*, 1943) are given.

The relevant conclusions reached in the M.R.C. report are (1) that the average levels found in the small group of children in the last wartime survey were no lower than those found in the pre-war period and in 1940-42, and (2) that in some age groups there was evidence of an increase in the hæmoglobin level.

In general, the relationship between hæmoglobin level and age in children was described as follows:—"The picture is one of a rise from age one up to age six or seven, followed by a plateau of little change up to age ten or eleven, and then a steady rise once more."

Mackay *et al.* (1946), in a survey of 493 children aged 4 to 14 years at a County Council school in a town in Surrey, showed that "when the proportion of wage-earners in the household was high the mean hæmoglobin level of the children was also relatively high; when the number of dependent children increased the hæmoglobin dropped correspondingly; findings which confirm those of an investigation undertaken earlier in the war. Children of men in the fighting services had a lower mean hæmoglobin level than the children of civilians at all ages under 10 years, after which home influence on the hæmoglobin level was masked by evacuation. There was a similar difference in hæmoglobin level between 'Forces children' and 'Civilian children' when results were analysed to show the effect of increasing numbers of dependent children. We have no evidence as to the optimum or normal mean hæmoglobin level for schoolchildren, but the lowered

hæmoglobin level in 'Forces children' and among the children belonging to larger families reflects, we believe, an inadequate standard of living among at least a proportion of those composing these groups."

#### CLINICAL METHODS USED

The reliability of clinical and other methods available for the determination of hæmoglobin were investigated by Macfarlane *et al.* (1948). Of these, the Haldane-Gowers hæmoglobinometer was recommended by the Medical Research Council for future surveys. However, after consultation with Professor Harrison, Department of Biochemistry, Queen's University, Belfast, it was decided to use the "copper sulphate" method. This had the advantage at the time of commencement of this survey of not requiring any elaborate laboratory equipment not already available, and at the same time of being a generally accepted accurate method.

The copper sulphate method is not a colorimetric procedure, which was also regarded favourably. As regards the dilution methods in common use (Haldane, Gowers, and Sahli), Macfarlane (1948) stated that on the whole they are less reliable than other methods and adds that "even with a single 'average' observer, differences of ten per cent. or more must be found before they can be regarded as significant. This finding is not unexpected, since the dilution method allows of a colour change in one direction only, provides bad conditions for colour matching, and involves technical errors of mixing and reading."

The technique used was based on the report issued by the United States Naval Research Unit at the Hospital of the Rockefeller Institute for Medical Research (1945).

#### PRINCIPLE OF METHOD

The technique consists of letting drops of plasma or whole blood fall into a graded series of solutions of copper sulphate, each of known and different specific gravity, and noting whether the drops rise or fall in the solution. Each drop, on entering the solution, becomes encased in a sac of copper-protein, and remains as a discrete drop without change of gravity for fifteen to twenty seconds. During that period the rise or fall of the drop indicates its gravity relative to that of the solution. The fall indicates that the copper sulphate solution content has lower specific gravity than that of the plasma or whole white blood and vice versa. Consequently, it is possible to determine the specific gravity of the plasma or whole blood by reference to the specific gravity of the solution in which the drop remains stationary. This method is capable of measuring gravities to  $\pm 0.00005$ , which is more than ten times the accuracy required for the estimation of the hæmoglobin either from the nomogram or from the formula set out below. The drops of plasma or whole blood may be of any volume, and hence no special pipette is needed for delivering the drops into the copper sulphate solutions. No temperature correction is needed, because the temperature co-efficients of expansion of the copper sulphate solutions approximate to those of blood and plasma.

The copper sulphate solutions automatically clean themselves after each test, because within a minute or two after the test is completed the material of the

drop settles to the bottom as a precipitate. These solutions are prepared by dilution of a stock solution, which has, at 25°C, 1.100 times the density of water.

With the whole blood and plasma specific gravity readings, the hæmoglobin level can either be read off from the nomogram (given in the original report), or calculated from the following formula (upon which the nomogram is based) :—

Grams of hæmoglobin per 100 c.c. of blood :—

$$33.9 \times \frac{G_B - G_P}{1.0970 - G_P}$$

where  $G_B$  = whole blood specific gravity, and

$G_P$  = plasma specific gravity.

#### PREPARATION OF THE GRADED SERIES OF COPPER SULPHATE SOLUTIONS

The crystalline copper sulphate used had the exact composition of  $\text{CuSO}_4 \cdot 5\text{H}_2\text{O}$ . Of the several techniques available, the method of weighing portions of pure copper sulphate was used. This method is easiest when an accurate balance is available.

The preparation of the stock solutions was carried out in the Department of Biochemistry at Queen's University, and, to ensure accuracy, the specific gravity (1.1000 times the density of water at 25°C.) was checked by an alternative method.

The range of the graded series of the copper sulphate solutions prepared from the stock solution was not as great as used for normal laboratory purposes, as it was expected that the samples of blood examined would fall within a much narrower range. This proved to be true. In general, for estimation of whole blood specific gravities the copper sulphate solutions ranged from 1.048 to 1.060, and for plasma from 1.024 to 1.030. To prevent a change in specific gravity due to evaporation, all solutions were kept tightly corked.

#### DRAWING AND PREPARATION OF BLOOD AND PLASMA

Venous blood was used and care was taken to see that the tourniquet was not applied for more than one minute, as longer application has been shown to force so much fluid out of the blood that the hæmoglobin concentration is measurably increased. It is equally necessary to use dry syringes and needles to prevent dilution of the blood. The sample of blood was immediately placed in a small bottle containing a fixed amount of ammonium and potassium oxalate (prepared from a Heller and Paul's oxalate mixture) and the bottle slowly rotated through its vertical axis about ten times.

It was necessary to make certain corrections, since it is known that a slight error occurs when using this oxalate mixture. It was, therefore, essential to note the amount of blood taken to enable this correction to be made.

#### DETERMINATION OF GRAVITY

When examining whole blood it was essential at the start of each test to invert the bottle not less than ten times to ensure adequate mixing of the cells and plasma.

The drop of plasma or whole blood was delivered from about the height of



one centimeter above the solution. It has been observed that if the drop falls from too great a height it may be broken up on striking the solution, or its momentum may carry it too far below the surface. On the other hand, if it strikes with too little force, the drop may not break through the surface film. It is preferable to use small drops for the reason that they permit more tests before the standard solution must be changed. Therefore, a dropper with a fine tip was used in preference to a coarse one. The drop was delivered with the dropper held steadied on the edge of the bottle.

The delivered drop breaks through the surface film of the solution and penetrates 2 to 3 cms. below the surface; within five seconds the momentum of the fall is lost and the drop then either begins to rise, or becomes stationary, or continues to fall. The gravity of the drop relative to the solution does not change appreciably until the drop has been immersed in the solution for another ten or fifteen seconds, and there is ample time to note its behaviour during this interval. If the drop is lighter than the test solution it will rise, perhaps only a few millimetres, and may begin to sink immediately afterwards. If the drop is of the same gravity as the standard test solution it will become stationary for this interval and then fall. If the drop is heavier it will continue to fall during the interval. The rise or fall of the drop indicates whether the next test should be carried out on a solution of copper sulphate of a higher or lower specific gravity.

To sum up, the behaviour during the ten seconds after the drop has lost the momentum of its fall into the solution indicates whether the drop is lighter or heavier than the test solution. If it rises at all during this period it is lighter than the test solution. The specific gravity of the solution in which the drop is stationary during this ten-second interval indicates the specific gravity of the blood or plasma.

In practice, the specific gravity of the whole blood was estimated first and the sample left overnight. The plasma specific gravity was then estimated. With these two corrected readings it was possible to read off the hæmoglobin (expressed in grams per cent.) from the nomogram or calculated from the formula given previously.

#### SPECIAL POINTS AND PRECAUTIONS IN THE DETERMINATION OF GRAVITY

1. The surface of the copper sulphate solution must always be clean and free from fragments. If this is not so it will prevent a clean break-through of the next drop. These fragments can be removed by tapping the container (when they sink to the bottom), or removed with a wooden applicator stick.
2. Avoidance of an air bubble in the drop—as even a minute bubble will make the drop of plasma or blood float. To prevent this, the tip of the dropper must be kept immersed below the surface of the blood or plasma; and when the drop is being expelled into the copper sulphate solution, some blood or plasma must remain in the pipette.
3. Movements of the copper sulphate solutions can give false readings; therefore, the solution must be allowed to settle before use. Convection currents, such as result from bringing cold bottles of copper sulphate into a warm room, may induce similar false readings when used at once.

4. Each test solution can only contain a certain number of drops before a change in specific gravity occurs. In the original investigation of the copper sulphate method, tests showed that a standard solution will receive about one-fortieth its volume of plasma or blood, or one small drop per c.c., under the condition of the tests, before the specific gravity of the standard is decreased by 0.0005. Some hæmolysis occurs when whole blood is tested, resulting in the colour of the solutions shifting from blue towards green and becoming slightly turbid from suspended unclaked cells. However, the gravity of the solution is not changed by more than 0.0005 until one-fortieth its volume of blood has been added.

#### THE VALUE OF HÆMOGLOBIN LEVELS

In interpreting the results of any survey based on hæmoglobin estimations, it should be remembered that the low hæmoglobin may also be secondary to defects in hæmopoiesis brought about by deficiency of a dietary constituent or to some independent disease. Therefore, in planning a survey of hæmoglobin levels, it is necessary to have in mind the fundamental physiological factors which influence hæmoglobin metabolism. Pathological factors, such as severe hæmorrhage, infection, or the taking of drugs influencing hæmoglobin levels, should be noted.

The physiological factors of particular importance are:—

1. Intake of hæmopoietic principles (iron, copper, cobalt, protein, etc.).
2. Absorption of hæmopoietic principles (gastric achlorhydria, etc.).
3. Increased demands for hæmopoietic principles (external blood loss, rapid growth, etc.).
4. Changes in plasma volume (exercise, posture, etc.).

The following factors may, therefore, be considered "criteria of normality" for the purpose of determining hæmoglobin levels:—

1. A diet adequate in all respects.
2. No abnormality of gastric or intestinal function.
3. No increased demand for hæmopoietic factors, which is not adequately covered by increased diet.
4. No change in plasma volume unless groups where such a change is present are being studied, and where the degree of dilution is actually measured.
5. No intercurrent disease affecting the hæmoglobin level.

Technical methods greatly influence the value of hæmoglobin estimation, especially when different observers are taking part in the same survey. It is apparent that a rigidly defined method must be adhered to by the observers. The "matching of colours" is a source of error that is very difficult to overcome. In this survey the author did all the clinical and laboratory work and the method used did not involve the "matching of colours."

#### SUBJECTS USED

The children selected as the subjects of this enquiry were all patients between the ages of six and twelve years attending the out-patient department for attention to minor injuries, their friends of the same age accompanying them and children

of the same ages admitted to the surgical ward for routine operation. Children suffering from conditions (e.g., shock or chronic sepsis) likely to affect their hæmoglobin level were not, therefore, included.

It should be realised that the source of the data therefore restricts the survey to the children described. The results cannot, thus, be reasonably attributed to the child population of the whole of Belfast or some wider area. But in so far as no known bias has entered into the selection of subjects and all those defined were included in the ultimate analysis, it is reasonable to conclude that the results do give an adequate description of the hæmoglobin level of children attending the hospital for reasons which are unlikely to cause abnormal hæmoglobin levels.

In the sample of 403 children examined, an assessment of social class of parents (using the occupational criterion of the Registrar General of England and Wales [1927] ) was made in respect of 367—the distribution was found to be :—

| Social Class    | No.   | Per Cent. |
|-----------------|-------|-----------|
| I and II ... .. | 2     | 0.5       |
| III ... ..      | 17    | 4.6       |
| IV and V ... .. | 348   | 94.8      |
|                 | <hr/> | <hr/>     |
|                 | 367   | 99.9      |

No information is available about the present distribution of the population by social class, but it is unlikely that over ninety per cent. would be drawn from the lowest classes, as is the case in the present series.

Again, the sex distribution of the sample was 291 males (72.2 per cent.) and 112 females (27.8 per cent.), which is significantly different ( $X^2=76.01$ ,  $n=1$ ,  $P < 0.001$ ) from the sex distribution of the County Borough of Belfast (Registrar General for Northern Ireland [1938] ) in 1931 at the ages concerned. This is the last available official figure which can be used for purposes of comparison, but it is unlikely that the sex distribution of the Borough has changed materially—it was 50.6 per cent. males and 49.4 per cent. females at the ages concerned.

Finally, one other test can be made and this is in respect of the age distribution of the sample :

| Years    | No.   | Per Cent. | Per Cent. |
|----------|-------|-----------|-----------|
| 6 - ...  | 53    | 13.2      | (14.3)    |
| 7 - ...  | 53    | 13.2      | (13.8)    |
| 8 - ...  | 63    | 15.6      | (13.9)    |
| 9 - ...  | 58    | 14.4      | (13.8)    |
| 10 - ... | 56    | 13.9      | (14.7)    |
| 11 - ... | 66    | 16.4      | (14.7)    |
| 12 - ... | 54    | 13.4      | (14.7)    |
|          | <hr/> | <hr/>     | <hr/>     |
|          | 403   | 100.1     | (99.9)    |

Using the same official figures as above (Registrar General for Northern Ireland [1938] ), the distribution shown in brackets is obtained. These two distributions do not differ more than could easily have arisen by chance ( $X^2=2.88$ ,  $n=6$ ,  $0.9 > P > 0.8$ ).

To sum up, although no evidence exists that the age distribution of the sample is unlike that of the similar age range of the children of Belfast as a whole, in other respects there is no evidence that the sample can be taken as an unbiased sample of all Belfast children.

#### DATA COLLECTED

Information was collected and estimations were made of the following variables. Unfortunately, complete records were not available in all cases and the numbers refer to the number of children for whom the information stated was available.

|  | No. |
|--|-----|
| Hæmoglobin calculation in grams per 100 c.c. ... | 403 |
| Age in years ... ..                              | 403 |
| Sex ... ..                                       | 403 |
| Cash allowance per head ... ..                   | 346 |
| Food allowance per head ... ..                   | 319 |
| Rent of family ... ..                            | 350 |
| Social class ... ..                              | 367 |
| Rank of birth ... ..                             | 243 |

The omissions considerably complicate the analysis, and, where the proportion of admissions is large, render suspect any conclusion drawn. For example, if it was found that, other things being equal, hæmoglobin estimation increased from low to high rent groups, the position might be completely altered by the inclusion of the fifty-three children for whom no rent particulars were available should it so have happened that the bulk of these children were drawn from one rent group. Therefore, as far as possible tests were made to see if the hæmoglobin level of the children for whom the social environmental data were lacking differed from that of children for whom such data were available.

These omissions in many cases were due to lack of detailed knowledge of the family circumstances by the adult in attendance with the child, e.g., an aunt sometimes reported for the interview, or a father would not know how the "family allowance" was spent. In a number of cases the family lived too far away to be easily accessible, but in only one case did the mother refuse to give any information. As well as actual omissions, difficulty was often experienced in finding out the amount of money spent on food per week, as no mother keeps detailed household accounts. However, as far as possible, the almoner made every effort to obtain full details in each case.

Two assessments were made of a financial nature as indices of the types of families from which the children were drawn. One of these measured the total weekly income available, after deduction of rent, expressed per head of the family

(allowance per head). The other represented the average weekly amount spent on food. An estimate of each of these variables was available for 310 children. From these 310 observations the relationship between the two "allowances" variables was measured. A fairly high degree of significant correlation was found to exist ( $V=0.63$ , standard error 0.034).

The numerical assessment of these allowances must, in nature of the difficulty of obtaining the required information, be extremely approximate. In view of this it was decided to use the information collected not as exact quantitative variables, but as a means of allocating the children to fairly broad allowance groups. This being so, and in view of the fairly high correlation between the "food allowance" and "total allowance" per head, it was considered that there was little to be gained by using both variables. Therefore, only the total allowance, for which more observations (346) were available, has been used.

With regard to rank of birth, only 243 (60.4 per cent.) patients gave this information—this is such a high rate of wastage that it was considered that any analysis of this variable would be meaningless, and it was consequently ignored. The large number of omissions of birth rank was due to lack of a specific question on the form relating to the social aspect of the survey, and it was only in the 243 cases that sufficient family history was included to enable the birth rank to be deducted accurately.

#### RESULTS OF THE SURVEY

*Sex and Hæmoglobin.*—The mean hæmoglobin level for males was found to be 12.58 grams per 100 c.c., compared with 12.59 for females. As might be expected, this small difference was not statistically significant. Similarly, after allowance was made for the possible difference in age constitution of the two sexes and the possibility of a relationship between hæmoglobin level and age, there was still no significant difference in hæmoglobin level between the sexes.

It was, therefore, considered justified to ignore the sex composition of these data in the remaining analysis. The mean hæmoglobin level for both sexes combined is 12.59, with standard deviation of 1.09 grams per 100 c.c. The hæmoglobin distribution of the 403 observations appears in Table I.

*Age and Hæmoglobin.*—The data was classified into seven age groups (6-, 7-, etc.), each of one year interval. The observed mean hæmoglobin level in each group is given in Table II.

The analysis of variance of these data revealed a marked significant variation between age groups (variance ratio=4.42,  $n_1=6$ ,  $n_2=396$ ,  $P < 0.001$ ). A small but significant positive correlation existed between age and hæmoglobin level ( $V=+0.27$ , standard error 0.05) and, consequently, an attempt was made to describe the complete series of observations by means of a linear regression line. The equation is:—

$$\text{Hæmoglobin in grams per 100 c.c.} = 11.59 + 0.105 \times (\text{age in years}).$$

The means of hæmoglobin level for each group and the line to this equation are shown in Figure 1.

The regression coefficient, 0.105, differed significantly from zero (standard error=0.027,  $t=3.89$ , and  $P < 0.001$ ), and the regression revealed a good relationship (variance ratio=15.17,  $n_1=1$ ,  $n_2=396$ ,  $P < 0.001$ ). This relationship was not truly linear, the deviations from the calculated line were greater than could easily have arisen by chance (variance ratio=2.27,  $n_1=5$ ,  $n_2=396$ ,  $0.05 \times P > 0.01$ ). However, it was not considered advisable to complicate the analysis by attempting to fit a curve linear line to the observations, since the deviations of the group means from the line were only just significant.

TABLE I

| DISTRIBUTION OF HÆMOGLOBIN ESTIMATION IN THE SAMPLE |     |     |                        |
|---|-----|-----|------------------------|
| Hæmoglobin in grams per 100 c.c.                    |     |     | Number of Observations |
| 8 -   | ... | ... | 1                      |
| 9 -   | ... | ... | 5                      |
| 10 -  | ... | ... | 22                     |
| 11 -  | ... | ... | 74                     |
| 12 -  | ... | ... | 142                    |
| 13 -  | ... | ... | 119                    |
| 14 -  | ... | ... | 39                     |
| 15 -  | ... | ... | 1                      |
| TOTAL   |     |     | 403                    |

TABLE II

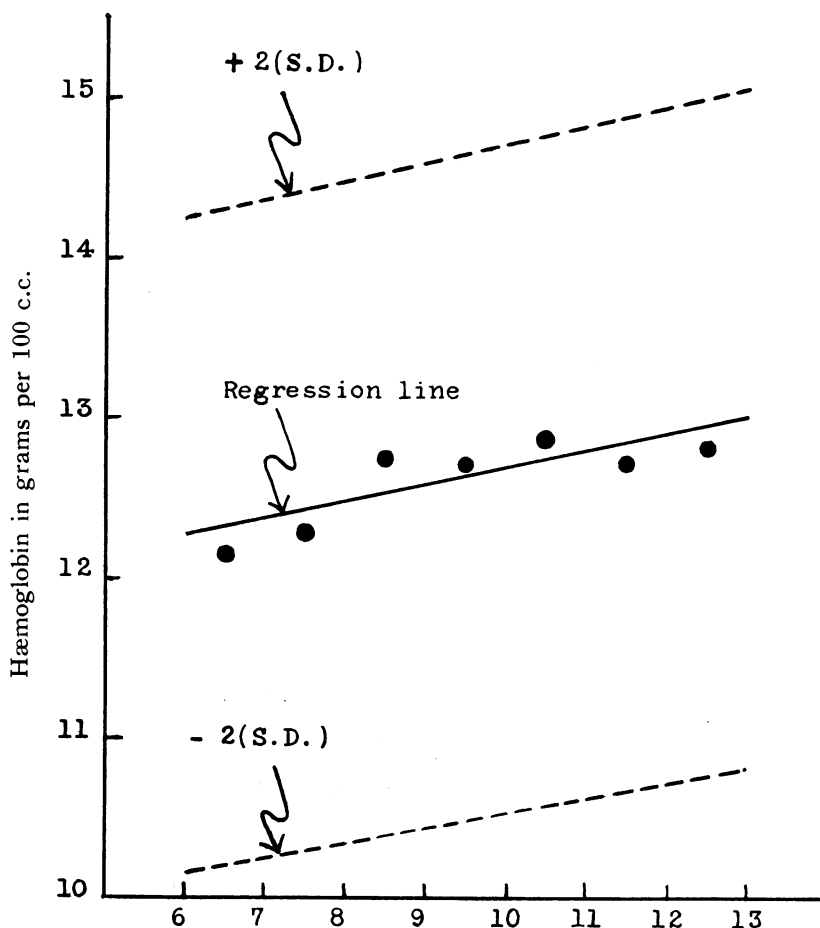
HÆMOGLOBIN LEVEL AND AGE

| Age Groups | Number of Observations |     | Mean Hæmoglobin in grams per 100 c.c. |             |
|------------|------------------------|-----|---------------------------------------|-------------|
|            |                        |     | Observed                              | *Calculated |
| 6 -        | ...                    | 53  | 12.11                                 | 12.27       |
| 7 -        | ...                    | 53  | 12.16                                 | 12.38       |
| 8 -        | ...                    | 63  | 12.77                                 | 12.48       |
| 9 -        | ...                    | 58  | 12.68                                 | 12.59       |
| 10 -       | ...                    | 56  | 12.88                                 | 12.69       |
| 11 -       | ...                    | 66  | 12.65                                 | 12.80       |
| 12 -       | ...                    | 54  | 12.77                                 | 12.90       |
| TOTAL      |                        | 403 | 12.59                                 |             |

\* Calculated from regression equation hæmoglobin in grams per 100 c.c.=11.59+0.105 x (age in years).

Standard deviation from regression=1.06 grams per 100 c.c.

FIGURE I  
 MEAN HÆMOGLOBIN LEVELS FOR EACH AGE GROUP AND THE CALCULATED  
 REGRESSION LINE



(Regression line : Hæmoglobin in grams per 100 c.c. =  $11.59 + 0.105 \times$  (Age in years)  
 (Standard deviation of regression = 1.06 grams per 100 c.c.)

In view of this association between hæmoglobin level and age it followed that the comparisons between the economic and social group which follow had to be corrected for possible age differences in the composition of such groups if the economic and social effects were to be observed unimpaired by the differential age influences.

Table III gives the observed hæmoglobin levels in this survey expressed as percentages. The standard used was that 14.8 grams of hæmoglobin per 100 c.c. as equivalent to one hundred per cent. of the National Physical Laboratory Haldane Hæmoglobin Standard (Macfarlane, personal communication, March, 1948).

TABLE III  
MEAN HÆMOGLOBIN LEVELS (EXPRESSED IN PERCENTAGES) FOR EACH AGE GROUP

| Age   |     |     |     | Mean Hæmoglobin Value |
|-------|-----|-----|-----|-----------------------|
| 6 -   | ... | ... | ... | 81.8                  |
| 7 -   | ... | ... | ... | 82.4                  |
| 8 -   | ... | ... | ... | 86.5                  |
| 9 -   | ... | ... | ... | 85.8                  |
| 10 -  | ... | ... | ... | 87.2                  |
| 11 -  | ... | ... | ... | 85.1                  |
| 12 -  | ... | ... | ... | 86.5                  |
| TOTAL |     |     |     | 85.1                  |

*Allowance and Hæmoglobin.*—The 346 children were classified into five allowances groups (0- shillings, 10- shillings, 15- shillings, 20- shillings, 25+ shillings). The significance of the variation in hæmoglobin levels between these groups was assessed by means of the method of analysis of variance. The variation between the means of the allowance groups was no greater than could easily have arisen by chance (variance ratio=1.75,  $n_1=4$ ,  $n_2=341$ ,  $0.2 > P > 0.1$ ). When the correction was made for age effects by the method of analysis of co-variance, even less variation was apparent between the adjusted means of the allowance groups (variance ratio=1.61,  $n_1=4$ ,  $n_2=340$ ,  $0.2 > P > 0.1$ ).

There is no evidence, from these data, of any significant difference in hæmoglobin levels of children drawn from families of different allowance groups, as classified in this investigation.

There were 57 children for whom no particulars relating to income were available. As far as hæmoglobin level is concerned, there seems no reason to suspect that these were a selected group. Compared with the 346 observations used, the difference between the means of the two groups is not significant (variance ratio  $< 1$ ) and the result is materially the same after correction for age (variance ratio=2.07,  $n_1=1$ ,  $n_2=400$ ,  $0.2 > P > 0.1$ ).

*Rent and Hæmoglobin.*—The data were similarly examined from the point of view of the rent paid by families from which the children were drawn. Rent levels were available for 350 children.

The rent groups used were 0- shillings, 4- shillings, 6- shillings, 10- shillings, and 14+ shillings. The variation in hæmoglobin levels between rent groups was no greater than could easily have arisen by chance and correction for differences in the age constitutions of the rent groups did not influence this result (variance ratio  $< 1$ ).

Amongst the 53 children who were omitted from this rent analysis were 13 whose parents were in the process of buying houses. While this number is too small to constitute a separate group, they have been included with the remaining



40 for whom no particulars of rent were available. The combined group of 53 omissions had a significantly greater mean hæmoglobin level than the 350 for whom rents were available (variance ratio=6.07,  $n_1=1$ ,  $n_2=401$ ,  $0.05 > P > 0.01$ ). This difference was not due to age differences in the two populations compared, since the co-variance analysis demonstrated even more strikingly the difference between the two groups (variance ratio=7.04,  $n_1=1$ ,  $n_2=401$ ,  $0.01 > P > 0.001$ ).

That this difference should arise suggests that the 53 observations omitted from the rent analysis represent a somewhat different group of children to those included. This being so, unless a completely different rent group is represented by the 53 omissions, it would appear that the 350 observations used might conceivably be a somewhat biased sample of the whole. This, therefore, decreases considerably the value of rent classification.

#### SUMMARY

This investigation had, as its aims, those set out in the introduction.

The first is summarised in Tables II and III and discussed in the report.

The interpretation of the social aspect of the survey is more difficult, largely because the social data were approximate or incomplete. However, the results did lead to the following conclusions :—

1. That there was no significant difference in the hæmoglobin levels between the sexes in the age groups reviewed.
2. That a small but significant positive correlation existed between age and hæmoglobin levels in the age groups reviewed.
3. That there was no evidence of any significant difference in the hæmoglobin levels of children drawn from families of different allowance groups.
4. That, although no evidence of an association between social status, as assessed by rent and hæmoglobin level, was shown, this is liable to reconsideration, since it is thought that the group for which rent particulars were available was not a representative sample of the total 403 children.

This survey was carried out at the Royal Belfast Hospital for Sick Children on the suggestion of Professor F. M. B. Allen. Such work had not previously been done here, and it was considered that it would be useful for comparison with similar surveys which may in the future be undertaken at the Hospital.

I would like to express my thanks to the almoners of the Royal Belfast Hospital for Sick Children (Miss Binns, Miss Eves, and Miss Brown) for their very valuable help in the social aspect of this survey; to Professor Allen, for his guidance on the clinical side.

The Department of Social and Preventive Medicine of Queen's University, Belfast, was not in being when this enquiry was planned and carried out, but Professor Stevenson and Dr. Cheeseman of that Department gave very willing help and encouragement in the statistical analysis of the collected data.

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## REVIEW

TEXT-BOOK OF SURGERY. By Patrick Kiely, B.Sc., M.D., M.Ch., F.R.C.S. (Eng.). Pp. 1184. N. K. Lewis & Co. Ltd. 45s.

THIS massive text-book, except for the chapters on disease of the ear, eye, nose, and throat, is the work of a single author. The writer lays before us his views and opinions after twenty-five years experience as a general surgeon and as a teacher at University College, Cork.

Professor Kiely is at pains to explain in the preface that this work is intended primarily for the student reading for his final examination, and not for the specialist or advanced postgraduate student of surgery. With this in mind he pays scanty attention to operative surgery and leaves out completely many of the classical operative procedures which have traditionally recurred in many of the standard British surgical text-books. He has the courage to leave unwritten the usual chapters on bacteriology and immunology, rightly believing that these subjects are to be studied more effectively in their standard text-books.

Criticism of a text-book, and particularly of a first edition, is easy, but an impression is created that this book is already a little out of date. The recent advances of surgery, particularly those associated with the introduction of the antibiotics and the anticoagulants, have led to many changes in surgical treatment, and this work is largely based on the surgery of the era before these changes. Many sections of the book are already out of date and require rewriting. This is no doubt in part due to the time taken for a single author to produce a work of such dimensions.

The subject matter is clear, concise, and the volume attractively produced. There are many long classifications which may appeal to the student, but are too full of rare and unlikely diseases to be of practical value. One might cavil at the lack of exact dosage where drugs are mentioned, and at the shortness and lack of detail relative to methods of treatment.

The text is liberally interspersed with diagrams, photographs, and microprints, all in black and white. The attractiveness of the book and the value of the reproductions would be greatly enhanced if some of these had been reproduced in colour.

It is refreshing to find a full-length text-book by an Irish author from an Irish medical school, and we welcome it as a new partner to the already established favourites from British medical schools.

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# BCG Vaccination in Stockholm

By A. W. DICKIE, M.D., D.C.H.

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From the Pædiatric Clinic of the Karolinska Institute at Norrtulls Hospital, Stockholm

THE first BCG vaccinations were carried out in Sweden in 1925. The vaccine was obtained from the Pasteur Institute in Paris and given orally to newborn babies as recommended by Calmette. In 1927, Wallgren, at that time in Gothenburg, introduced the intradermal method and laid down the criteria for vaccination. His recommendations have been in use ever since. The Swedish National Association for the Prevention of Tuberculosis was interested from the beginning and financed research and propaganda. The result to-day is that the Swedish public understand the value and the limitations of Calmette vaccination and the necessity for proper tuberculin testing before and after.

In the early years the vaccination was most often carried out in and around Gothenburg, and it is in this city that the vaccine is still produced. Gradually knowledge of the technique and proofs of its value spread throughout Sweden. From 1941 it has become universally used. The School Medical Services and the Medical Services of the Armed Forces made the vaccine available to all tuberculin negative schoolchildren and conscripts.

## AIM OF BCG VACCINATION

The aim of BCG vaccination is to increase the resistance to primary tuberculosis. The immunity is relative rather than absolute. It affords protection to tuberculin negative individuals and is, therefore, of greatest value in children and young adults. It is very valuable in those sections of the population (nurses, medical students, etc.) most exposed to infection, but in Sweden the vaccine is not restricted to these groups. As a child grows older he comes more and more into contact with people outside the immediate family circle and his chance of contracting tuberculosis greatly increases. It is well known that the older the child the less often is the source of infection discovered. For this reason, Swedish physicians, once they were convinced of the value of the Calmette vaccine, encouraged vaccination of all tuberculin negative people.

In large cities it is almost impossible to escape infection at some time during life. When the first infection occurs with virulent tubercle organisms during the period of increased resistance following BCG vaccination, there is, as a rule, no clinical disease, but the tuberculin test remains positive for a very long time, and very likely for life. Tuberculous meningitis and military tuberculosis are practically unknown in vaccinated children. A few cases have been reported, but often the

child was exposed to a source of infection prior to the onset of allergy following the vaccination. The extreme rarity of these serious complications of primary tuberculosis is one of the very good arguments in favour of the universal use of BCG vaccination.

#### TUBERCULIN TESTS

A tuberculin (Mantoux) test is considered negative only when 1.0 mg. (1/100), read after seventy-two hours, is less than 10 x 10 mm. redness and infiltration. If read after forty-eight hours the danger of pseudoreactions is considerable. The majority of false positive results have disappeared on the third day, but if there is the slightest doubt the test must be repeated. In general, a non-specific reaction will appear earlier and disappear sooner than a specific. The stronger the test dose the greater is the danger of a false positive reaction. The majority of cases of active primary tuberculosis will react to the ointment test or 0.01 mg. (1/10,000) Mantoux. Doses greater than 1.0 mg. are used only to exclude tuberculosis.

Two types of tuberculin tests are in common use in Stockholm. One is percutaneous and the other intradermal (Mantoux).

*Percutaneous Test.*—The skin over the centre of the sternum is chosen, as this is believed to be the most sensitive area. It is first cleaned with ether and the ointment\* applied in one of two ways.

(a) Patch Test—this is commonly used in private practice and in the out-patient department of this hospital. Adhesive plaster is placed over a small quantity of the ointment. It is removed by the mother in twenty-four hours and read by the doctor in seventy-two hours. The reaction will usually last about a week. A positive result may occasionally be delayed for five or six days. A control is not used. Deliberate scratching may give a false positive result.

(b) In hospital the ointment is rubbed into the skin and the area not washed for twenty-four hours. No cover is used. Otherwise it is the same as the patch test. It is absolutely necessary to rub in the ointment thoroughly, either with one hundred strokes, or for not less than one minute. The area is usually about the size of a half-crown. A reliable preparation must be used. The ointment test will become positive in the majority of cases of active primary tuberculosis in children.

*Intradermal Test.*—This is the familiar Mantoux test. The usual doses are 0.1 mg. (1/1,000) and 1.0 mg. (1/100). The 0.01 mg. (1/10,000) dose may be used as an alternative to the ointment. Higher doses, such as 3, 5, and 10 mg., are used only exceptionally to rule out tuberculosis.

The usual routine is an ointment test first, followed by 0.1 mg. and 1.0 mg. as necessary. The degree of tuberculin sensitivity is always much lower in infants than older children, and 0.1 m.g. as the starting dose can always be used with safety under one year. The ointment test is roughly equivalent to 0.01 mg. Old Tuberculin.

\* For the past ten years a French preparation, "Néotuberculin," produced by the Mérieux Laboratory in Lyon, has been used and found satisfactory.

## TUBERCULIN TESTING IN GENERAL PRACTICE

It is now a routine for all Swedish doctors, whether in the child welfare centres, the school medical services, general practice, etc., to tuberculin test all children during or after any febrile illness, and, in this way, many cases of primary tuberculosis are diagnosed during the initial fever stage. It is felt that even the most busy doctor has at least time to do an ointment test. For example, eighty-six out of eighty-eight cases of primary tuberculosis admitted last year were positive to the ointment test; the remaining two children reacted to 0.1 mg. O.T.

Concentrated Old Tuberculin keeps for several months, but diluted solutions do not. These should be changed, if possible, every week, but should not be used for more than a fortnight.

The following method of dilution is convenient. The concentrated solution contains 100 mg. in 0.1 c.c. Exactly 0.1 c.c. of this concentrated solution is taken into a 1 c.c. syringe and diluted to the mark 10 with sterile water, so that now 0.1 c.c. contains 10 mg. Old Tuberculin. The contents are thoroughly mixed and 0.9 c.c. ejected. The procedure is repeated with the remaining 0.1 c.c. After this dilution, 0.1 c.c. contains 1.0 mg. (1/100). A third dilution will give 0.1 mg. (1/1,000), and a fourth 0.01 mg. (1/10,000) in 0.1 c.c.

Accurate records of tuberculin tests are very valuable. The size and description of the reaction should always be noted.

## ROUTINE AND TECHNIQUE OF BCG VACCINATION

The vaccine should be used within one week of preparation and should be stored in a refrigerator. The bottle must be thoroughly shaken to ensure an even distribution, and, therefore, a more accurate dose.

The intradermal method developed by Wallgren in 1927 is in general use. The dose is 0.05 mg. contained in 0.1 c.c. of the vaccine, and the inoculation is done exactly like the Mantoux test. Throughout Sweden the site of the inoculation is on the upper outer aspect of the left thigh. This standardization has the advantage of helping the patient (and the doctor) to differentiate between a Mantoux test and a BCG vaccination. The intradermal method has proved so effective that the other methods (multiple puncture, etc.) are seldom used.

The patient must be tested immediately before the vaccination and be negative to 1.0 mg. Old Tuberculin. A check tuberculin test is carried out at six weeks and *only if this is positive* can a vaccination be said to have taken. If negative, it is better to repeat the tuberculin tests again in three or four weeks before re-vaccination. The child is then tuberculin tested at every opportunity and is re-vaccinated if the response to 1.0 mg. becomes negative. Wallgren recommends, as a minimum, tuberculin testing at 3, 7, 10, 15, and 20 years. The aim should be, especially where facilities are available as in schools, factories, etc., a yearly tuberculin test.

Experience has shown that the majority of vaccinated people will respond to the ointment test at six weeks. Infants, however, do not respond so readily. They are tuberculin tested three months after vaccination. While some may respond to

the ointment test, it is more usual under one year to start with Mantoux 0.1 mg. At this age the dose of the vaccine is usually increased 2-4 times. It is not necessary to carry out preliminary tuberculin tests on newborn babies in maternity hospitals.

The importance of a proper record system is easily understood. Every vaccination is recorded on a card, and, after the check test, this card is forwarded to the Central Tuberculosis Dispensary. This accurate information is then available to every doctor.

As pointed out previously, every child is tuberculin tested at every opportunity. These tests are routine in all hospitals (for adults as well as children), child welfare centres and children's homes, in all schools, in the armed services, in the industrial medical services, etc. Vaccination is always offered when the tests are negative, and, if accepted (as it almost always is), the vaccine is available immediately in each centre. Every doctor, whether in general practice or in other branches of the medical services, carries out his own routine tuberculin tests and vaccinations.

#### ONSET OF ALLERGY AND DURATION OF IMMUNITY

A positive tuberculin test is the only certain way of knowing that a vaccination has brought about an increased resistance to virulent tubercle bacilli. It is very probable that some immunity is present a few days before the onset of allergy and that this immunity exists for a few months after 1.0 mg. has become negative, but the degree of resistance is too low to be relied on. For practical purposes a sufficiently high degree of immunity can only be assumed to be present when the tuberculin test is definitely positive.

In primary tuberculosis the onset of allergy is sudden, and the reaction to tuberculin remains high over a long period of years. The following case, admitted to Norrtull's Hospital shortly after I arrived, demonstrates the suddenness of the onset of allergy and the possibility of carrying out the BCG vaccination during the incubation period of a primary infection.

Boy: aged 1 year; negative to 1.0 mg. Mantoux on 3/10/1949 and BCG vaccinated on this day. Erythema nodosum developed three days later and there was a reaction at the site of the vaccination (Koch's phenomenon). On the 10th, seven days after the original negative Mantoux, the ointment test was positive on admission to hospital.

When a vaccination is carried out during the incubation of a primary infection there is no danger to the patient. The danger is to the good name of Calmette vaccination, as the parents may unjustly blame this for the development of the active process.

Allergy following BCG vaccination has a slower onset, is never so intense as in a primary infection, and will only last for a number of years. The exact duration is very variable in different individuals. A small number become negative within one year, others in three or four years' time, while some are positive at the end of ten years. It is likely that when a patient is found positive a long number of years after vaccination, there has been a superinfection with virulent organisms.

It is this possibility of superinfection which makes it so difficult to estimate accurately the probable duration of immunity. When a positive tuberculin reaction is found, say ten years after vaccination, there are two possibilities. The patient may have had an infection with virulent tubercle bacilli during the period of BCG allergy. At this time, in all probability, there would have been a flare-up in the tuberculin sensitivity, with little or no clinical or X-ray evidence of active disease. On the other hand, the BCG allergy and immunity may have disappeared before the exposure to virulent organisms and the child will react as if he had never been vaccinated; that is, in the example just described, the child will have responded to the virulent infection by the tuberculin test becoming spontaneously positive again, without definite clinical evidence of disease. However, it does happen occasionally that clinical primary tuberculosis develops in a child vaccinated a long number of years previously. In such cases the result of frequent tuberculin testing has shown that the tuberculin allergy has disappeared. On the other hand, no tests may have been carried out and, therefore, the disappearance of the tuberculin allergy may only be presumed.

#### PRIMARY TUBERCULOSIS AND BCG VACCINATION

The diagnosis of primary tuberculosis in a BCG vaccinated child is often very difficult. A known possible source of infection is useful. There may be little or no evidence of hilar adenitis. The sedimentation rate may be only slightly raised and come back to normal fairly rapidly. The disease in the majority of such cases (and they are extremely few compared with the number of children who must get a superinfection) is both mild and of shorter duration than in the non-vaccinated child. The risk of complications (miliary spread and meningitis) is minimum. The isolation of virulent tubercle bacilli from gastric washings would, of course, clinch the diagnosis.

In the years 1945-1949, inclusive, 1,352 vaccinated and 7,279 non-vaccinated children were admitted to Norrtull's Hospital. Of the non-vaccinated, 599 ( $8.2 \pm 1.0$  per cent.) had tuberculosis, compared with 26 ( $1.9 \pm 0.14$  per cent.) in the vaccinated group. During this period nine patients, who had been vaccinated during the incubation of primary tuberculosis, were admitted. There were 28 cases of tuberculous meningitis and/or miliary tuberculosis in the non-vaccinated groups and none in the vaccinated group. Among the 26 children with primary tuberculosis, 12 were mild and the remainder had more or less typical disease; some of the latter had been vaccinated so long before that it is very likely the immunity had disappeared. In addition, there were 137 cases with spontaneously positive tuberculin tests.

The difference in the number of vaccinated and non-vaccinated is surprisingly great and is not representative of the children of Stockholm to-day. There has been a very great increase in the number of vaccinations during this five-year period, so that the proportion for 1949 is quite different from 1945. Parallel with this rise in the number of vaccinated children there has been a fall in the number of cases of primary tuberculosis—from 183 admissions in 1945, to 88 in 1949, with an



average of 123 in the intervening years. This has resulted in more beds than patients—one of the most striking things in visiting a Swedish children's sanatorium.

#### CONCLUSION

Since the introduction of BCG vaccination into Sweden twenty-five years ago it has become accepted and is universally used by all physicians. The importance of accurate tuberculin testing and a strict technique for the vaccination is emphasised. The aim of BCG vaccination is stressed. The part played by the general practitioners in the diagnosis of the initial fever stage of primary tuberculosis and the vaccination of all tuberculin negative people is described. A comparison of tuberculosis in vaccinated and non-vaccinated children, admitted to Norrtull's Hospital during the past five years, is given. The absence of tuberculous meningitis and miliary tuberculosis in the vaccinated group is pointed out. Of the 623 cases of primary tuberculosis, only 26 ( $4.2 \pm 0.6$  per cent.) had been vaccinated.

I wish to thank Professor Wallgren, Docent Vahlquist and the other members of the staff for their kindness and help since my arrival in Stockholm.

This description of Calmette vaccination was written during the period of tenure of a "Cow & Gate" Travelling Scholarship from the Department of Child Health, Queen's University, Belfast.

#### REVIEW

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While most, if not all, of the many minor errors and misprints of the seventh edition have been eliminated, some new ones have crept in, which, to the junior student at least, must constitute a possible source of confusion. For example, the legends on fig. 635 pointing to the areas of insertion of the mylo-hyoid and thyro-hyoid muscles now reverse the true positions of these muscle attachments; an error even more misleading than that in the previous edition in which two areas of insertion were shown for the mylo-hyoid and none was shown for the thyro-hyoid muscle.

The decision to eliminate from the text of the seventh edition the embryological and histological descriptions previously given throughout the book with each of the sub-sections, and to replace this scattered, but nevertheless very useful and usefully placed material, by a single chapter on the general growth and development of the body, has again been adhered to. This chapter is well worth reading, not only by the undergraduate and the candidate for a higher qualification, but also by the general practitioner and the specialist who has any dealings with children. The debt to the late Professor Symington of this school for much of our still scanty knowledge of the anatomy of childhood is acknowledged, but I think that all readers of this chapter must, in their turn, be very grateful for the brilliant way in which Professor Wood Jones has incorporated that material, and much of his own gathering besides, into a very readable compass and for the many interesting correlations and suggestions which he has made.

The remainder of the book is devoted to gross topographical anatomy, which is dealt with regionally in the clear, concise manner always associated with this handbook, and so admirably suited to the needs of students.

W. R. M. M.

average of 123 in the intervening years. This has resulted in more beds than patients—one of the most striking things in visiting a Swedish children's sanatorium.

#### CONCLUSION

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# BCG

By HONOR M. PURSER, F.R.C.P.I.

## *A Paper read to the Ulster Tuberculosis Association*

### 1. SYNOPSIS OF HISTORY

I HAVE been asked to speak to you this evening on the subject of B C G vaccination against tuberculosis. For the benefit of those of you who are not familiar with the story, I thought I would give a brief outline of the history of the development of the vaccine.

The vaccine is named after Calmette and Guérin, the two pioneer workers in this field of medical research.

In 1908 Calmette started to grow this particular strain of bovine tubercle bacillus on special culture media. The research continued for many years, using different experimental animals to determine first, the harmlessness of the vaccine and second, to test its immunizing powers. Finally, in 1923, the plunge was taken and the vaccine was given by Weill Hallé to newborn infants in Paris. At this period it was given by mouth dissolved in milk.

These years of experiment were required in order to produce a vaccine that is stable in virulence and potency, one that is incapable of producing a progressive tuberculous disease and at the same time has the power of conjuring up the protective mechanisms of the body, so that a possible future virulent infection will be successfully resisted.

The vaccine used in every country is grown from the original strain of Calmette bacillus obtained from the Pasteur Institute in Paris, but the subsequent methods of culture vary in different countries as do the technique of vaccination and the dosage prescribed.

In France the organism, originally cultured on bile potato, is now grown on Sauton potato in Roux tubes, and it is administered in the main by the scarification method. In Russia the vaccine is still largely given by the oral route, while in Norway, the multiple puncture method of administration is favoured by some workers. The administration of the vaccine commenced in Scandinavia in 1925 and about a year later Heimbech of Norway was the first to try giving the vaccine by subcutaneous injection, but this method was soon abandoned, as it gave too high a proportion of local abscesses. In 1928, Dr. Wallgren of Sweden introduced the intradermal technique for B C G inoculation, which is now the standard method used in the Scandinavian countries and the one which has been adopted here in Northern Ireland.

The vaccine used by the Northern Ireland Tuberculosis Authority is supplied from the State Serum Institute in Copenhagen and is delivered by air mail once a week to Belfast.

## 2. LABORATORY TECHNIQUE IN PRODUCTION OF B C G

In Denmark the laboratory concerned with the production of this vaccine, is a self-contained unit and is kept solely for this purpose; no other type of tubercle is allowed into the laboratory and a most rigorous control is kept on all the personnel. These precautions are now taken in all laboratories manufacturing B C G and are enforced to prevent any possible repetition of the Lübeck disaster, when a virulent culture of human bacilli was accidentally mixed with the vaccine.

Originally, as I have said, the culture medium was bile potato which had the power of fixing the virulence of the bacilli at a very low level. Nowadays in Denmark the strain is kept on a synthetic fluid medium called the Sauton medium and is transferred regularly from one culture flask to another every ten days. Sauton consists of an amino-acid, asparagine, various salts and glycerine. It is diluted 1 in 3 with redistilled water. This medium is preferred for growing B C G as its constituents are not variable quantities like beef or broth or egg.

The keeping of the strain, so that the potency of the vaccine remains constant, demands great experience and skill and even with this it has shown some variations, but only within certain limits. These variations are noted and controlled by animal inoculations, carried out on a special breed of white guinea-pig, from each batch of vaccine manufactured. The results of these investigations are not known, of course, until months after that particular batch of vaccine has been used, but this is not considered to be of great practical importance as variations in potency of the vaccine and vitality of the organism only occur very gradually.

For the preparation of the vaccine, Calmette bacilli are grown on Sauton medium and form a thick, crinkled, cream-coloured veil on the surface of the fluid. A fourteen-day-old culture is used. The flask containing the culture is shaken vigorously and poured into a Birhaug apparatus from which the dried culture mass is extracted and weighed. After this it is placed in a large bottle and emulsified by shaking up with stainless steel pellets so that the bacilli lie almost individually and not in clumps. By adding fluid, a suspension is made so that it contains 1/4 mg. B C G culture in 1 c.c. of fluid. Diluted Sauton medium is used as the diluent.

The vaccine is manufactured once a week, but not packed or released from the laboratory until the results of two sets of sterility tests become available. Tests for aerobic organisms are made on blood agar plates and for anaerobic organisms, on half solid agar with and without hæmoglobin. Any contaminated flask is destroyed forthwith. The vaccine is thus six days old before it leaves Denmark and it is intended for use within fourteen days of manufacture, as after this its efficiency deteriorates rapidly. The optimum temperature for preserving the vaccine is 2°-4°c., and once the vaccine is delivered in Belfast it is stored in a refrigerator used solely for this purpose and kept at this temperature. The vaccine is made up in sealed glass ampoules of 1 c.c. and 5 c.c., enough for ten and fifty cases, respectively, and each batch has a separate number and is stamped with the date of manufacture. There is hope of producing a dried vaccine, in the near future, which can be diluted before use and which will keep indefinitely.

### 3. SELECTION OF CANDIDATES FOR B C G

At the present time it is not possible to offer B C G to everyone who wants to have it and special groups of the population are being selected for priority treatment.

These groups include :—

- (a) Newborn babies of tuberculous mothers, born in maternity hospitals and isolated at birth from their source of infection. As a placental infection is a medical curiosity this has the advantage of eliminating all pre-vaccinal testing and isolation. The disadvantage lies in the fact that infants have poorly developed powers of producing anti-bodies and take a long time about it, so that a considerable proportion require to have the vaccination repeated and the successful cases take eight to twelve weeks to effect conversion, which places a considerable strain on the cot situation in the maternity hospitals. I would like to say at this point, how extraordinarily co-operative both the Jubilee Hospital and the Royal Maternity Hospital have been in affording us this facility.
- (b) Non-infected child or adolescent contacts of cases of tuberculous disease.
- (c) Young adults subjected to special risk of infection, e.g., nurses, medical students and social workers, laboratory technicians, etc.

As the scheme expands, we hope to be able to offer vaccination to all who are suitable and who wish to avail themselves of the protection.

### 4. SIGNIFICANCE OF TUBERCULIN TESTING

Tuberculin is the toxin of the tubercle bacillus and was isolated by Robert Koch in 1889. This substance gives a reaction when it comes in contact with the skin and tissues of someone who has been infected by tubercle bacilli, but no reaction in those who have not been so infected.

A number of well controlled surveys have demonstrated that the reactor to tuberculin has a higher resistance to tuberculosis than the non-reactor, when the general resistance is the same and if two groups of the population are taken one composed of reactors to tuberculin and the other of non-reactors, living under the same conditions and equally exposed to tubercle bacilli, five times as many cases of tuberculosis will develop amongst the non-reactors as among the reactors.

Herein lies the rôle of B C G, for if the skin sensitivity is co-existent with the development of specific immunity (which is generally but not universally admitted) then with B C G one can protect a previously non-infected person by giving him a controlled infection, without any risk of acquiring the disease, but providing at least as good protection as that afforded by a naturally acquired infection.

### 5. CHOICE OF TESTS AND TECHNIQUE OF TUBERCULIN TESTING

Prior to vaccination the routine procedure of tuberculin testing adopted by the Northern Ireland Tuberculosis Authority depends on whether the candidate for vaccination is a child or an adult, as in the former we try to avoid any unnecessary injections.

In children under twelve years we do a preliminary skin test with Allen & Hanbury's tuberculin diagnostic jelly, using an abrasive on the skin and cleaning the area with acetone first. If this test is negative, X-ray examination and checking of contacts is completed and then three days before the proposed date of vaccination, we do an intradermal tuberculin test with Mantoux 1/100 giving 1/10 of a c.c. into the outer aspect of the middle third of the left forearm. This test is read in 72 hours and if negative the child is vaccinated immediately.

In the case of adults and older children the preliminary test is done with 1/10,000 Mantoux, if the result is unequivocally negative in seventy-two hours, we go straight to 1/100, if the result is doubtful we do an intermediate test with Mantoux 1/1,000.

If, for any reason, it is impossible to read tuberculin tests after three days, it is preferable to read them on the fourth rather than the second day, as non-specific reactions have not always subsided in forty-eight hours and these may confuse the interpretation of the result.

We try also to remind ourselves that tuberculin testing should not be carried out when there is an epidemic of measles and whooping-cough about, as these infections, and possible others, also, have the power of temporarily changing tuberculin positive reactors into tuberculin negative ones.

On occasions we have to ask the patient's general practitioner or district nurse to read and report on the results of tuberculin tests, when this occurs we tell him that to read a tuberculin jelly test as positive, vesticulation must be present and to read an intradermal test as positive, there must be œdema or induration present to a minimum diameter of 6 mm. Redness alone does not constitute a positive reaction in either case.

## 6. TECHNIQUE OF CALMETTE VACCINATION

Now to come to the vaccination itself. As I have explained, vaccination is only given to people with negative tuberculin tests to a strength of Mantoux 1/100, the final test being made three or four days before the proposed date of vaccination.

At this point I would like to say that no actual harm is done if tuberculin positive case is inadvertently vaccinated, but it may give rise to unpleasant local reaction at the site of vaccination which would discourage people from being vaccinated and would certainly discourage me from being a vaccinator. I have not yet had personal experience of a Koch's phenomenon, but a colleague working with B C G described a case to me, where the result was not only a local reaction in forty-eight hours resembling an acute cellulitis, but also a temperature of 105° F., with meningeal symptoms and a widely distributed urticarial rash.

Another reason for taking all precautions to avoid vaccinating tuberculin positive reactors is that some of these cases might be latent or early cases of tuberculosis, and if the disease manifested itself soon after the administration of the vaccine the vaccine might be blamed for producing the disease.

Instructions for use :—

The vaccine must be vigorously shaken before use, as it is necessary to have the bacillary particles evenly distributed in suspension.

The vaccine is given intradermally into the left deltoid region. A successful injection shows a bleb about 10 mm. in diameter with indentations for the hair follicles. If this does not occur the injection has been given too deeply. The dose is  $1/10$  of a c.c. except in infants under six months, when two injections of .1 c.c. are given, e.g.,  $1/10$  c.c. into each deltoid region.

No antiseptic is used on the skin. Any solution left over in the ampoule is destroyed at once.

The syringe used for the vaccination is the ordinary 1 c.c. tuberculin syringe, divided into tenths. The needles used are No. 20 short bevelled platinum-iridium needles, if we are lucky enough to get them.

The syringes and needles used for B C G are sterilized by boiling and must not be used for tuberculin testing or any other purpose, as the vaccine adheres to the surface and is not removed by sterilization.

#### 7. POST-VACCINAL CONTROL

In order to see if the vaccination has taken, a control should be made six to eight weeks after vaccination in adults and eight to twelve weeks after in children.

At this examination the site of vaccination is observed and a tuberculin test is made with tuberculin jelly in children under twelve years and with Mantoux  $1/1,000$  in adults and older children. If negative on reading at seventy-two hours, the test should be repeated using Mantoux  $1/100$  in both cases. An interesting point is that the tuberculin test after successful vaccination looks different from an ordinary positive reaction. The area is flatter and bluer in colour. If the individual gets super-infected subsequently the appearance changes to that of a natural tuberculin reactor. Further tuberculin testing will be carried out every year to see that tuberculin positivity is preserved and that the protection is being maintained. If the patient has reverted and is negative, re-vaccination is recommended.

Local reaction at the site of vaccination at six to eight weeks, measures about 5 mm. and there is usually a slight oozing from the centre which persists for about a week. A dressing with a gauze strip can be applied but bathing and all normal activities are permitted. A crust forms when the oozing stops, and finally there remains a small depressed white scar. Occasionally there is some enlargement of the supra-clavicular and axillary glands about two months after vaccination. There is no constitutional disturbance at any time.

Complications with the intradermal method of vaccination are, fortunately, rare but they do occur and include local abscess formation, and suppuration of the regional lymph glands, they occur most frequently in small children.

These complications clear up spontaneously after a time, though the lymph node may require aspiration and this may need to be repeated in about a month. Sometimes the vaccinated person does not report until the abscess has burst. If this

has occurred no special treatment is indicated and incision should be avoided; it must be remembered that these lesions heal by themselves and should not be considered or treated as ordinary tuberculous abscesses.

Hitherto it has not been our practice to make any mention about complications to prospective candidates for vaccination but I think it may be necessary to revise this policy, as otherwise the child or its parent may be unduly alarmed when they do arise.

#### 8. ISOLATION OF CONTACTS OF ACTIVE CASES

Special isolation precautions are taken when vaccinating contacts of active cases of tuberculosis.

In these cases a preliminary tuberculin test is made in the normal way. The person to be vaccinated is then separated from the source of infection for a period of six weeks, either by placing the patient in a sanatorium or by removing the vaccinee from the home. At the end of the six weeks pre-vaccinal period another tuberculin test is made and if this is negative, vaccination is performed immediately on reading the test. The vaccinated person must remain away from the source of infection until the vaccination has "taken" and the tuberculin test is positive, an overall period of about three months.

The precaution of pre-vaccinal isolation is to avoid giving B C G when the patient may be in the preallergic stage of an active infection, and that this is a very real risk is demonstrated by the fact that in our own small series of cases we have had nearly a dozen cases of natural conversion in the pre-vaccinal period. A couple of these I shall be discussing later in further detail.

The object of the post-vaccinal isolation is to avoid any chance of the individual contracting an infection before the specific protection given by the vaccine has had time to develop.

#### 9. DURATION OF PROTECTION AFFORDED BY B C G

A good many of our patients enquire how long the vaccination will remain effective in affording protection against tuberculosis and how complete the protection is.

The answer to the first question probably depends on whether or not the individual after vaccination comes in contact with active tuberculous infection. If this occurs the protection afforded by the vaccine will be constantly boosted and will probably last indefinitely. In the case of those who have no contact with infection after vaccination, it is believed that the protection will last for at least five years.

The figure has been based on an investigation at an island called, Bornholm, where bovine tuberculosis has been completely eradicated and where there are practically no sufferers from tuberculosis. Here a mass vaccination of the entire non-reacting inhabitants was carried out; in a follow-up examination five years later 90 per cent. of the population were still Mantoux positive.

In answer to the second question as to how complete is the protection afforded, I quote some of the claims for vaccination which came from Scandinavia.



## AIMS AND CLAIMS OF B C G VACCINATION

In over a million cases of successful vaccination, no case of tuberculous meningitis or miliary tuberculosis has developed in a child under two years, though the majority of cases have been in close association with infective disease after inversion.

In the Copenhagen district where systematic vaccination of non-reactors in tuberculous families is carried out, there has been no death from tuberculosis among vaccinated children. Infections, when they do occur, and whose tuberculous nature is confirmed by finding the causative organism are usually benign in character, and clear as quickly as though they were non-specific in origin, e.g. hilar adenitis or pleural effusion.

There is still a good deal of controversy about the amount of protection afforded against progressive pulmonary tuberculosis of human origin, but in Scandinavia it is considered to be at least as high as that conferred by a natural process of infection and the infection incidence is quoted as one-fifth of that which occurs in people who were not reactors though having had a primary exposure.

### 10. B C G EXPERIENCE IN NORTHERN IRELAND

Before finishing I would like to give a brief outline of our experience with B C G since the programme started about eight months ago.

|   |     |     |      |
|---|-----|-----|------|
| The number of vaccinations carried out is approximately   | ... | ... | 1000 |
| Number of people with completed post-vaccinal tests about | ... | ... | 800  |
| Number of non-convertors after B C G                      | ... | ... | 2    |

1. Newborn infant. No local reaction and negative to Mantoux 1/100 at six weeks. Re-vaccinated at nine and a half weeks, when still negative to 1/100. The second vaccination with a double dose was successful.
2. New-born infant. No local reaction and Mantoux 1/100 negative at six weeks. This child was removed to an orphanage outside Northern Ireland, and I have not pursued the case, as no further contact is anticipated with the tuberculous mother.

### NUMBER OF DELAYED CONVERSIONS

About a dozen cases mainly of the student age groups have been negative at six weeks to Mantoux 1/100 but strongly positive when tested about ten weeks after vaccination.

I think it is probably inadvisable to re-vaccinate under twelve weeks, especially if the vaccination site shows a typical "take".

### COMPLICATIONS

One small subcutaneous abscess 15 mm. in diameter in a child of four years, with ulcerated core 3 mm. in diameter with purulent discharge. Now healing satisfactorily.

Two axillary lymph nodes abscesses in children of two years and seven years respectively. Each required aspiration. The general health of the children was

excellent and pre- and post-vaccinal X-rays were normal. Healing is now occurring in both cases.

#### SEVERE REACTIONS

About six severe local reactions confined to members of two exceptionally dirty families. I attributed these sores to scratching and secondary infection and not to the vaccination. The sites were cleaned and covered with a dressing and the nurse visited within a fortnight and reported that the local reactions were healing satisfactorily.

About twelve cases altogether have had palpable glands in the axilla, typically placed anteriorly and up against the chest wall; when these are present they are clearly visible on X-ray.

#### GENERAL OBSERVATIONS

All vaccinations are done on a voluntary basis and a consent form, signed by the parent or guardian, is obtained for any vaccinee under twenty-one years.

The vaccination period necessitates a minimum of five visits:—e.g. preliminary test, X-ray, pre-vaccinal test, vaccination and post-vaccinal test. Subsequently, each case is to have an annual review with tuberculin test and X-ray. Any revertors will be offered re-vaccination.

For every vaccinated case, an average of four other members of the household are reviewed apart from the known source of contact. This involves X-ray in the majority of cases and sputum examination in a few. In all; explanation, exhortation, encouragement and reassurance.

#### NATURAL CONVERSIONS

As I have already said, about a dozen cases of natural "conversion" have occurred in the pre-vaccinal period:—

In each case the patient has been referred back to the clinic for observation.

In about half the cases there was X-ray evidence of disease and about the same number had some disturbance in health, only one case complained of a cough.

Several of them have been very instructive, notably:—

1. Male infant  $1\frac{1}{2}$  years. Examined at Central Tuberculosis Clinic, Belfast, in July, 1949. T. Jelly negative. Mother admitted sanatorium in November, 1949. Child admitted to a Children's Hostel 7/11/49.

T. Jelly negative 15/11/49. X-ray nil same month. Six weeks pre-vaccinal isolation to January, 1950, when Mantoux 1/100 was ++. Admitted to Sanatorium with Tb. meningitis same month.

2. Male infant 18 months. T. Jelly and Mantoux negative in October, 1949. Mother quiescent disease, but living in house with infectious case. Vaccination deferred till seven weeks after removal to new home, and when called up Mantoux 1/100 was strongly positive.

3. Male child,  $4\frac{1}{2}$  years. T. Jelly negative October, 1949. Infectious uncle out on pass from sanatorium at Christmas. Mantoux 1/100++ at pre-vaccinal test in January, 1950. X-ray shows suspicious shadowing at left base.

4. Two nurses repeatedly negative to Mantoux 1/100 isolated from tuberculous wards for pre-vaccinal period and found Mantoux 1/100 positive at pre-vaccinal test. No X-ray lesion, or disturbance in health in either case.

The response to the B C G campaign has been very gratifying and in addition to contact cases, there have been numerous enquiries from institutions, factories and schools. It would seem that expansion of the programme will be limited only by our powers to expand.

Two developments most urgently required are increased X-ray facilities and isolation for contacts of active cases during the vaccination period.

The technique of vaccination does not present any obstacle, but I think judgement is required in deciding whom to vaccinate and when to vaccinate them. Moreover, it must always be borne in mind by B C G enthusiasts, that vaccination against tuberculosis is a good servant and a bad master, and that unless the people who are vaccinated are assured that it is only one small factor in the preventive campaign against tuberculosis, it may do more harm than good, by engendering in tuberculous families a feeling of false security.

## REVIEW

RECENT ADVANCES IN SOCIAL MEDICINE. By A. C. Stevenson, B.Sc., M.D., M.R.C.P., D.P.H., Professor of Social and Preventive Medicine, The Queen's University of Belfast. J. & A. Churchill Ltd. 18s.

THIS book is a welcome addition to Churchill's well-known Recent Advances Series. In its 238 pages it deals in nine chapters with "a small group of subjects which appear to be of importance."

The first chapter, on "Measurement of Growth in Children," is a serious attempt to present briefly some aspects of anthropometry, and to assess to what extent the formulæ of Tuxford and others can be used to add or to check clinical findings. The author concludes that "no satisfactory method has been devised whereby states of nutrition can be assessed by anthropometry." In this chapter the meaning of the text is not always clear, e.g., on page 13 it is stated that, according to Tuxford, weight/height ratios increase by age roughly in harmonical progression; a few lines later we are told that height/weight ratios appear to advance in harmonical progression.

A quarter of the book, in two chapters, is devoted to the subject of infant mortality. A careful study of these chapters will well repay the student or the practitioner, and they can be commended in particular to the general practitioner. Indeed, the author has taken a great deal of trouble to collect much useful information and to present it in an attractive form.

Then follow chapters on day nurseries, the unmarried mother, problem families, and school medical inspection, which will readily commend themselves to all students of social medicine.

Chapter VIII, written by Dr. E. A. Cheeseman, Lecturer in Medical Statistics, Queen's University, Belfast, deals clearly with "Some Applications of Statistical Methods." It can readily be understood, even by non-mathematical readers.

The bibliography at the end of the chapters indicates how widely Professor Stevenson has studied the subjects discussed.

It is to be hoped that at no distant date the second edition of this book, in a much enlarged form, will appear, and will contain a similar clear account of a further group of important subjects.

J. B.

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J. B.

# Home Care for Sick Persons organised by Hospitals

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FOR a variety of reasons, which will be discussed later, a number of hospitals in the United States have organised programmes whereby patients suffering from chronic disorders are looked after in their own homes. The pioneer hospital and the one which has the most carefully studied programme is Montefiore Hospital in the Bronx, New York. It is proposed briefly to discuss the programme of that hospital and to endeavour to see what features of the "Home Care" activities organised by that hospital would be of value in Northern Ireland.

It should be made clear first that both in Montefiore and in other hospitals which operate such programmes, the original intention was simply to save hospital beds by moving patients to their homes and organising attention for them there when it had become clear that they were going to be sick for very long periods, perhaps for the rest of their lives.

That this aim has been achieved is clear, as will later be shown, but all those concerned with established schemes have in time become convinced by their experience that even if money were not saved, the programmes would be amply justified. This is because of the happiness and contentment of patients in their homes and of the remarkable efforts of their families to help them. The humanising and "individualising" of attention so difficult to attain in hospital are common happy results of home care. In addition, it appears that people in their own homes frequently make surprising recoveries, or, at least, retain function longer than would be expected in hospital.

The "man in the street" probably gives very little consideration to the purposes of hospitals, but regards them as places where very sick people are admitted. In practice, the factors which determine whether or not a sick person shall be admitted to hospital are numerous, complex, and interrelated, and it is by no means easy to estimate the importance of single factors in any given area.

It is difficult to find convincing figures indicating whether the overall average duration of stay in hospital is increasing or decreasing, but it is certain that the cost of hospitals, measured on the usual basis per bed per day, is increasing at an alarming rate. It is, therefore, important to note the factors which will influence the admission of patients to hospital, even if the relative importance of these factors cannot be assessed.

The new therapies, such as sulphonamides and anti-biotics, which help to control infectious conditions have modified the types of cases admitted and have diminished materially the mean duration of many infective conditions. However, over the period in which these therapies have been introduced, there have been parallel developments in the methods and techniques of investigations, and new methods of treatment of the variety of "replacement therapy" have been elaborated. These

latter procedures usually call for admission for investigation and adjustment to treatment of many persons who would previously have been investigated briefly and then discharged to "observation," so that the new diagnostic procedures have increased the pressure on beds.

The right of access to hospitals instituted by the National Health Service and the extreme pressure of work on family doctors have, no doubt, contributed also to increases in the numbers of persons seeking admission to hospital.

There are, in addition, a number of purely social factors which have influenced the demand on hospital beds. As the years pass, people are more willing to go to hospital, because hospitals are less feared and the thought of treatment more easily accepted. In many instances people seek admission to hospital or are advised to go there by their doctors, not so much because admission is necessary for diagnosis or treatment, as because of home circumstances and the difficulty of procuring nursing and domestic attention. It is probably not unimportant that hospital care is free, while help necessary to keep a patient at home is costly at the present time. It would seem, however, that poverty should not be a reason either for or against admission to hospital.

Finally, the distribution and type of illness in the population has been greatly altered by the reduction of wastage in the first two decades of life and by an increase in the absolute number of old people, so that there is an increasing relative and absolute amount of degenerative disease which is chronic and slowly progressive.

The factors of staff difficulties and the trend in favour of reducing bed congestion in old, overcrowded hospitals inevitably aggravate the problem and the numerous difficulties attending new construction do not offer much hope that new beds or hospitals provided in the next decade will relieve the problem entirely.

In essence, then, the problem facing hospital administrators at the present time is that diagnosis, treatment, and nursing care in hospital are extremely expensive. If equally good care can be given out of hospital or with fewer days in hospital, and if that attention can be given at less cost, then it would seem worthwhile making an attempt to provide a new kind of home care service.

Such a home care programme must not be envisaged merely as the making of arrangements whereby the existing duties of health and welfare authorities to provide nursing and domestic assistance are implemented fully. Nor must it be thought suitable only for the aged and infirm, and, therefore, it should be independent of (although collaborating freely with) a geriatric unit. Rather its function should be to provide, where social circumstances make it possible, for patients, who, unless this special service were available, would have to be treated in hospital because of the nature or severity of their illness.

#### THE MONTEFIORE HOME CARE PROGRAMME

In the Montefiore Hospital there is a Home Care Section under the direct administration of Dr. Martin Cherkasky, the Deputy Medical Superintendent of the Hospital. The staff consists of full- or part-time physicians, the equivalent of

the services of two full-time physicians being required. In addition, the entire staff of the hospital is available for consultations. At present some 160 patients are being looked after by Home Care who would otherwise be in hospital. There is one full-time and one part-time social worker and the part-time services of one supervisor of social work. There is one full-time occupational therapist and the part-time services of physiotherapists are available also. Nursing is provided by the Visiting Nurse Service of New York. Housekeeping services are obtained from the New York State Employment Bureau and from various voluntary agencies.

Patients accepted by the Home Care Section are still regarded technically as "on the books" of the hospital and their records are kept in exactly the same way as if they were in-patients. Most of the patients are transferred to the Home Care Section from a ward of the hospital, but some are referred from the out-patient department and some are accepted when referred by other hospitals in New York.

The procedure in the hospital is that the physician or surgeon in charge, having decided that the continuation of treatment or supervision in hospital is no longer advisable or necessary, notifies the Home Care Section of his opinion, using the proforma reproduced in the appendix to this note. On receipt in the Home Care Section, the patient is seen by a physician from that Section and, if he decides that the medical condition of the patient makes it reasonable to attempt home care, then he agrees to accept the patient, subject to agreement by the social service. The social worker sees the patient, the relatives, and, if necessary, the home, and decides whether there is a good chance of meeting the medical and social needs of the patient in his or her own home.

The grounds of acceptance by the medical service will clearly depend on whether the procedures necessary for comfort, diagnosis, and treatment can be carried out in the home, or at least with an occasional visit to hospital. At times a trial is considered well justified. The social worker's decision rests in the first place on whether the patient can afford proper medical attention in his own home. If he can, then he is not accepted, but referred to his own doctor. For the rest, the accommodation, sanitary condition, and equipment in the home are assessed. The decision finally rests on the relatives or friends in the home who are prepared to face the tasks of giving the patient such assistance as is required. It may be emphasized again that it will obviously be impossible for the physician or social worker always to be true prophets, and a trial will often be worthwhile.

After the patient goes home, a visit is paid by the physician and by the social worker as soon as possible. Arrangements are made for nursing and domestic assistance, as required. It may be noted that it is frequently found that relatives prefer (before or after experience) to assume nursing and domestic responsibilities, which in the preliminary stages have been adjudged too great by them or by the social worker.

The occupational therapist always visits the patient, and specialist consultations by the hospital staff are arranged as required. Apparatus and equipment are loaned from the Home Care Section or nursing organisations, and remedies are prescribed exactly as for a patient in hospital. Thereafter the patient is visited as often as is

necessary by the doctor and the other members of the team, and a wide variety of treatment and minor diagnostic procedures are carried out in the home. For example, dressings, replacement of self-retaining catheters, paracentesis, taking of specimens of blood, and blood transfusions are commonly performed by the doctor on his visits.

If a more complex investigation is required, the patient may be taken by ambulance to hospital for the purpose and either returned the same day or accommodated for a few days and then taken home. Examples of procedures necessitating such journeys to hospital are X-ray examinations, radiotherapy, minor operations, or metabolic studies. When a patient has improved, he may be discharged completely or he may be discharged to attend as an out-patient. If his condition indicates, he may be re-admitted to hospital and this is frequently done in cases of neoplasm before the patient's death.

The following quotations from the Second Annual Report of the Department of Home Care, Montefiore Hospital, may serve as a starting point for estimates of the cost of such a scheme in Northern Ireland :—

| EXPENDITURES ITEMIZED                   |     |     |     |     |             |
|---|-----|-----|-----|-----|-------------|
| Medical Services                        | ... | ... | ... | ... | \$16,940.33 |
| Home Care Executive                     | ... | ... | ... | ... | 2,749.95    |
| Clerical Services                       | ... | ... | ... | ... | 3,189.85    |
| Social Service                          | ... | ... | ... | ... | 4,268.96    |
| Occupational Therapy                    | ... | ... | ... | ... | 1,417.91    |
| Hospital and other equipment            | ... | ... | ... | ... | 2,414.26    |
| Medications                             | ... | ... | ... | ... | 4,283.19    |
| Laboratory                              | ... | ... | ... | ... | 311.47      |
| Transportation                          | ... | ... | ... | ... | 1,901.41    |
| Visiting Nurse Service                  | ... | ... | ... | ... | 3,995.27    |
| Housekeeping Aid                        | ... | ... | ... | ... | 3,286.90    |
| Physical Therapy                        | ... | ... | ... | ... | 3,152.02    |
| Administrative Expenses and Supplies    | ... | ... | ... | ... | 1,776.08    |
| <hr/>                                   |     |     |     |     |             |
| Total                                   | ... | ... | ... | ... | 49,687.60   |
| Total Funds for 1948                    | ... | ... | ... | ... | 68,274.86   |
| Expenditures                            | ... | ... | ... | ... | 49,687.60   |
| <hr/>                                   |     |     |     |     |             |
| Balance remaining as of January 1, 1949 | ... | ... | ... | ... | \$18,587.26 |

“\$49,687.60 represents the cost to the Department of Home Care for services to 197 patients during the year (several of the patients were on home care more than one time, but are here counted as only one patient). The total days of services rendered was 19,842. The average cost to the Department of Home Care was \$2.50. The cost has risen since last year, due primarily to (1) expanded service, and (2) to the increased cost of personnel.



“Two dollars and fifty cents was the actual amount expended per patient day. However, there are certain other costs which should be included if we take into account contributions made to the programme. First of all, the Visiting Nurse Service, in providing care for patients who were receiving welfare subsidies from the City of New York received part of the cost of the call (\$1.75 as opposed to \$2.25 for a full call) from the City. In addition, some of the patients had insurance policies which entitled them to a certain amount of nursing. These two items would total \$890.85, which should be added to the cost of nursing service. The City of New York also provided medications for those patients who were on the Welfare Panel. We estimate this would have cost the Department of Home Care an additional \$900. One other item which we were not required to pay for, due to the generosity of Montefiore Hospital, was overhead, which would be a factor in any Home Care programme which might be contemplated. We figure this at eight per cent. The United Order of True Sisters supplied us with some of the equipment used in the home, such as bed trays, bedpans, etc., and with some of the dressings. (Including all these items, a corrected figure of \$2.82 per patient day is the net cost.) *The present cost of hospital care is more than \$13 per day*, so that it can be safely said that Home Care is less than a quarter of the cost of hospital care. It must also be recognised that in Home Care costs, physicians’ fees are included and this represents about a third of our budget. These fees are not ordinarily considered in the cost per patient day for voluntary hospitals, since most physicians’ services are rendered free of charge in such institutions. The question arises whether the cost per patient day on Home Care should be compared to the cost per patient day in the hospital.

“We estimate that about 85 per cent. of the patients who are on the Home Care programme at this time would have to occupy a hospital bed were it not for our ability to care for them at home.

“We have increased Montefiore Hospital’s capacity without costly construction, and at a cost per day which is about one-quarter of hospital cost. We have found that suitable patients can be taken care of at home with great benefit to them. We have accumulated a great deal of information on the social aspects of long-term illness.”

An excerpt from the editorial on Home Care appearing in the *American Journal of Public Health*, February, 1949, might be very pertinent here in view of the expanding interest in Home Care.

“This programme makes a contribution of major importance to the technology and to the philosophy of medical care. It is essential, however, to remember that its successful application depends on a high degree of hospital development, in the fields of medical staff, social service, nursing, physical and occupational therapy. It would be most unfortunate if so fruitful a concept were to be discredited by wholesale displacement of patients by hospitals which lack the facilities to carry the programme out successfully.

“There are many human interest stories which can be told about patients, who, having come on Home Care and been seen in their family settings, have been helped almost miraculously by the combined skill of our doctors, our

social workers, and our public health nurses, along with the ancillary services. The doctors on the programme have developed an insight into the rôle that social factors play in disease, and, in view of the increasing importance of long-term illness in our society, such understanding is of great importance.”

#### APPLICATIONS IN NORTHERN IRELAND

There are one or two local circumstances in the United States which differ quite radically from those pertaining here and which render even a trial of Home Care on precisely the same pattern impossible. The fundamental difference is that every person here has a family doctor and has no financial worry about doctor's fees. The family doctor has already had too many of his responsibilities removed and he must come into any scheme which proposes to offer hospital facilities to any of his patients in their own homes.

The vast majority of general practitioners are already fully occupied and it is unreasonable to expect them gratuitously to offer their services for the heavy and trying work of looking after chronic invalids in their own homes, if by expressing an opinion they can be cared for in hospital. The rigidity of the per capita system of payment in this, as in many other ways, reacts very unfavourably on the initiative and will to constructive work of general practitioners. However, it seems reasonable to expect that in every area there will be found a number of practitioners who would be willing to take an active part in a hospital Home Care scheme, but it would be quite unreasonable to expect them to do so unless they were paid for the onerous extra duties to which they would commit themselves.

It would appear that, properly used, such a Home Care scheme could be utilised as an instrument of “rehabilitation” of the practitioner, rather than as a further curtailment of his responsibilities and prestige in the community. It might well be one way of giving the practitioner a useful hospital connection, and it would seem that if hospitals were saving beds and expense by arranging for medical attention of a hospital standard in the patient's home, they would be amply justified in paying the practitioner who gave much of the medical attention to the patient.

Then there are the two committees and their servants of the local authorities who have statutory duties in this field of Home Care—the Health Committees and the Welfare Committees. Any scheme would be ill-starred which failed to take into account the duties of these bodies and the organised nursing and domestic services which they can provide. There are other public authorities whose co-operation would also be essential: Education Committees, the Assistance Board, and the Ministry of Labour are examples. There are also interested voluntary bodies.

Nevertheless, such a scheme stems from the hospital; its justification lies in the possibility of providing as good or better medical care than the patient would get in hospital, and therefore the control should lie ultimately in a Home Care section of a hospital. The principle of having patients under Home Care “on the books” of the hospital and regarded as patients receiving active treatment from the hospital seems most valuable and should be retained.

## A SUGGESTION FOR DISCUSSION FOR A TRIAL HOME CARE SCHEME

Clearly, supposing that a trial is deemed advisable, there would be many difficulties to be overcome in getting it under way. There are many considerations which would influence the organisation of such a trial. Nevertheless, I venture to make suggestions in the hope that this outline serves only as a starting point for discussions which would carry the possibility of a trial much further and which would finally launch a trial on sound lines.

### A HOSPITAL FOR A TRIAL RUN

Supposing the trial could be begun in one hospital, then the following considerations affecting the hospital chosen would be important. The hospital would require to be large, so that the number of suitable cases would justify the trial. For similar reasons, the hospital would have to provide a considerable number of cases of chronic or long-term illness. The consultant staff of the hospital would have to agree to act as consultants in the scheme and to co-operate by putting forward suitable cases and by accepting the final decision of the Home Care section. Finally, in the area served by the hospital there would have to be a group of practitioners who would be willing to participate in a trial.

### A HOME CARE SECTION

In the chosen hospital it would be necessary to set up a Home Care Section, headed by a physician on the staff of the hospital. There would also have to be one full-time qualified almoner and probably a nurse (preferably with Health Visitor's qualifications). The part-time services of an occupational therapist would also be required.

It is suggested that the rest of the medical staffing should in the first instance be by a group of general practitioners known to send a considerable number of their patients to the hospital. They would act in respect of their own patients only. Finally, transport for the staff is an absolute essential and the scheme would fail if this were not provided.

Patients would be accepted for Home Care on agreement of the permanent staff in the first instance, always with the approval also of the general practitioner of the patient. In time, it would, no doubt, be possible to find general practitioners who were willing and able to see their own patients in the wards, when they had been proposed for Home Care, and to make the medical decisions as to acceptance.

It does seem very important, however, that the decision as to suitability should primarily be determined by whether the patient's medical needs can be met in the social circumstances existing in his home and, therefore, the decision as to a trial of Home Care should rest ultimately with the Home Care Section of the hospital, in consultation with the patient's family doctor.

### REFERENCE

Montefiore Hospital, New York City: "Home Care"; 1947.



# Isolated (Fiedler's) Myocarditis

By PROFESSOR J. HENRY BIGGART, C.B.E., M.D., D.SC.

## *A Paper read to the British Cardiac Society*

THE following three cases appeared of general interest, as the essential findings at post-mortem were limited to a peculiar form of myocardial degeneration, of which we can find no record in British literature.

### CASE I

The patient, D. L., was a female child aged 22 months. On 4/11/43 she had what her mother described as a "choking turn," following which she held her left arm limply and stumbled when attempting to walk. She appeared to be in pain, held herself rigid, with her head retracted, and rolled her eyes from side to side. She did not develop any increase in temperature. On the next day there was twitching of the left side of her face and she refused to use her left arm. Following this her condition appeared to improve.

On examination at this time (15/11/43) the deep reflexes were present. There was a positive Babinski on the left side. The pupils were equal and reacted normally, and the fundi were normal. No lesions were noted in the throat, heart, or lungs. There was some weakness of the left hand.

On 13/11/43 she developed swelling of her face. Swelling of the feet and ankles was seen on 17/11/43 and continued until death.

On 26/11/43 the urine contained sugar, but no albumen. The next day it showed albumen, but no sugar. The oedema gradually increased and there was some increase in the rate of respiration. Respiratory distress, cyanosis, and rales at the base of each lung led to a diagnosis of cardiac decompensation of unknown origin.

Apart from the fact that she was given an immunising course of combined A.P.T. and whooping cough vaccine on 23rd and 30th September, 6th October, and 3rd November, 1943, there was no previous history of any illness. The child was in good social circumstances, fed well, and appeared healthy until the sudden episode on 4/11/43. Death occurred on 6/12/43.

*Post-mortem, A. 3910 (Dr. J. E. Morison).*—The following essential findings are abstracted from the post-mortem report.

The body is that of a very well-developed, well-cared-for female infant. There is a slight icteric tinge of the conjunctiva. Oedema is present in both ankles and over the sacrum. There is no enlargement of the superficial lymph nodes. On incision, there is considerable oedema of the anterior abdominal wall.

*Body Cavities.*—The pleural sacs contain about 3 oz. of clear fluid. The

pericardial sac contains 2 oz., whilst the peritoneal cavity is distended with over 1½ pints. There are no adhesions.

*Heart.*—This is large, relative to the size of the child. The epicardium is smooth and translucent. The right auricle is dilated. The tricuspid valve is thin and delicate. There is no hypertrophy of the right ventricle, but the myocardium is pale. The left auricle, mitral, and aortic valves appear normal. The ventricular muscle has a rather peculiar appearance. It has a pale pink appearance, which has a waxy translucency. Though obviously altered, the muscle has a firm, rigid texture. The endocardium shows a small ante-mortem thrombus in the left ventricle.

*Lungs.*—The pleura is smooth. There is no enlargement of the lymph nodes at the hilum, and the large bronchi are normal. There is an area of consolidation at the lower posterior part of both lungs. These areas stand out above the level of the adjacent pleura. On section, the alveolar tissue is œdematous, but the areas of consolidation are seen to be almost black in colour from effused blood with loss of alveolar pattern. They are sharply demarcated from the adjacent lung tissues and appear to be infarcts.

*Liver.*—The liver is enlarged. On section, it presents a strongly contrasting pattern of congested areas alternating with pale yellow areas.

*Spleen.*—This is about twice the normal size. It is firm and the cut edge remains sharp. The pulp is a dark-red colour and the essential structure is poorly seen. Apart from a marked degree of retro-peritoneal œdema, no other lesions were seen in the thoracic or abdominal viscera.

*Brain.*—The meninges appear normal. The organ feels soft, especially over the right cerebral hemisphere. There is no tentorial herniation, but the vessels over the right hemisphere are somewhat congested. On section, the convolutions in the region of the right Rolandic fissure show softening. The normal tissue is replaced by a yellowish soft tissue with cystic spaces. No other lesions were seen.

*Microscopical Examination.*—Sections of all organs were examined. The lungs, brain, and pancreas show no lesions, apart from those due to congestive heart failure.

*Heart.*—This presents a very varied and unusual picture. Focal areas of recent necrosis are seen. The capillary bed in such foci is generally dilated, but there is no inflammatory infiltration. Adjacent muscle bundles are vacuolated and their fibrillar arrangement indistinct, so that they acquire a rather homogeneous structure. This vacuolisation does not appear to be due to fatty change, for the specific stains show very little stainable fat. It, therefore, probably represents some form of hydropic change.

In other areas the necrotic muscle has disappeared, leaving the stroma collapsed. The nuclei of some of these disintegrated muscle fibres appear to survive. There is a slight proliferation of fibroblasts, but generally the fibroblastic reaction to the muscular lesions is surprisingly slight.

There is little inflammatory infiltration. A few lymphocytic aggregations are seen in the epicardial fat, but, in general, the myocardium is free from infiltration.

The coronary arteries and their branches in the numerous sections examined are

free from disease. The capillary walls in the necrotic areas show swelling of their endothelium, but this appears to be the result, rather than the cause, of the necrosis.

*Lungs.*—The alveoli show much œdema and scattered aggregates of large mononuclears containing hæmosiderin. There is much distension of the alveolar capillaries. Several sections show necrosis of the alveolar walls, with extravasation of red cells and fibrin into the alveoli. A branch of the pulmonary artery contains an organising thrombus.

*Pancreas.*—There is œdema of the interstitial tissue and an infiltration of the stroma with lymphocytes. This infiltration is much more intense than that seen in the heart. The acinar and islet tissue cells show no lesions, and the ducts are patent.

*Brain.*—The area of softening is found to be infiltrated by cerebral histiocytes, many of which are filled with phagocytosed fat. There is no inflammatory infiltration and the glial cells show only early signs of proliferation.

*Anatomical Diagnosis.*—Isolated myocarditis (Fiedler); intraventricular thrombi; infarcts lungs and brain; subacute venous congestion of lungs, liver, spleen, kidneys, and adrenals; lymphocytic infiltration of pancreas.

## CASE II

The patient, a farm labourer, aged 16 years, was admitted to the Royal Victoria Hospital on 18/5/43 as a case of nephritis. He gave a history of having suffered from scarlet fever in 1935, but there were no known complications. He had been subject to frontal headaches since childhood. He had been fit for his work on the farm and complained of no symptoms until three weeks before his admission, when he began to suffer from shortness of breath. However, he continued at work until 9/5/43, when, following a wetting, his face, legs, and ankles became swollen and he complained of pain in his ankles and calves.

Examination showed him to be a well-nourished youth, with no anæmia, but slight cyanosis and puffiness of the face. His legs and ankles were œdematous. His heart was enlarged, the apex beat diffuse, and in the fifth space half an inch outside the nipple line. The pulse was 160-180. Blood pressure was 115/90 and a mitral systolic murmur was present. The lungs showed dulness at the right base. There was free fluid in the abdomen and the liver appeared enlarged. The urine was scanty and showed albumen ++ but no casts. A catheter specimen was sterile. Blood urea 18 mg. per 100 cc. The electro-cardiogram showed a 2—1 auricular flutter, and X-ray confirmed the presence of cardiac enlargement.

His general condition improved under digitalis treatment and there was some diminution of the œdema. The pulse rate, however, remained high. The degree of albuminuria varied and was absent for a few days. The urinary output increased from 12 to 15 oz. per day to 40 to 70 oz.

As the tachycardia and auricular flutter continued, he was given a course of quinidine, starting with gr. V on 20/6/43 and increasing by 5 gr. daily up to 20 gr. per day. On 26/6/43 his pulse rate fell to 32 and his general condition became worse. He felt uncomfortable and was very dyspnoëic. Electro-cardiographic examination showed heart block with right ventricular extrasystoles. The quinidine

was stopped and digoxin given. His pulse rate mounted again to 160 and he continued in much the same condition for the subsequent two months. On 18/8/43 he complained of precordial pain and a pericardial friction rub was found. He occasionally showed a slight evening rise of temperature. Blood cultures were sterile on three occasions. The urine continued sterile. Leucocytes were 9,500 per c.mm. Signs of congestive heart failure gradually became prominent. The œdema became generalised and he died on 27/10/43, having been semi-comatose for three days. Some degree of terminal jaundice was noted.

*Post-mortem, A. 3856.*—Only the essential findings are abstracted.

*Body Cavities.*—The pericardial sac contains 4 oz. of amber clear fluid; the peritoneal cavity 2 pints. Both pleural cavities are normal.

*Heart.*—This is enlarged—weight  $19\frac{1}{2}$  oz. It is globular in shape and flabby in consistency. All four chambers are dilated. The tricuspid and mitral valves are rendered insufficient by dilatation of the valve rings. The pulmonary and aortic valves are normal. The myocardium is uniformly pale. At the apex of the right ventricular cavity and extending for one inch up the septum is a firm ante-mortem thrombus. Beginning about one inch below the mitral valve ring a thrombus is firmly attached to the left ventricular wall. This extends down to the apex and along the septum to within two inches of the aortic valves. It varies in consistency, but parts of it appear to be organised. The major vessels are patent.

*Lungs.*—The pleura is smooth. The lungs feel heavier than normal, and, on section, œdema fluid pours from the cut surface. There are several areas of infarction. The bronchi and major pulmonary vessels appear normal.

*Liver.*—Weighs  $3\frac{1}{4}$  lb. The gall-bladder is thin-walled and contains no stones. The cystic and common bile ducts are patent. On section, the lobules are distinct, and show dark-red central zones with relative pallor of their periphery, resulting in the granular nutmeg pattern of chronic venous congestion. There is slight bile-staining.

*Kidneys.*—Weigh 5 oz. each. The capsule strips readily, except over a small area in each kidney, where it is adherent to a bright yellow area in the cortex. The surface is smooth, and, on section, the cortical striæ are regular and the cortex of normal width. The medulla and pelvis show no lesions. Section of the yellow areas noted on the surface shows the presence of wedge-shaped infarcts.

*Spleen.*—Weighs 6 oz. The capsule is smooth. The organ feels firm and the cut surface maintains a sharp margin. At the upper pole there is a yellow area of infarction.

All the other organs appeared normal.

*Microscopical Examination—Heart.*—The mitral valve and valve ring show no lesions. Scattered through the left ventricle, but more particularly in the subendocardial zone and in the papillary muscles, are irregular areas of fibrous replacement of muscle. These areas of fibrosis vary in their cellularity, the majority being relatively acellular. Others, however, show the presence of many fibroblasts and the formation of new capillaries. More rarely a small mass of necrotic muscle is seen with a commencing fibroblastic reaction. In relation to these areas are small



foci of lymphocytic infiltration, but these are never numerous. A few such focal aggregates are also to be seen in the interstitial tissue unrelated to necrotic muscle or areas of fibrosis. Similar lesions are found in the right ventricular muscle and septum. Overlying the endocardium is an organising thrombus. The degree of organisation varies from field to field and seems to represent different age periods in the formation of the clot. There is no histological evidence of recent or healed rheumatism. The coronary arteries and their branches are normal. The muscle fibres which persist show little change. There is no fatty infiltration. Around the areas of scarring, scattered nuclei appear hypertrophied and an occasional muscle cell, with two large nuclei, is seen. The heart, therefore, shows lesions of at least three different age periods, a sparse lymphocytic infiltration, and a normal coronary system.

Histological examination of the other viscera merely confirms the presence of chronic venous congestion and infarcts in lung, spleen, and kidney. There is no evidence of nephritis.

*Anatomical Diagnosis.*—Isolated myocarditis (Fiedler); ventricular mural thrombi; chronic venous congestion; infarcts of lung, spleen, and kidney; terminal cardiac jaundice.

### CASE III

Admitted 1/4/45. Died 5/4/45.

The patient was a male, aged 48 years. He came to hospital complaining of shortness of breath over the preceding three weeks. For about one year he had noted palpitation on mild exertion and an increasing tendency to the development of fatigue. Before the present illness patient had felt quite well, and had not suffered from any illness of note.

On admission, the patient was seen to be pallid and moderately cyanosed. There was no clubbing of his fingers and no œdema. His pulse was 100, regular, and of poor volume and tension. Blood pressure was 105/85, rising later to 128/105. The apex beat was in the fifth space,  $4\frac{1}{2}$  inches from the midline. The heart sounds were faint, with a well-marked gallop rhythm. No murmurs were heard.

Crepitations were present at the lung base. The liver was slightly enlarged. The urine contained albumin. E.C.G. showed a left bundle branch block.

Throughout his stay in hospital there was little change in the patient's condition. Dyspnoea continued, and on 4/4/45 there was hæmoptysis. Enlargement of the liver became more marked. The pulse became weaker and the patient died on the morning of 5/4/45.

*Post-mortem, A. 4521* (only the essential findings are abstracted).

*Heart.*—This is enlarged. Weight 20 oz. It is globular in shape. The epicardium is smooth and there is no excess of fluid in the pericardial sac. The right auricle appears normal. The tricuspid valve ring is dilated, but the valvular cusps are quite thin and show no vegetations. Between the valve cusps and the ventricular wall is a laminated thrombus, extending almost completely round the valve ring. The ventricular muscle appears slightly thickened. The left auricle shows no thrombi, but its wall is thickened and it preserves its shape when cut open. The

mitral valve ring is dilated. The left ventricular endocardium is extensively covered by laminated thrombus. The muscle itself has a peculiar salmon-pink colour, but there is no gross evidence of an infarct. The coronary arteries are patent.

*Lungs.*—The pleura is smooth. There is no pleural effusion. The lungs feel heavier than normal. The major vessels and bronchi are patent. On section, the alveoli are seen to contain a rather hæmorrhagic œdema fluid. There are no infarcts.

*Liver.*—Weight  $3\frac{3}{4}$  lb. The gall-bladder is thin-walled and the cystic and common bile ducts are patent. On section, the lobular pattern is accentuated, due to the congestion of each central zone and the relative pallor of the peripheral zone. The radicles of the bile-ducts and portal veins are patent.

*Spleen.*—Weight 7 oz. The capsule is smooth and the organ feels firm. On section, the pulp is dark-red from congestion. The malpighian corpuscles are rather indistinct.

All other organs appear normal.

*Microscopical Examination—Heart.*—Numerous sections from all cavities were examined. The endocardium of both ventricles is seen to be covered by blood clot. Organisation is progressing, and is, in general, most advanced within the lumina of the thebesian veins. Over some areas the clot is completely organised. The underlying endocardium shows fibrous thickening and focal collections of cells, which are mainly lymphocytes, are present. Similar lymphocytic infiltrations, but of lesser intensity, are seen in the fibrous septa of the myocardium. Individual muscle fibres, more especially in the subendocardial layer, appear necrotic, whilst there are small scattered areas of fibrosis unrelated to blood vessels. The coronary arteries are patent. The intima is a little thickened, but nowhere is any atheromatous lesion seen of sufficient extent to be held responsible for the myocardial fibrosis.

Other organs show only the results of congestive heart failure. Unlike the other two cases, there are no infarcts.

#### DISCUSSION

Clinically, these cases resemble each other in showing a congestive heart failure with intermittent albuminuria and embolic episodes. There was nothing in their history or symptoms, however, to point to any possible ætiological factor which might have allowed of the more exact diagnosis of the condition. Pathologically, the lesions appear to be identical, though the longer duration of illness in cases II and III has allowed of the development of more fibrosis in the areas of myocardial degeneration. The curious fact emerges from the pathological examination that, apart from the presence of infarcts, there is no disease process in any other viscus or tissue which might be regarded as being associated with the myocardial lesion. Such "isolated" (Sellentin, 1904) myocardial lesions were first described by Steffen in 1888, but the name of Fiedler, who reported a similar process in 1890, is most often associated with the condition. Saphir (1941) stated that "one is justified in accepting the occurrence of isolated myocarditis in the sense of a more or less diffuse inflammatory lesion if every known cause for this type of myocarditis is ruled out, and if the myocarditis is found in the absence of any major pathological condition involving either the endocardium and pericardium or the entire body."

The present three cases seem, therefore, to justify the diagnosis of "isolated myocarditis."

Scott and Saphir in 1929 were able to collect thirty-six cases, and a cursory review of the literature shows that at least forty-one additional cases have been reported since that date. The disease, though rare, has therefore probably been often overlooked and is not so uncommon as the paucity of reports before Scott and Saphir's paper would seem to indicate.

Examination of the cases reported in the literature as examples of isolated or Fiedler's myocarditis indicates the probability that several conditions of different ætiology have been included under this term. Some authors stress the infiltration of the interstitial tissues of the heart by inflammatory cells. This infiltration may or may not be associated with necrosis of the myocardial fibres. Others (Jonas, 1939; Magner, 1939; Miller, 1935) describe a granulomatous type of lesion which apparently simulated the granulomata of tuberculosis, syphilis, or sarcoidosis. The lesion found in the present cases, however, appears to be essentially different from either of these types. Inflammatory infiltration is by lymphocytes, and nowhere are these present in large numbers. They tend to occur mainly in relationship to the areas of necrosis or repair and may well be a reaction to the products of the degenerating muscle rather than called forth in response to the presence of any bacterial or virus agency. It would appear, indeed, that the essential lesion is a degeneration of the muscle cell—a degenerative process which is peculiar in that, in some cells at least, it affects the sarcoplasm rather than the nuclei (Case I). Muscle nuclei occasionally survive in the degenerated area, and may proliferate to produce giant forms. The method whereby the degenerating muscle is removed is not easily apparent, for there is little evidence of phagocytosis. This degeneration of the myocardium is evidently associated with alterations in the endocardium, which favour the initiation of thrombi. In cases I and III there is well-marked endocardial fibrosis, and this is also present in Case II, though here the presence of subendocardial scarring suggests that the organising thrombi may be related to foci of necrotic muscle.

It would seem, then, that the cases reported as Fiedler's myocarditis are really heterogeneous in type and that no purpose is served in endeavouring to find for them a common ætiology. Cases showing a very similar pathological process have been reported by Gouley, McMillen, and Bellett (1937). These authors record four cases of cardiac failure occurring in pregnancy which showed foci of muscle degeneration, with a tendency to preservation of the muscle nuclei and subsequent fibrosis. Cases have been described in infants by Lindberg (1938), Mazzeo (1929), Maslow and Ledderer (1933), Singer (1932), Smith and Stephens (1938), and Bluhdorn (1924) and others. Under the title of "myocarditis perniciosa," Boikan (1931) described a similar case in a woman of 28 years. This author attempted to formulate a classification of isolated myocarditis, but beyond the recognition of acute and chronic and recurrent forms his classification adds little to our knowledge. In his report, Lindberg (1938) suggests that the lesion may represent the end stage of the beri-beri heart, and more recently this opinion has been supported by Smith

and Furth (1943) and Toreson (1944). However, there is little evidence that chronic beri-beri heart has produced a lesion similar to that under discussion, and Levy (1930) found vitamin B of no therapeutic value in similar cases. Remissions occurred without any vitamin therapy in the patients reported by Smith and Furth (1943). Whilst we have no detailed knowledge of the diet of Case I, the child was living in excellent social conditions and at autopsy appeared well nourished. Case II, the farm labourer, was on regular hospital diet for several months before death and showed both healed and acute lesions at post-mortem. Case III was of the artisan class, was not addicted to alcohol, and had the same rations as the rest of the community. Furthermore, the recent experimental work of Pantridge (1948) in this department has shown that in the pig thiamin deficiency results in necrosis and degenerative changes in the conducting system of the heart. Apart from some foci in the auricles, necrosis of myocardial muscle was not a feature of the deficiency. It is unlikely, therefore, that the myocardial degeneration was due to an avitaminosis. The occurrence of similar lesions in the heart in association with apparently allergic skin reactions has also suggested an allergic mechanism for the cardiac injury. Maxwell and Barrett (1934) report a case developing after a severe dermatitis, due to applications of a sulphur ointment. Brown and McNamara (1940) found a similar process in the heart following the administration of arsphenamine, whilst Nelson's case (1934) was associated with an exfoliative dermatitis. Against this interpretation must be urged the fact that in experimental sensitisation the cardiac lesions are much more akin to those seen in rheumatic carditis and polyarteritis nodosa, and the lesions so produced tend to involve the interstitial septa rather than the muscle tissue of the heart (McKeown, 1947).

The histological examination in these three cases does suggest a degenerative, rather than an inflammatory, lesion. The absence of any important incidents in the clinical histories of the patients renders it impossible to state what may have been the cause of this degeneration.

#### SUMMARY

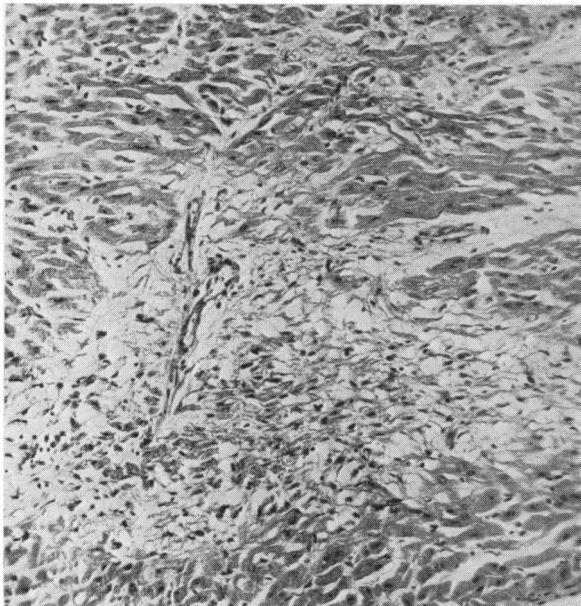
1. Three cases of heart failure in the absence of any of the usual cardiac lesions are reported.
2. In all three were areas of myocardial degeneration, with various stages of scar formation and fibrosis of the endocardium.
3. Mural thrombi were present in the ventricles and had produced infarcts.

My thanks are due to Drs. Boyd Campbell and F. M. B. Allen for the clinical notes, and to Drs. J. E. Morison and Y. MacIlwaine for post-mortem reports on two of the cases. I am indebted to Mr. D. Mehaffey, A.R.P.S., for the illustrations.

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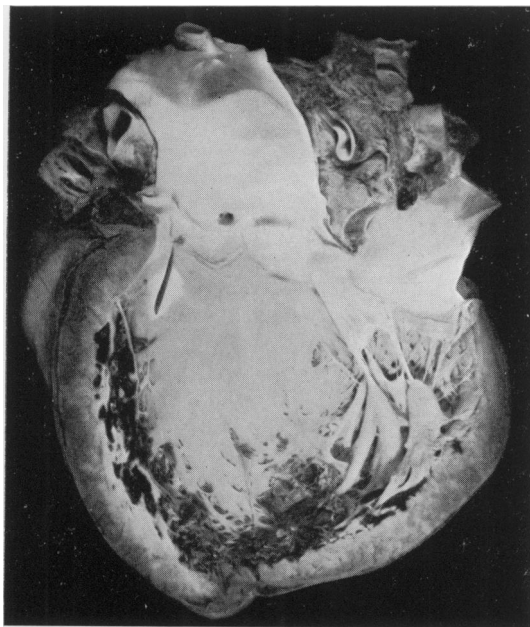
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ISOLATED (FIEDLER'S) MYOCARDITIS



**Fig. 1**

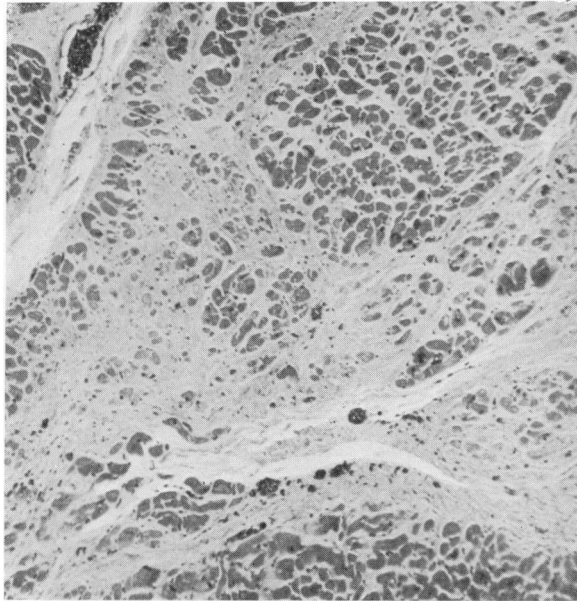
**Case I. A.3910.** To show one of the recent focal areas of muscle necrosis. Adjacent fibres show vacuolisation, and there is little inflammatory infiltration.



**Fig. 2**

**Case II. A.3856.** To show the dilated heart with its lining of ante-mortem thrombus.

ISOLATED (FIEDLER'S) MYOCARDITIS



**Fig. 3**

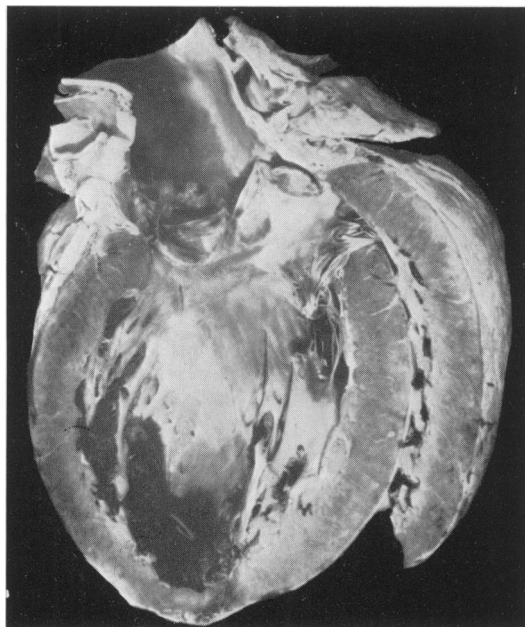
**Case II.** To show the irregular fibrosis of the myocardium. There is no cellular infiltration.



**Fig. 4**

**Case II.** To show the fibrous thickening of the endocardium resulting from organisation of mural thrombus.

ISOLATED (FIEDLER'S) MYOCARDITIS



**Fig. 5**

**Case III. A.4521.** To show the dilated heart and the extensive mural thrombus.

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 DR. EUSTACE CRESSER is well known for his work on sex education and marriage guidance, and this Pelican book, written for adolescents, will maintain his high reputation.

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# Rheumatic Heart Disease in Pregnancy

By FLORENCE McKEOWN, M.D.

Institute of Pathology, Belfast

## *A Paper read to the British Cardiac Society*

OUR post-mortem material of fatal cases of cardiac disease in pregnancy consists of rather a small series of twelve patients. Three of these are excluded, because, though the patients suffered from mitral stenosis, they died non-cardiac deaths; one from a cerebral lesion, the second from obstetric shock, and the third, while suffering from active rheumatism, had also pulmonary tuberculosis. This leaves nine cases which fall naturally into two groups.

The first consists of three cases of mitral stenosis with decompensation.

### MITRAL STENOSIS WITH DECOMPENSATION

|        |     | Age |     | Parity | Previous<br>Decompensation | Time of Decompensation  |
|--------|-----|-----|-----|--------|----------------------------|-------------------------|
| Case 1 | ... | 38  | ... | 2      | ...                        | — 26 weeks.             |
| Case 2 | ... | 31  | ... | 3      | ...                        | — 20-28 weeks.          |
| Case 3 | ... | 32  | ... | 1      | ...                        | — Delivery—2 weeks p.p. |

The first patient had rheumatic fever at the age of 24. Since then she had been troubled with her heart. Whether there was a history of decompensation with her first pregnancy is uncertain. At any rate, from the onset of the second pregnancy she suffered from breathlessness, and spent the last two weeks in hospital with increasing decompensation.

The second patient had no antecedent history of rheumatism. It was doubtful whether decompensation had been present earlier than the twentieth week, but from that time she had asthmatic attacks and died six days after admission with congestive heart failure.

The third patient was diagnosed as having pre-eclamptic toxæmia during her pregnancy, with no evidence of heart disease. She was discharged following delivery, still with some œdema of the ankles. This gradually became worse at home, and, on re-admission almost two weeks after delivery, she was in the terminal stages of heart failure.

At post-mortem all three patients had severe mitral stenosis, the third case having an associated tricuspid lesion. The valve histologically showed gross thickening and vascularisation, and there was fibrillar paravascular scarring in the myocardium.

It is generally stated that decompensation in cardiac disease in pregnancy usually occurs either in the early weeks, or about the sixth month, or shortly after delivery. We have examples of each in the three present cases. In the later months

of pregnancy it is readily understood why decompensation should occur, when a heart which is already handicapped by a valvular lesion is no longer able to meet the increased demands associated with pregnancy. If, however, a patient has had previously normal pregnancies while suffering from mitral stenosis, it is more difficult to understand why decompensation should develop in a later one. Some attribute this to the "progressive nature of the valvular lesion," but the valvular lesion does not appreciably progress unless there is a reactivation of a rheumatic valvulitis. If the valve lesion is quiescent, then further fibrosis should not occur.

The cause of failure in such cases is therefore more probably related to the state of the myocardium. Rheumatism not only affects the valves and interstitial connective tissue of the myocardium, but it also damages the coronary arteries, producing necrosis in the acute stage, which is followed by fibrosis. In addition, it may accentuate the normal age changes which occur in the coronary vascular system, so that, while we are dealing with an age group which normally should be relatively free from coronary sclerosis, yet the effect of these vascular changes is similar, and the thickening of the vessels which results from repeated attacks of rheumatic arteritis leads to a great diminution in cardiac reserve. This involvement of the coronary arteries varies from case to case, and helps to explain why one patient should develop congestive failure during pregnancy, while another with an identical valvular lesion does not.

Heart failure in the early months of pregnancy is another problem. Why should the heart fail when the pregnancy is in its early stages and can add little to the circulatory load? It may be that compensation has already been impaired, or that the cardiac reserve is lowered to a minimum, and the circulatory balance is easily disturbed. At this stage we must also exclude a recrudescence of rheumatic carditis as the causative factor.

It is the second group of cases which is of special interest.

| DECOMPENSATION AND RECRUDESCENCE |     |        |                            |                           |                        |  |
|----------------------------------|-----|--------|----------------------------|---------------------------|------------------------|--|
| Case                             | Age | Parity | Previous<br>Decompensation | Time of<br>Decompensation | Valve Lesion           |  |
| 4.                               | 23  | 1      | —                          | 14-18 weeks               | Mitral Stenosis        |  |
| 5.                               | 45  | 14     | with last 4<br>pregnancies | 22-26 weeks               | Mitral Stenosis        |  |
| 6.                               | 30  | 3      | —                          | 23rd week                 | Mitral Stenosis        |  |
| 7.                               | 38  | 4      | —                          | 34-36 weeks               | Slight Mitral Stenosis |  |
| 8.                               | 23  | 1      | —                          | Immed. p.p.               | Slight Mitral Stenosis |  |
| 9.                               | 30  | 1      | —                          | Immed. p.p.               | Slight Mitral Stenosis |  |

The first three patients had severe mitral stenosis, the second three had only a minimal valvular lesion. On histological examination, all gave evidence of a recent attack of rheumatic carditis. Case 5 was interesting in that of the whole group it was probably the one case in which decompensation was to be anticipated, and which could be attributed solely to myocardial exhaustion following on fourteen pregnancies, since there was a history of decompensation in the last four. Yet

she is the only patient of the entire series in whom it was suspected clinically that a fresh attack of rheumatic fever had occurred. She was admitted to hospital with early signs of cardiac failure, and during her stay she developed pains in the right knee and ankle, which were regarded as rheumatic manifestations.

Cases 8 and 9 were also unusual. Both patients attended the ante-natal clinic during their pregnancy and were in excellent health. Labour was slightly prolonged, but not abnormal, in either case. After delivery, however, both patients showed signs of shock, for no apparent reason, and died rather suddenly a few hours later. At post-mortem there were vegetations on the mitral valve, the myocardium was soft and flabby, and histologically Aschoff nodules of all ages were found in the muscle.

The Aschoff nodule passes through a cycle of changes, so that when final healing is taking place it is about nine months old. It was possible, therefore, in all these cases to determine roughly the age of the rheumatic lesion, and it was found that the onset of the rheumatic attack slightly preceded the onset of symptoms of decompensation. When a patient, however, has suffered from cardiac failure throughout most of her pregnancy, a failure which was dictated by a renewed attack of rheumatism at its commencement, it may not be apparent on post-mortem examination that the decompensation resulted from anything but the valvular lesion. An attack of rheumatism nine months previously may quite easily be overlooked.

The real problem raised by these cases is the question of subclinical rheumatism. We have considered six cases in which there was active rheumatic carditis and yet in only one was it clinically suspected. Sheehan (1940), in an analysis of 108 fatal cases of cardiac disease in pregnancy, found no evidence of a recrudescence in any of them, and Bramwell (1938) states that he has not met with any case of acute articular rheumatism or acute rheumatic carditis during pregnancy. If the recognition of such an attack rests on clinical grounds alone, then this is not surprising. But it is obvious that subclinical rheumatism must occur, for many patients with mitral stenosis, at post-mortem, give no antecedent history of rheumatic fever.

We investigated all our post-mortem material from this point of view in an attempt to estimate the incidence of healed rheumatic carditis in our total of three thousand cases, and to compare our results with known clinical figures. We found rheumatic stigmata in eight per cent. The incidence of rheumatism as observed clinically varies from one to four per cent. of the population, so that in a large number it must occur in a subclinical form.

Another conclusion which could be made from examination of these three thousand cases was that acute rheumatic carditis, while predominantly a disease of the younger age groups, knows no age limit, occurring in the later decades and even beyond the age of seventy. It is essentially a repetitive disease, recurring in the same subject over a period of many years. It perhaps pursues a less fulminant course in the adult whose reactions to most allergic processes become modified with age, and this, perhaps, explains its occurrence in a subclinical form.

Nevertheless, it does recur in many patients, and a realisation of this is important in dealing with cardiac disease in pregnancy. The patient who is suffering from mitral stenosis and who is pregnant is open to two risks; the first being that the damaged heart may be unable to withstand the strain of pregnancy; and secondly, that while successfully meeting the demands of pregnancy, heart failure may be precipitated by the presence of a fresh attack of rheumatic carditis.

The major problem lies in the detection of a recrudescence. It may be possible in the patient who is known to be a cardiac, and who is kept under careful clinical observation during her pregnancy. But the majority of these patients are admitted as emergencies, already entering upon the stage of decompensation, when, even if there is an exacerbation of rheumatic carditis, its recognition is of lesser importance.

One fact which emerges from this study is the necessity for post-mortem examination. If we consider the two groups of cases on a purely clinical basis, there is nothing to distinguish them and they both fall into the one class of mitral stenosis with decompensation. As long as we have no reliable clinical means of detecting whether the heart lesion is active or not, the final proof must await post-mortem. A further reason for autopsy is that an attempt should be made to determine the exact nature of the valvular lesion. To quote Sheehan and Jensen, both workers are impressed with the large number of cases in their series in which there was an error in diagnosis as to the type of valvular lesion present, and its degree of severity. These facts stress the inaccuracy of statistics based on clinical findings only.

Post-mortem should include not only macroscopic study of the heart, but a thorough histological examination also, including preparation of blocks from the mitral valve, left auricle, septum, and right ventricle, since the rheumatic process tends to occur in these sites. The absence of rheumatic lesions in one block does not exclude its presence from the heart.

In summary, it may be said that in some cases of heart disease and pregnancy, the severity of the valvular lesions, the condition of the myocardium, and the strain of pregnancy are sufficient of themselves to cause heart failure. In others, and probably an increasing number, if post-mortem examination were carried out, it is not the valvular lesion nor the strain of pregnancy which are the primary factors, but an exacerbation of rheumatic carditis, which is responsible for the development of decompensation. In most cases of this type the active rheumatic infection is subclinical, and can only be verified by autopsy.

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# Heart Disease and Pregnancy

By ROBERT MARSHALL, M.D., F.R.C.P.(LOND.), F.R.C.P.I.

*A Paper read to the British Cardiac Society in June, 1949*

THE material on which this short paper is based has been derived from one's experience in seeing cases of heart disease complicating pregnancy in the Royal Maternity Hospital during the past twenty years, and I am indebted to the members of the visiting staff of that hospital for this privilege, and, in particular, to Doctor Kenneth Hudson, lately Obstetric Tutor, for the statistical data which I shall briefly present to you.

During the past fifteen years there has been a steady fall in maternal mortality for various reasons, to which attention has repeatedly been drawn by His Majesty's present Government with, perhaps, unpardonable pride, but, as MacRae<sup>1</sup> has pointed out, heart disease is actually gaining a more prominent position as a cause of maternal deaths. During the years 1943-1947, 743 women in England, Wales, Scotland, and Northern Ireland died from heart diseases associated with pregnancy and childbirth (Table 1). Each of these deaths was a tragedy, affecting other lives than

TABLE 1  
DEATHS FROM HEART DISEASE ASSOCIATED WITH PREGNANCY AND CHILDBIRTH  
1943-1947

| International<br>List-Number |  | England & Wales | Scotland          | N. Ireland |
|------------------------------|--|-----------------|-------------------|------------|
| 90                           | Pericarditis ... ..                                    | 3               |                   | 1          |
| 91                           | Acute Endocarditis ... ..                              | 27              |                   | 1          |
| 92                           | Chronic Affections of Valves and<br>Endocardium ... .. | 402             | Not<br>Classified | 19         |
| 93                           | Diseases of Myocardium ... ..                          | 107             |                   | 5          |
| 94                           | Diseases of Coronary Arteries ... ..                   | 12              |                   | 3          |
| 95                           | Other Disease of Heart ... ..                          | 17              |                   | 2          |
|                              |  | 568             | 144               | 31         |

Live Births—England and Wales, 3,817,494; Scotland, 495,115; Northern Ireland, 152,816.

Percentage of Deaths to Live Births—England and Wales, 0.016; Scotland, 0.03; Northern Ireland, 0.02.

their own. Some of the deaths were avoidable, and I believe that we can prevent the repetition of many such deaths in the future. In Scotland the diseases were not classified, but, as everyone knows, the largest group was in category ninety-

two, and the predominating cause was mitral stenosis. The proportion of maternal deaths to live births was higher in Northern Ireland than in England and Wales, but lower than in Scotland. In the Royal Maternity Hospital, Belfast, in the years 1937-1948 (Table 2) there were 24,078 total admissions, with an incidence of 1.8 per

TABLE 2  
ROYAL MATERNITY HOSPITAL, BELFAST, 1937-1948

|                            |     |        |                 |
|----------------------------|-----|--------|-----------------|
| Total Admissions           | ... | 24,078 |                 |
| Incidence of Heart Disease | ... | 442    | 1.83 per cent.  |
| Maternal Deaths            | ... | 26     | 5.88 per cent.  |
| Emergency Admissions       | ... | 43     | 9.75 per cent.  |
| Maternal Deaths            | ... | 20     | 46.51 per cent. |

cent. of heart disease, as compared with MacRae's finding of 0.8 per cent. in a review of 29,713 patients admitted to Queen Charlotte's. This was not unexpected, as rheumatic heart disease is common in Northern Ireland. The maternal death rate was higher than in Queen Charlotte's, where it was 3.1 per cent., but you will note that 46.5 per cent. of these deaths occurred among the 43 emergency admissions. This is comparable to the figures presented by Bramwell<sup>2</sup> in the table which I have reproduced with his kind permission (Table 3). Kenneth Hudson has

TABLE 3  
"URGENCY" DEATH-RATE

|                                      | Cases | Total Deaths | "Urgency" Deaths | Per Cent. Urgencies |
|--------------------------------------|-------|--------------|------------------|---------------------|
| 1. Bramwell and Longson (Manchester) | 350   | 25           | 14               | 56                  |
| 2. Carr and Hamilton (Boston)        | 500   | 32           | 16               | 50                  |
| 3. B.P. Watson (New York)            | 240   | 17           | 8                | 47                  |

TABLE 4  
CLASSIFICATION, NEW YORK HEART ASSOCIATION  
ALL ADMISSIONS

|         |     |     |     |                 |
|---------|-----|-----|-----|-----------------|
| Class 1 | ... | 213 | ... | 48.29 per cent. |
| Class 2 | ... | 127 | ... | 28.97 per cent. |
| Class 3 | ... | 76  | ... | 16.96 per cent. |
| Class 4 | ... | 26  | ... | 5.88 per cent.  |

DEATHS

|         |     |     |     |    |
|---------|-----|-----|-----|----|
| Class 1 | ... | ... | ... | 0  |
| Class 2 | ... | ... | ... | 2  |
| Class 3 | ... | ... | ... | 3  |
| Class 4 | ... | ... | ... | 21 |

76 per cent. of deaths were emergency admissions.

50 per cent. of deaths occurred in the first pregnancy.



classified his cases according to the New York Heart Association Grouping, and here again the results are comparable with those found elsewhere. It is always difficult to be rigid in such classification, as patients have a trick of moving from one classification to another (Table 4). The ætiological factor (Table 5) was found

TABLE 5

| ÆTIOLOGICAL FACTOR             |     |     |     |     |                 |
|--------------------------------|-----|-----|-----|-----|-----------------|
| Rheumatic Fever                | ... | ... | 176 | ... | 39.81 per cent. |
| "Ambulatory" Pains             | ... | ... | 68  | ... | 15.38 per cent. |
| Chorea                         | ... | ... | 53  | ... | 11.99 per cent. |
| Scarlet Fever                  | ... | ... | 27  | ... | 6.14 per cent.  |
| Tonsillitis                    | ... | ... | 22  | ... | 4.97 per cent.  |
| No History of Relevant Disease | ... | ... | 96  | ... | 21.71 per cent. |

to be rheumatic fever in only 39.8 per cent. of cases, and, in chorea, in almost 12 per cent. Between these two there occurred those debatable cases giving a history of limb pains, which I have called "ambulatory" for two reasons: because the pains were not sufficiently severe to confine the patients to bed, and because I am almost afraid even to hint my secret opinion that so-called "growing pains" are quite frequently manifestations of rheumatism. Like other people, we have found 20 per cent. would not admit ever to have suffered from any form of rheumatism. There is nothing new in all this. Similarly (Table 6), valvular lesion was mitral stenosis in 85 per cent. of cases. Curiously enough, there were only three classified as due to congenital defect. The termination of pregnancy (Table 7) need not

TABLE 6

| VALVULAR LESION                        |     |     |     |     |                 |
|--|-----|-----|-----|-----|-----------------|
| Mitral Stenosis                        | ... | ... | 376 | ... | 85.06 per cent. |
| Mitral Regurgitation                   | ... | ... | 32  | ... | 7.46 per cent.  |
| Mitral Stenosis & Aortic Regurgitation | ... | ... | 22  | ... | 4.70 per cent.  |
| Aortic Regurgitation                   | ... | ... | 8   | ... | 1.80 per cent.  |
| Aortic and Mitral Stenosis             | ... | ... | 1   | ... | 0.22 per cent.  |
| Congenital Defect                      | ... | ... | 3   | ... | 0.66 per cent.  |

TABLE 7

| TERMINATION OF PREGNANCY—442 CASES           |                             |           |                             |           |                                      |
|--|-----------------------------|-----------|-----------------------------|-----------|--------------------------------------|
| Method                                       | Class 1 & 2<br>"Ante-natal" |           | Class 3 & 4<br>"Ante-natal" |           | Emergency<br>Admissions<br>Per Cent. |
|  | Per Cent.                   | Per Cent. | Per Cent.                   | Per Cent. |                                      |
| Vaginal delivery live child at term          | ...                         | 90        | ...                         | 54        | 29.5                                 |
| Cæsarean section, cardiac indications        | ...                         | 1.2       | ...                         | 14.8      | 5.3                                  |
| Therapeutic Abortion                         | ...                         | —         | ...                         | 9.05      | 14                                   |
| Died (Conception to 6 months after delivery) | ...                         | 0.3       | ...                         | 8.1       | 46.5                                 |

delay us, except to point out that though Cæsarean section was performed with decreasing frequency, you will note that there is no mention whatever of the induction of labour, a proceeding of which our obstetricians disapprove.

Crichton Bramwell's figures and our own both show the same high mortality in cases which are admitted, usually at a late stage of pregnancy, as emergencies, and it is to adequate ante-natal care that we must look for a reduction in the numbers of maternal deaths. I had been somewhat concerned as to whether the trend of recent legislation was to place the patient under the care of the midwife, rather than the doctor, but I am assured that this will not be the case, and that a doctor practising obstetrics under the National Health Service is required to make proper ante-natal examinations on at least three occasions. It is, therefore, most important that he should be taught to look carefully for evidence of mitral stenosis or other cardiac lesion. Here may I say that students in the Belfast Medical School are well trained in this aspect of cardiology by the obstetricians, who are most expert in it. In Belfast the energy and enthusiasm of the obstetricians and gynæcologists has led to the student devoting a very great deal of his available time to these subjects. He has one month's residence in medical wards, one month's residence in surgical wards, he has two month's residence in the maternity hospital, and frequently a month's resident pupilship in gynæcology as well. Belfast was a pioneer in this system of resident pupilship, which was originally begun in the Belfast General Hospital in 1820. Of recent years English and Scottish teaching hospitals have recognised that it is a most valuable part of the curriculum. The odd thing is that young graduates, so admirably equipped to practise obstetrics, now find that when they go to the other island they are permitted to practise medicine and surgery, but they are not eligible to bring a baby into the world without the express permission of the appropriate local obstetric committee.

While it is frequently easy to determine the presence of mitral stenosis, and equally easy to miss it in a perfunctory examination, it is not always easy to assess the importance of the early symptoms of cardiac distress. As MacRae has put it: "Changes in the heart and circulation in pregnancy, such as the increase in volume of the circulating blood, the greater vascularity of the enlarging uterus, the increase in body weight, the splinting of the diaphragm during the later months, with rotation and displacement of the heart, may cause breathlessness and palpitation, even in a healthy patient." The cardinal signs of early congestive failure may also be more difficult to assess in the pregnant woman. As Evans<sup>3</sup> has said, the extent of venous engorgement in the neck has not always proved satisfactory as a clinical measure of the degree to which venous pressure has risen as the result of right heart failure. I have found that hepatic distension is peculiarly difficult to assess after the early months of pregnancy, because I believe the forward thrust of the abdominal wall makes both palpation and percussion of the liver unreliable. Some slight swelling of the ankles may occur in normal pregnancy, and does not necessarily mean congestive heart failure. X-ray examination may, of course, be diagnostic, but here again the raised diaphragm adds to one's difficulty; therefore, it is desirable that radioscopy should be undertaken at an early stage of pregnancy.

To my mind the most useful clinical hint is given by finding what number of pillows the patient requires at night. If she can rest comfortably on two pillows it is an excellent prognostic sign. Pallor is, on the other hand, a disquieting sign, and one which is not always easy to explain, though it suggests toxæmia, possibly of pre-eclamptic type.

In any case, where there is doubt about the patient's cardiac condition, she should be admitted to hospital during her pregnancy for a period of rest and observation. MacRea recommends admission at about the twenty-eighth week for complete rest and assessment. A fortnight or more at this phase of pregnancy may be of the utmost value, part of which is psychological, because it gives the patient not only rest, but reassurance. All heart cases should be re-admitted at least a week before the date of expected confinement. It is, therefore, obvious that the closest co-operation must be ensured between the doctor and the midwife, and that doctors must be trained to look for and recognise heart disease in pregnancy, and that midwifery is not enough.

To turn for a moment to another aspect of the matter; in recent years there has arisen a tendency to regard the coincidence of heart disease and pregnancy in a much less unfavourable light than formerly, and Bramwell,<sup>4</sup> Gilchrist,<sup>5</sup> and Jensen<sup>6</sup> have published the results of the follow-up of cases. I have recently tried to see all the heart cases which were in the Royal Maternity Hospital in the years 1942-1946. Of 150 patients, 8 are now known to have died, 14 could not be traced, 103 reported for examination. Of these, 3 were regarded on review as not having suffered from organic heart disease, leaving an even 100 cases actually reviewed. The results of this enquiry can be briefly stated. Of the 8 fatal cases, 6 had been diagnosed as mitral stenosis, and 2 as mitral stenosis and aortic regurgitation. Seven had died within two years of leaving the Royal Maternity Hospital. Of the survivors, the data may be briefly presented on slides, the age group (Table 8), the number of pregnancies (Table 9), 29 per cent. having had only one child, but one patient with mitral stenosis had no fewer than thirteen children. She states that she had no cardiac symptoms until her sixth child, but had increasingly severe symptoms with each successive pregnancy. She give no history of rheumatic

TABLE 8  
AGE-GROUPS ON REVIEW

|       |     |     |     |    |
|-------|-----|-----|-----|----|
| 20-25 | ... | ... | ... | 2  |
| 25-30 | ... | ... | ... | 16 |
| 30-35 | ... | ... | ... | 27 |
| 35-40 | ... | ... | ... | 20 |
| 40-45 | ... | ... | ... | 26 |
| 45-50 | ... | ... | ... | 8  |
| 50-55 | ... | ... | ... | 1  |

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100

TABLE 9  
REVIEWED CASES

|    |             |     |     |    |
|----|-------------|-----|-----|----|
| 1  | Pregnancy   | ... | ... | 29 |
| 2  | Pregnancies | ... | ... | 27 |
| 3  | „           | ... | ... | 21 |
| 4  | „           | ... | ... | 5  |
| 5  | „           | ... | ... | 6  |
| 6  | „           | ... | ... | 2  |
| 7  | „           | ... | ... | 3  |
| 8  | „           | ... | ... | 1  |
| 9  | „           | ... | ... | 3  |
| 10 | „           | ... | ... | 1  |
| 11 | „           | ... | ... | 1  |
| 12 | „           | ... | ... | 0  |
| 13 | „           | ... | ... | 1  |

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100

fever or chorea. She is now 43 and maintains that her general health is better than it was, and thinks that having her babies did her no harm. The causes (Table 10) in this group correspond almost identically with Hudson's figures already shown you, and the lesions (Table 11) show a similar distribution. We electrocardiographed our reviewed cases (Table 12), but were unable to X-ray them because no screen was available, and there was an acute shortage of X-ray films.

TABLE 10

*Reviewed Cases.*

| CAUSES                     |     |     |     |    |
|----------------------------|-----|-----|-----|----|
| Rheumatic Fever            | ... | ... | ... | 46 |
| Rheumatic Fever and Chorea | ... | ... | ... | 4  |
| Chorea                     | ... | ... | ... | 13 |
| “Ambulatory Pains”         | ... | ... | ... | 11 |
| Scarlet Fever              | ... | ... | ... | 5  |
| Tonsillitis                | ... | ... | ... | 1  |
| No Rheumatic History       | ... | ... | ... | 20 |

---

100

TABLE 11

*Reviewed Cases.*

| LESIONS                                  |     |     |     |    |
|--|-----|-----|-----|----|
| Mitral Stenosis                          | ... | ... | ... | 82 |
| Mitral Stenosis and Aortic Regurgitation | ... | ... | ... | 11 |
| Mitral and Aortic Stenosis               | ... | ... | ... | 1  |
| Mitral Regurgitation                     | ... | ... | ... | 3  |
| Auricular Septal Defect                  | ... | ... | ... | 2  |
| Myocardial Disease                       | ... | ... | ... | 1  |

---

100

TABLE 12

| ELECTROCARDIOGRAMS ( <i>Leads 1, 2, 3 only</i> ) |     |     |     |    |
|--|-----|-----|-----|----|
| Normal   | ... | ... | ... | 31 |
| Large or bifid P waves                           | ... | ... | ... | 35 |
| Right axis deviation                             | ... | ... | ... | 10 |
| Left axis deviation                              | ... | ... | ... | 10 |
| Flat T waves                                     | ... | ... | ... | 2  |
| Inverted T waves                                 | ... | ... | ... | 5  |
| Auric. Fibrill                                   | ... | ... | ... | 7  |

100

TABLE 13

| ONSET OF CARDIAC SYMPTOMS      |     |     |     |    |
|--------------------------------|-----|-----|-----|----|
| <i>Before</i> Pregnancy        | ... | ... | ... | 40 |
| <i>During</i> First Pregnancy* | ... | ... | ... | 34 |
| <i>During</i> Second Pregnancy | ... | ... | ... | 4  |
| <i>During</i> Third Pregnancy  | ... | ... | ... | 7  |
| <i>During</i> Fourth Pregnancy | ... | ... | ... | 3  |
| <i>During</i> Fifth Pregnancy  | ... | ... | ... | 1  |
| <i>During</i> Sixth Pregnancy  | ... | ... | ... | 2  |
| Patient's Answer Uncertain     | ... | ... | ... | 9  |

\* Of these 34 patients:—

16 had rheumatic fever;

4 had chorea;

14 had no relevant history.

The relationship of pregnancy to the onset of cardiac symptoms (Table 13) shows that only forty per cent. did admit that they had such symptoms before pregnancy. Thirty-four per cent. stated that they first became aware of undue breathlessness during their first pregnancy. Of these, twenty knew that they had had rheumatism or chorea, but in most cases this attack was during childhood, and they had not been told that their hearts had been affected. Not uncommonly the girl who has rheumatic fever with carditis before puberty is genuinely free from symptoms during the age period from 14 to 21, and her apparent health dulls her mother's memory of the doctor's warning. It is at this age period that she drifts out of the care of the children's hospitals, and even out of the ambit of the school medical services. Again, the desire to be married and to have a child may have closed her eyes to symptoms which she preferred not to think about.

Perhaps most pitiable are those—fourteen in this series—who had no reason to fear heart disease. It is, above all, this group who present the greatest reason why every doctor examining a young expectant mother for the first time should ask himself: "Am I satisfied that this patient has *not* got mitral stenosis?"

It is not always easy to assess their present symptoms (Table 14), but we tried to determine whether their child-bearing had adversely affected their health. The next table (Table 15) represents their answers to the simple question: "What

Reviewed Cases.

TABLE 14

| PRESENT SYMPTOMS |     |     |     |     |
|------------------|-----|-----|-----|-----|
| None             | ... | ... | ... | 9   |
| Slight           | ... | ... | ... | 27  |
| Moderate         | ... | ... | ... | 42  |
| Severe           | ... | ... | ... | 22  |
|                  |     |     |     | 100 |

Reviewed Cases.

TABLE 15

| EFFECT OF PREGNANCY ON CARDIAC SYMPTOMS |     |     |     |     |
|---|-----|-----|-----|-----|
| Health Improved                         | ... | ... | ... | 5   |
| No Difference                           | ... | ... | ... | 42  |
| Health Impaired                         | ... | ... | ... | 53  |
|   |     |     |     | 100 |

effect do you think having your baby (or babies) has had on your health?" It is admitted that there is probably no group of patients whose answers are more likely to be biased. To the majority of women, the achievement of motherhood blunts the edge of pain and minimizes sacrifice. To a minority, childbirth is the curse of Eve, on which all ill-health and unhappiness may be blamed. Nevertheless, I suggest that we must remind ourselves that it is not merely a question of nursing our patient with mitral stenosis until her baby is safely born, but that child-bearing is only a prelude and a part of child-rearing, on which depends the future of the race.

I am grateful to Mr. Ian McClure, for his advice and help; to Mr. Kenneth Hudson, for his valuable data; and to Dr. Arnold Lyons and Dr. Richard Vine, for their collaboration in the examination of patients who attended for "review."

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## REVIEW

A HISTOLOGY OF THE BODY TISSUES, with consideration of their functions.

By Margaret Gillison. Edinburgh: E. & S. Livingstone Ltd. 1950. 15s.

AN elementary introduction to histology. The author is to be congratulated on her physiological approach to the subject. The book is divided into chapters, each dealing with a particular group of tissues. The major part of each chapter is taken up with a correlation between the structure and function of the tissues and is followed by brief descriptions of the individual types. The book is illustrated by numerous clear diagrams.

D. H. S.

Reviewed Cases.

TABLE 14

| PRESENT SYMPTOMS |     |     |     |     |
|------------------|-----|-----|-----|-----|
| None             | ... | ... | ... | 9   |
| Slight           | ... | ... | ... | 27  |
| Moderate         | ... | ... | ... | 42  |
| Severe           | ... | ... | ... | 22  |
|                  |     |     |     | 100 |

Reviewed Cases.

TABLE 15

| EFFECT OF PREGNANCY ON CARDIAC SYMPTOMS |     |     |     |     |
|---|-----|-----|-----|-----|
| Health Improved                         | ... | ... | ... | 5   |
| No Difference                           | ... | ... | ... | 42  |
| Health Impaired                         | ... | ... | ... | 53  |
|   |     |     |     | 100 |

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D. H. S.

# Coarctation of the Aorta and Subarachnoid Hæmorrhage with Recovery

By ROBERT MARSHALL, M.D., F.R.C.P.(LOND.), F.R.C.P.I., and  
J. M. BARBER, M.D., M.R.C.P.(EDIN.)

SUBARACHNOID hæmorrhage is recognised as a possible complication of coarctation of the aorta, but its occurrence with recovery at the age of sixty is sufficiently unusual to warrant a detailed report.

Mrs. M. McC., aged sixty years, was admitted to the Royal Victoria Hospital on 3rd February, 1950, at 11.15 p.m. in a semi-comatose condition.

A few hours previously she had complained of the sudden onset of a severe headache. This was followed by vomiting.

The family history revealed that one sister had died of "cerebral hæmorrhage" at the age of forty years. Six other sisters and one brother were alive and well.

The patient was said to have suffered from "rheumatic fever" in 1925. This was the only notable illness in her sixty years. She had worked as a sweeper in a linen mill from the age of eleven years until the onset of her present illness, with an interval of ten years following her marriage in 1924.

On admission, the most striking physical sign was marked neck rigidity. Her blood pressure was 250/150 in both arms. Both pupils reacted to light, but the left was larger than the right. All four limbs showed an increase in tone with increased reflexes. The plantar responses were flexor.

Lumbar puncture produced blood-stained cerebro-spinal fluid containing 1,790,000 red blood cells and 1,200 leucocytes per cubic millimetre, with 2,200 milligrams of protein per 100 c.c. and a gross increase in globulin. Kahn and Wassermann reactions were negative. Headache persisted to ten days and neck rigidity was noted for a further seventeen days.

When full clinical examination was possible the following signs were present. Arterial pulsation was seen on both sides of the neck and in the suprasternal notch. Dilated, pulsating arteries could be seen and felt between the scapulæ. The apex beat was heaving in character and displaced to the left. Femoral, popliteal, and dorsalis pedis pulses were absent. The blood pressure was unobtainable in the legs. There was a systolic murmur of moderate intensity maximal in the pulmonary area, but equally well heard over the fourth dorsal spine. No diastolic murmur was heard. Specific gravity range was 1.003 to 1.020, with a blood urea of 44 milligrams per 100 c.c. The retinæ showed arteriolar narrowing and venous nipping, but no hæmorrhages or papillœdema.

Considerable thoracic scoliosis interfered with interpretation of the X-ray films. The straight film revealed notching of the ribs. The ascending aorta was prominent



but there was no aortic knob. On fluoroscopy, the pulsating left subclavian artery contrasted with the absence of pulsation in the adjacent part of the descending aorta. In the left anterior oblique position left ventricular hypertrophy of moderate degree was present. The aortic window was wide and clear and the aorta could not be followed beyond the base of the aortic triangle.

The electrocardiogram showed marked deviation of the electrical axis to the left. The depression of the RST segment and inverted T wave in the unipolar left arm lead indicated left ventricular hypertrophy.

The phonocardiogram was recorded with the microphone of the Elmqvist apparatus. Tracings were made from the second left intercostal space at the sternal border and from over the fourth dorsal spine. The record from the base of the heart showed a systolic murmur of moderate intensity, but no diastolic murmur. Over the back a systolic murmur of about the same intensity was recorded. There was also a faint diastolic murmur; this appeared to be continuous with the systolic murmur and to be decrescendo in configuration.

The patient made a complete recovery from her subarachnoid hæmorrhage and was discharged, free from symptoms, on 22nd March, 1950.

#### AUSCULTATORY SIGNS

In Abbott's extensive review she quotes Laubry in stating that "a systolic murmur with maximum intensity along the left sternal border and heard also at the back, which is thought to be generated at the construction is characteristic."

Reifenstein, Levine, and Gross considered that a systolic murmur in the back accompanying a murmur of only moderate intensity anteriorly is more diagnostic. Especially is this so if the murmur is louder over the spine, but they thought that this was rarely the case. They stated that a diastolic murmur is not found in uncomplicated coarctation, but only when there is associated deformity of the aortic valve or patency of the ductus arteriosus.

Wells, Rappaport and Sprague investigated fifteen cases phonocardiographically. All of these had a systolic murmur over the dorsal spine and all but one had a similar murmur over the præcordium. On clinical auscultation a diastolic murmur was present in six over the spine and in five over the præcordium. Occasionally both types of murmur were of greater intensity over the dorsal spine. In no instance were the murmurs louder over collateral vessels. The phonocardiogram revealed diastolic murmurs in tracings from the dorsal spine in every patient and from the præcordium in ten patients.

In our case no diastolic murmur could be heard clinically, but the sound recording from over the fourth dorsal vertebra recorded faint diastolic vibrations.

#### RADIOLOGICAL DIAGNOSIS

The most important sign is notching of the ribs. This was first noticed at necropsy by Meckel in 1827. His artist showed the excavations on the upper surfaces of the third and fourth right ribs. Rossler noted the erosions in an X-ray film in 1928, but Railsback and Dock (1929) were first to stress the diagnostic value of

this sign. The notching characteristically appears on the lower edges, but, as Edwards and his associates have shown, it actually involves the internal surfaces in the costal grooves. It is rarely seen before the age of twelve and is seldom found on the upper rib or on the lower two or three ribs. Proudfit and Ernstene claim that a line of increased density near the inferior rib margin can be seen, both with and without typical notching. Ordinary films and screening do not visualise the site of coarctation. The aortic knuckle is usually absent or small. The left subclavian artery is widened and may give an S-shaped outline to the left superior mediastinum. There is a discrepancy between the increased pulsations in this artery and the diminished pulsation in the adjacent descending aorta. The ascending aorta may be prominent, and, owing to the failure to visualise the descending aorta, the aortic window is unusually clear and wide.

In uncomplicated cases the degree of enlargement of the left ventricle is less than one would expect to see in the presence of long-standing hypertension. If it is, marked aortic incompetence from bicuspid valves is frequently present.

Angiocardiography or retrograde aortography can be used to assess the site and length of the narrowed segment prior to operation. Tomography may occasionally be of value.

#### PROGNOSIS

Maude Abbott (1928) reviewed a series of two hundred fatal cases, of whom thirteen per cent. had died from cerebral hæmorrhage. In only six of these patients, in whom cerebral hæmorrhage was found at necropsy, was the cause proved to be the rupture of an aneurysm, but she inferred its probable presence in the majority of such cases, and her inference has met with general acceptance.

Reifenstein and his associates (1947), reviewing a later series of 104 fatal cases, found, that while coarctation occasionally was compatible with long life, at least sixty-one per cent. of the patients died before or during their fortieth year, the average age of death being 35 years. The causes of death and their frequencies were as follows:—incidental causes, 26 per cent.; rupture of the aorta (most commonly the ascending portion), 23 per cent.; bacterial endocarditis or aortitis, 22 per cent.; congestive failure, 18 per cent.; and intracranial lesion (exclusive of embolism from bacterial endocarditis), 11 per cent. The commonest cause of an intracranial lesion was rupture of an arterial aneurysm, probably congenital, which produced subarachnoid hæmorrhage. Survival rarely occurred after this type of rupture or after rupture of the aorta itself. Similarly, a further element of risk is provided by the phenomenon of "intermittent leakage," which was stressed by Wichern and Fearnside as a feature of the majority of the cases of coarctation who died of subarachnoid hæmorrhage.

Because of the severe degree of coarctation, the very marked hypertension, and the considerable subarachnoid hæmorrhage which our patient suffered at the age of sixty years, it is felt that the outlook is precarious and that her expectation of life cannot be good; but the fact that she survived this very serious incident is worthy of report, and we can find no parallel case in the literature available to us.

## SUMMARY

A case of coarctation of the aorta in a woman aged sixty is described. She survived a subarachnoid hæmorrhage. Auscultatory and radiological findings in coarctation are briefly discussed.

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The Lyceum was founded by Aristotle, whose father was a doctor. It was succeeded by the Museum of Alexandria, established by the Macedonian monarchs, with "a sort of American opulence." There was established the art of writing treatises, the most famous being that of Euclid, which entitles this period to be known as the "Age of the Text-book." The text-book had its origin in practical needs.

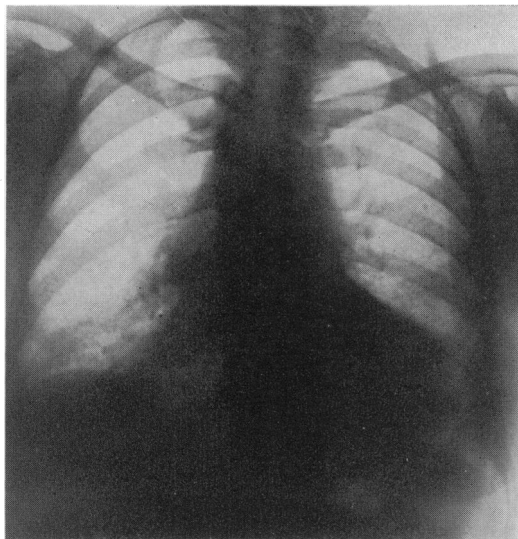
Hirophilus (300 B.C.) wrote a "Handbook for Midwives," an example of that "humanitarian zeal, which shines out of the pages of Greek medicine." He was the father of Neurology and "the nomenclature of parts of the brain bears traces of his work." His contemporary, Erasistratus, was the first physiologist. The most important event of these centuries was the steady rise of Rome to world power. Science became Græco-Roman, and the culture of the Roman world was bilingual. Cornelius Celsus, a Roman physician, has left us "the best general treatise on medicine of all the ancients," and Sir Clifford Allbutt has called him the "creator of scientific Latin." His work was a compilation from various sources, it might serve as a "manual for a professional school." He laid "immense emphasis on first-hand clinical observation," and he was "in the line of the great healers."

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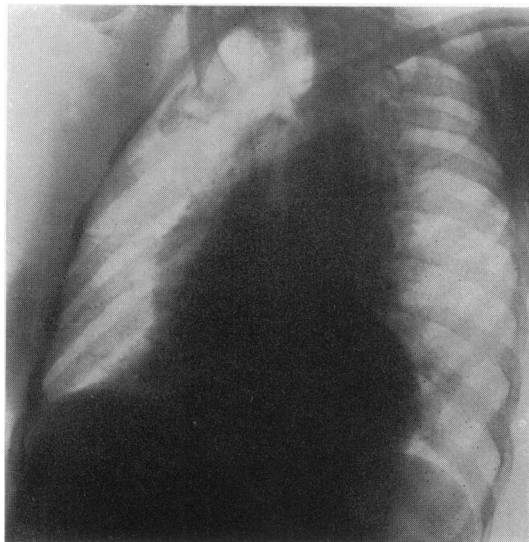
S. I. T.

COARCTATION OF THE AORTA



**Fig. 1**

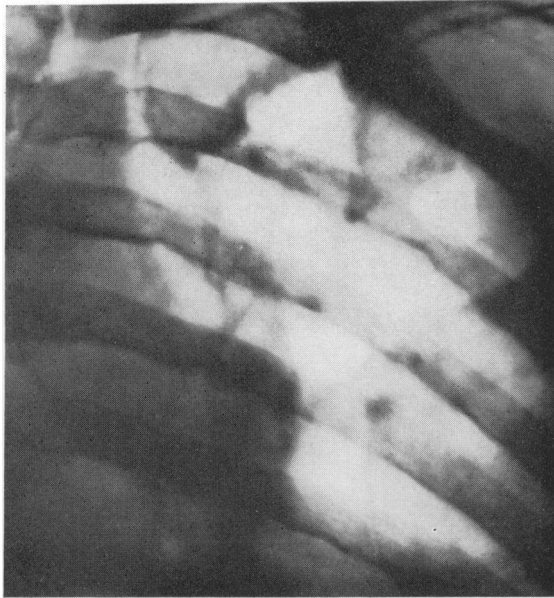
Anterio-posterior film of the chest showing prominence of the ascending aorta and absence of the aortic knob.



**Fig. 2**

The left anterior oblique film showing moderate left ventricular enlargement.

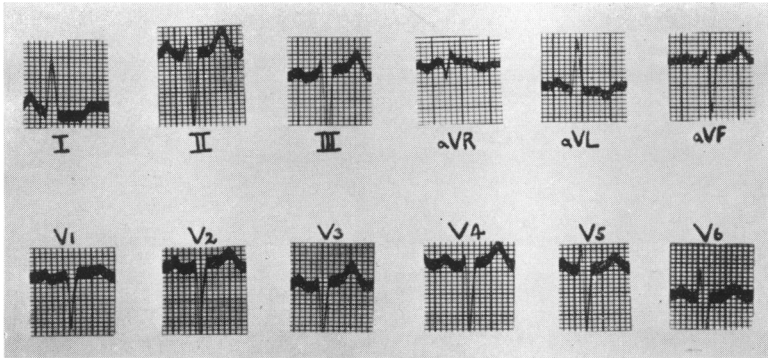
COARCTATION OF THE AORTA



**Fig. 3**

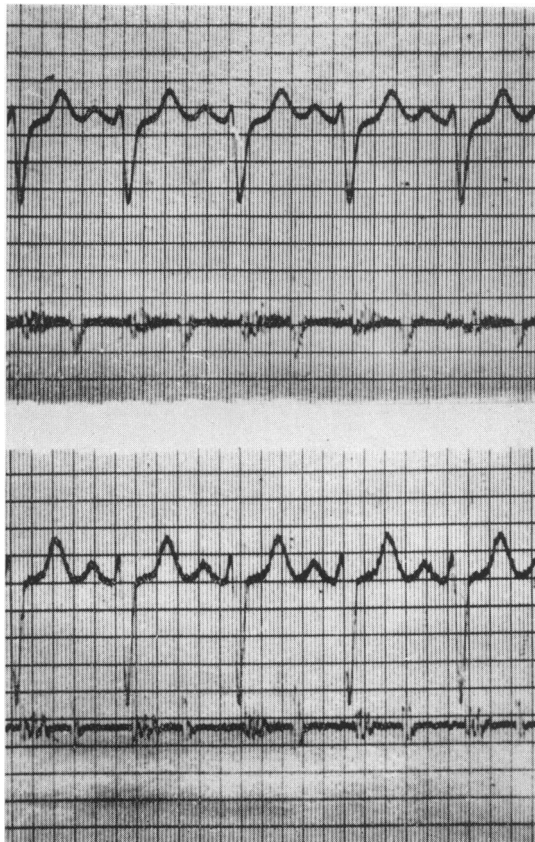
Enlargement of a penetrating film to show the rib notching.

COARCTATION OF THE AORTA



**Fig. 4—Electrocardiogram**

Note depression of the RST segment and inversion of the T wave in the unipolar left arm lead.



**Fig. 5—Phonocardiograms**

(above) Tracing with the microphone over the fourth dorsal spine showing systolic and diastolic murmurs.

(below) Recording from the second left intercostal space anteriorly. Systolic murmur only.

## SUMMARY

A case of coarctation of the aorta in a woman aged sixty is described. She survived a subarachnoid hæmorrhage. Auscultatory and radiological findings in coarctation are briefly discussed.

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S. I. T.

# Neo-Natal Surgery

By MURIEL J. L. FRAZER, B.A., M.D., M.R.C.P.(LOND.), F.R.C.S.I.

*Based on a Paper read before the Ulster Medical Society*

THIS paper is based on experience—often bitter experience—and makes no pretence at summarizing the literature, which makes the whole problem sound far too easy. As we all know, text-book cases occur only in text-books. Nor shall I quote the incidence of various conditions per one thousand live births. To know that something happens once in every two thousand babies is no help in deciding whether any given infant is number 2,000, or number 1,999.

There are various ways of classifying the neo-natal lesions requiring surgery, which, incidentally, should aim only at being lifesaving, surgery of election being very limited in amount.

Thus, the scope of neo-natal surgery may be classified :

A—*According to Cause* :—

    Congenital deformities : potentially lethal, e.g., exomphalos; distressing, e.g., hare lip.

    Birth hazards : e.g., sub-dural hæmatoma.

B—*According to their degree of urgency* :—

    Infection present or imminent : Imminent, e.g., exomphalos; Present, e.g., abscesses, thrush.

    Obstruction present or imminent : The common sites being : œsophagus, duodenum, mid gut, large bowel.

    The rest (not of immediate urgency).

C—*Those in which the diagnosis is* :—

    Manifest (imposing no strain on the diagnostician), e.g., exomphalos.

    In doubt (extremely worrying, since delay or unnecessary operation are almost equally hazardous).

I shall deal first with exomphalos and omphalocele, which appear in all these groups. They differ only in severity, the first term being applied to cases in which almost the whole abdominal contents are enclosed only by a thin transparent membrane, while the second term covers such lesser abnormalities as the presence of a single loop of bowel contained in a small sac intimately connected with the umbilical cord. The cord then has the appearance of being unduly vascular, and may be ligatured with more than usual firmness on that account. These children need urgent operation on account of the risk of peritonitis, which is inevitable if the sac ruptures, while the minor degrees of omphalocele carry the additional risk of strangulation at the neck of the sac if any more bowel forces its way out as the child becomes more active. Operation should be undertaken in a matter of minutes rather than hours and no special pre-operative treatment is required.



The next most urgent problem is that of obstruction, and the higher, the more urgent. Thus the first group is oesophageal atresia, with or without a fistula into trachea. Bitterly as I oppose artificial feeding, there is something to be said for the first mouthfuls being sterile water. If milk regurgitates from the blind pouch or leaks through the fistula, a fatal lipoid pneumonia is inevitable. The diagnosis is suggested by cyanosis and spluttering at the first effort to feed, and by excessive secretion of mucus between feeds; confirmed by passage (by mouth) of a fine rubber catheter; demonstrated by X-ray after instillation of lipiodol. My own experience is only of one of the lucky ones without fistula, and should I encounter another such, I should demand a gastrostomy and time to think. If a fistula exists, the risk is so bad that primary repair is worth trying, but I should still insist on a gastrostomy as well.

The next big group of obstructions are characterized by vomiting and provides our most worrying diagnostic problems.

Obviously the next probable site of obstruction is at the pylorus, but pyloric stenosis is not one of the forms of obstruction which presents as an emergency, and I shall leave it for discussion later.

The other common sites of obstruction are :—

1. *The Duodenum*, where the condition may be complete—duodenal atresia; incomplete—stenosis, compression (by a band or by a malrotation of mid gut, bringing the root of mesentery in front of duodeno—jejunal flexure).
2. *The Mid Gut*—(a) Malrotation may be complicated by rotation of the whole small bowel mesentery through 360°, 720°, or more. This may cause only partial obstruction at first, but gradually interference with blood supply, and strangulation.  
(b) Multiple patches of atresia are possible, but I have not seen them.  
(c) Meconium ileus, where the contents, rather than the bowel, are abnormal.
3. *The Large Bowel*, where obstruction may occur at any level and may be of any degree of severity, from a simple, membranous, imperforate anus, to cases where the colon ends in a blind pouch above the brim of the true pelvis.

Except in atresia, the obstruction is not complete and there is therefore time for reflection. As in cases of obstruction at any age, vomiting, constipation, and distension are the cardinal signs and vary in degree with the extent and level of the obstruction, though it is noteworthy, in passing, that the baby's condition deteriorates much more rapidly than one would expect in obstruction of the large bowel.

The points which have to be considered in any individual case are :—

1. Is obstruction—partial or complete—present?
2. Is operation urgent?
3. What is the site and nature of the obstruction? (note the order of these points).

Vomiting is the first disquieting symptom in anything above the pelvirectal junction and, of course, failure to pass meconium is the sign of abnormality below this level (these latter cases are diagnosed early, because every midwife thinks of imperforate anus and knows no other cause of failure to pass meconium, while she knows a lot of good—and bad—reasons for vomiting).

The types of vomiting that give me the most worry in this connection are :—

A. Many babies vomit altered blood, which is often so dark as to be “coffee ground” and, therefore, as I shall mention later, suggests a volvulus. If it occurs in the first twenty-four hours, and especially in the first twelve hours, it is most likely to have been swallowed, and a stomach washout will ultimately produce clear fluid and end the vomiting. The abdomen is not distended. Hypoprothrombinæmia is not seen till forty-eight to seventy-two hours, and by this time meconium, also containing altered blood, has been passed.

B. Cerebral œdema is a common cause of vomiting, which may be persistent and projectile and very suggestive of obstruction. This is my most difficult problem. If seen very early the fulness of the fontanelle, compared with the degree of dehydration, plus the history of delivery, should help diagnosis. Later, it is most difficult (and, in fact, I have seen babies become so dehydrated as to require intravenous fluids, which seems ridiculous in cerebral œdema). When there is no bile in the vomit, duodenal atresia is excluded, but one is left wondering whether (especially in a boy child) one may not be dealing with pyloric stenosis of unusually early onset.

When bile is present, the search for cornified epithelial cells in a meconium smear defatted with ether and stained with iodine might help, for these cells are derived from swallowed liquor and therefore indicate a through passage in the gut! In any case suggesting cerebral œdema it is worth trying the effect of chloral by mouth and magnesium sulphate per rectum.

In broad outline, persistent vomiting which is not relieved by gastric lavage and is not associated with a fontanelle which is doughy, or even tense, is possibly obstructive. A clean tongue and greedy appetite, with willingness to attack another feed immediately the first has been vomited, are suggestive. (The appetite is lost ultimately, but we have found this to be of fatal prognosis.) Bile is usually present in some or all of the vomits, but well diluted with milk, is hard to see in artificial light. Brown vomit not due to swallowed blood or to hypoprothrombinæmia means strangulation, usually of a volvulus. I think myself it is quite possible to distinguish this obstructive vomit, which is nigger brown, with no tinge of red, and has a finely particulate appearance, from altered blood, which inclines to be shreddy, and, indeed, the interior of the shreds is often distinctly red. Meconium is less in amount than usual and is bottle- rather than olive-green. Abdominal distension develops, with a large single epigastric bulge where the obstruction is not below the duodenojejunal flexure, and ladder patterns when the origin is lower. Rectal examination, of course, is never to be omitted. The little finger should be used.

Another cause of intestinal obstruction which I have not yet succeeded in diag-

nosing pre-operatively is meconium ileus, one manifestation of a condition which is due to—or, as some say, only associated with—a fibrocystic condition of the pancreas, with deficiency of pancreatic secretion. In our two cases the infants have suffered from persistent vomiting and increasing abdominal distension. Laparotomy disclosed small bowel filled with abnormal meconium, the removal of which—in obedience to transatlantic theory—was attempted, but whose glutinous tenacious nature—sticking to gloves and being withdrawn in long, black, sticky strands—created what I should imagine to be the most perfect conception of a surgeon's nightmare. Nor was the attempt to digest it by introduction of a pancreatin any more successful. In view of one's experience with cases of pancreatic fibrosis in later infancy, the fiasco is not perhaps to be regretted.

So much for getting the diagnosis to what you might call the stage of probability. Having decided on the probable presence of obstruction, the next decision is as to the urgency of operation. (Note: I put this before deciding on the site and nature of the obstruction, because you can sit and think while the theatre is being prepared.) In general, the working rule should be "look and see" rather than "wait and see." We find the risk of laparotomy less than the risk of delay, for there is one sword of Damocles which hangs over every obstructed baby, and that is aspiration pneumonia. Out of seven cases during 1949, four deaths out of five were due to pneumonia. Once obstruction has been seriously considered, a fine catheter should be left in the stomach and aspiration performed every few minutes. Apart from inhalation of gastric contents, the other factor determining urgency is strangulation. Brown vomit, toxic appearance, greyish cyanosis, raw, red tongue, tense and tender abdomen are all signs of strangulation and peritoneal irritation, but all, except the first, are of such bad prognosis that they entitle the case to be regarded as inoperable, though the hope of acquiring greater technical skill may encourage one to delude oneself! In a Mongol baby, brown vomit always seems to mean strangulated gut, and, regarded purely as a surgical problem, I should advise laparotomy on this evidence alone. We have had three of these cases: one was in a 3 lb. premature infant, where the presence of a suspected volvulus was confirmed at autopsy, the general condition not having justified operation; another Mongol was admitted with a small omphalocele, but a history of brown vomit. As it was a bad Mongol, the omphalocele was repaired with Nelson's eye on the probable complication, but at autopsy the entire small bowel was found to have herniated and strangulated through a gap in the mesentery. In a third case, diagnosed within twenty-four hours and operated on, duodeno-jejunoscopy for duodenal obstruction was performed, but the infant did not survive.

If strangulation can be excluded, operation is not a matter of urgency measured in minutes, though I should not be disposed to delay more than twelve hours. It must be admitted that two of our successes were operated on on the seventh and eleventh days of life respectively (this being the first chance we had!), and one of these was volvulus, which might well have strangulated before coming to operation.

The waiting period is used in narrowing down the diagnosis, and in pre-operative treatment (of which I shall speak later). The most useful diagnostic aid is a series of

radiographs to show the distribution of gas in the bowel, its distribution, and the degree of dilatation. In duodenal obstruction the stomach is greatly dilated. In late cases, small bowel can be so unbelievably dilated that it is hard to think one is not dealing with large bowel, but a picture taken with the infant suspended by the feet is useful if one suspects an atresia in the rectum. Barium, we feel, is dangerous in view of the possibility of converting a partial into a complete block. Our lethal experience, so to speak, is limited to one of the cases of fibrocystic pancreas, in which at post-mortem we found the barium packed in the sigmoid rather like cement. In cases where obstruction is probably not present we do allow ourselves the luxury, often with very salutary effect on the vomiting.

I shall leave the question of pre-operative treatment and post-operative care for the moment, and continue the discussion of the type of cases in which surgery is required in the neo-natal period.

Of the causes of obstruction which do not present as emergencies, pyloric stenosis is not, strictly speaking, always a disease of the neo-natal period (first four weeks of life), but since the cases which have come our way have all been under ten pounds weight, we have stretched a point in their admission to the Neo-Natal Sick Unit. This year's cases (fourteen) have comprised three under fourteen days; two between fifteen and thirty days; four between thirty to forty; two between forty to fifty; three between fifty to sixty. At the same time it is only fair to say that in all cases the history dated back to the first fortnight, so that one suspects that the diagnosis might profitably have been made earlier. In a baby on any reasonable feed and *a fortiori* in a breast-fed baby (a very strong argument, to my way of thinking, for breast feeding, at least for the first two or three weeks), the only probable causes of vomiting, after the earliest period, of which I have spoken already, are air swallowing (due to nasal obstruction, poor technique, or under feeding) and pyloric stenosis. The first step towards establishing the diagnosis is to watch a feed when the first two will be apparent, or, if the case be pyloric stenosis, visible peristalsis will be seen. If it has not been seen after watching for twenty minutes, arrange for a twenty-four-hour test-weigh. If this excludes under-feeding, watch another feed until peristalsis is seen. There is nothing which required greater patience (or which can make one feel more ridiculous) than sitting with one's eyes glued to a baby's epigastrium. I do not myself insist on feeling a pyloric tumour, though I am always relieved if it is palpable. Note, too, the lively, sensible way in which the baby goes about its job, albeit with a worried expression. Even when starving he is lively, clear-tongued, bright-eyed. Moreover, he is constipated. Vomiting associated with infection or indigestion is always associated with coated tongue and usually with diarrhœa.

All except one of our cases have been males. The single female was also singular in starting her symptoms on the fifth day of life, a singularity which nearly cost her her life, for, despite the discovery of a hypertrophied pylorus, I would not be persuaded that there was nothing more, and insisted on an inspection of the entire small gut. So much manipulation was followed by a venous thrombosis

(presumably), for she developed œdema of legs and ascites, and was extremely ill until recanalization or a collateral circulation occurred.

So much for the congenital malformations, which by threatening infection or obstruction, demand relief. They form the bulk of neo-natal surgery, but there are other types of cases which crop up from time to time and where operation has seemed, after earnest consideration, to be the lesser evil. Where a fatal outcome has seemed certain we have not hesitated to accept a 99.9 per cent. risk. These cases are rare, so that one does not see series of them, and I can give only isolated examples. Thus we have, for instance, obstructive jaundice. I am sure that such cases, if they are not consequent on survival from erythroblastosis fœtalis, are worth exploring, in the hope of being able to do a cholecystgastrostomy. Unfortunately, our only case so far had nothing emerging from the portal fissure except fibrous tissue, in which no lumen of any kind could be found. Again, we have, fruitlessly, removed a spleen from an eighteen-day-old girl with idiopathic purpura hæmorrhagica (Hb 30 per cent., R.B.C. 3.2 m., C.I. .7, Bleeding time thirty-six minutes, Clotting time two minutes, spleen palpable). She was admitted at age five days, between which and the age of eighteen days she had four blood transfusions totalling 1,100 c.c. After her fourth transfusion, while the hæmoglobin was eighty per cent., the spleen was removed. She stood the operation perfectly, but in forty-eight hours developed acute respiratory distress and died. Post-mortem examination showed fresh hæmorrhages throughout the body, including the lungs, the latter being the immediate cause of death.

Having got rid of the failures, there is a more cheerful side to the picture. Thus we have the story of the first-born son of a lady of 45. He had from birth a large abdominal tumour, the nature of which was considered to be either renal (hydronephrosis, polycystic kidney) or mesenteric. Removal, though tempting, was considered unjustifiable, since the tumour did not seem to incommode the child at all. However, at three weeks he was brought back hurriedly with fever, vomiting, constipation, and a tense, tender abdomen. Laparotomy allowed easy delivery (after aspiration) of a cyst the size of a grapefruit, with a torsion of its pedicle and hæmorrhage into its cavity, this accounting, I suppose, for development of the urgent symptoms.

Again, a boy was born with a hard, somewhat enlarged right testis, which did not seem to be tender. This was considered to be a teratoma and, for fear of malignant change, the testis was removed on the sixth day of life, histology revealing that malignant change was, in fact, already present. Interference at this early date would therefore seem to have been justified.

Or, consider the localized subdural hæmatoma, which has received more attention in U.S.A. than here. Perhaps it is commoner in transatlantic obstetric practice, for, although we have suspected it several times, we only have only seen it once—in a nineteen-day-old infant, admitted apparently moribund, with right-sided Jacksonian convulsions, bulging fontanelle, and papilloœdema. After twelve hours treatment with sedatives and rectal magnesium sulphate, it was thought that exploration was practicable. Under local anæsthesia a burr hole was made on the

left side and large amounts of clot washed out. Recovery was uneventful and the child is now three years old and perfectly normal.

There is one type of case which I should like to mention, because their inclusion in a neo-natal series is, to say the least, controversial, and that is the bad hare-lips. It is generally held that the mother will be quickly reconciled to the appearance of the infant, but I find them desperately sensitive and unwilling to have the child seen by their other children and by the neighbours. They are disheartened, and the difficulties of feeding are exaggerated, so that the children run the risk of never getting fit for operation. Since operation is feasible at 10 lb. weight if the baby is gaining, and since we have been able to feel it safe to retain an infant almost indefinitely without risk of hospital infection, we have, as a lifesaving expedient, kept the really bad hare-lips, which, in any case, cannot suckle, until the defect has been repaired.

The infections of the neo-natal period, hard though one may strive to avoid them, do occasionally occur and do (though rarely) call for surgery. One group of these cases is so unorthodox that no other series has, so far as I know, been reported. Perhaps other people who have had them have been willing to admit the presence of thrush in their units! Three years ago we had a case of thrush (I am thankful to say that our incidence is less than .5 per cent.), which spread to the œsophagus and made swallowing impossible. For three days, hydration was maintained with intravenous drip. However, the mouth lesions showed no improvement and it occurred to me that healing was not likely to be hastened by starvation. With great trepidation I therefore suggested a gastrostomy should be performed. Full feeds were administered by this route and in ten days healing was complete and the gastrostomy was allowed to close spontaneously. Since then we have admitted two other cases of œsophageal thrush, who have also been treated by gastrostomy, with equally satisfactory results.

Having surveyed at, I fear, tedious lengths, the conditions in the newborn requiring surgical intervention, I propose to step still further outside the limits proper to a physician and state my beliefs on pre- and post-operative treatment and even on operations themselves. My only excuse is, while the pædiatrician may know very little about surgery, one does get a good deal of experience of the metabolism of the newborn—those fragile, labile organisms in whom half a pint of water and half a teaspoonful of salt may make all the difference between death and survival.

The most important part of pre-operative treatment is early diagnosis, for the following reasons:—

1. Babies stand operation well in the first forty-eight hours of life, and badly for some time thereafter.
2. In the earliest days the fluid requirements are very small and dehydration does not have to be controlled. Conversely, if delay occurs, dehydration develops rapidly and is complicated by starvation, which hinders tissue repair after operation. Moreover, a dehydrated baby has no resistance to infection and thrush is especially prone to invade the dry, thirsty, mouth.

Whether seen early or late, no baby must ever come to operation with dehydration uncorrected. In almost all instances this can now be done by means of half-normal saline given by subcutaneous drip after the injection of hyaluronidase. Up to 250 c.c. can be given in one infusion by this method and the precious veins spared for the post-operative period which may require blood or plasma, as well as saline.

In all cases of obstruction a pre-operative injection of vitamin K should be given, since bacterial action in the bowel is not available for its synthesis.

Finally, the catheter, already passed into the stomach, should remain throughout (and after) the operation to avoid distension and inhalation of gastric contents.

The actual operative procedure is really not the province of the pædiatrician, but I have been borne with very patiently both by anæsthetists and surgeons. As regards anæsthesia, I believe in pre-medication with atropine and a barbiturate, followed by local anæsthesia, unless a great deal of manipulation, or an anastomosis, is likely to be required. Repair of an omphalocele, reduction of a volvulus, division of a band compressing the duodenum, and the cure of pyloric stenosis can all be carried out, without shock, under local anæsthesia.

A generous incision is always indicated. There is, for example, a great temptation to try to reduce an omphalocele through the pre-existing hole in the abdominal wall, but this involves too much handling of the intestines, and young infants are very prone to acute dilatation of the stomach and to paralytic ileus.

The actual operative procedure should be limited to absolute essentials: a limited first operation, with the certainty of having to do more later, is better than a good operation and a dead baby. The same rule applies to wound closure: a ventral hernia is better than a corpse.

Post-operative treatment naturally varies with the condition treated. Apart from uncomplicated pyloric stenosis, any operation involving interference with the bowel is usually safer with indwelling suction and parenteral fluids: saline, plasma, or blood given intravenously. After a period varying with the severity of the operation, small amounts of expressed breast milk may be offered and these quantities very gradually increased, full requirements being given by the third day.

The nursing of these cases requires special skill and patience and each pædiatrician will feel happiest with nurses whose training has been personally supervised. For my own part, I am convinced that midwives make the best neonatal nurses. Their experience of large numbers of healthy infants provides them with a standard of normality against which the slightest untoward sign stands out, so that impending trouble is foreseen and forestalled. Moreover, this same experience of thriving babies inspires a confident optimism which expects (and often secures) survival, even in desperate cases.

The location of a unit such as that from which the present material has been drawn is a matter of opinion. Again, for my own part, I am certain that it should be attached to a maternity, rather than to a children's hospital. In a maternity hospital the standard of asepsis is probably higher than in any other institution, and the incidence of infection extremely low. Cross-infection should therefore be

negligible. This makes it certain that an infant will incur no risk other than that inherent in its own illness : that a pyloric stenosis will not, for example, develop gastroenteritis, nor a cerebral hæmorrhage, thrush.

Finally, I should like to express my gratitude to all those who have made this paper possible : to Mr. Lavery and the other surgeons of the Belfast City Hospital, who have borne with me so patiently; to Dr. Field, our pædiatric registrar; to a series of house physicians, who have never known what it is to have any fixed off duty; and, above all, to a nursing staff whose skill and enthusiasm have done more to secure the survival of these babies than all the rest of us put together.

## REVIEW

TUBERCULOSIS : A GLOBAL STUDY IN SOCIAL PATHOLOGY. By John B. McDougall, C.B.E., M.D., F.R.C.P.(Edin.), F.R.F.P.S.(Glas.), F.R.S.E. Pp. 445. Edinburgh : E. & S. Livingstone Ltd. 1949. 32s. 6d.

DR. JOHN McDOUGALL, who is now Tuberculosis Advisor to the World Health Organisation, has had a wide experience of every aspect of tuberculosis in England and throughout Europe. The objects of this work, first the presentation of information on infection, morbidity, and mortality from tuberculosis, and second, an analysis of the factors considered to have a bearing on the problem, have been very fully achieved.

The first part consists of a review of the world incidence of tuberculosis, so far as this is known. The author has unique opportunities for acquiring and weighing the relevant data and has been able to present a comprehensive picture of world epidemiology. This part of the work will be indispensable to the tuberculosis workers for reference.

There follows a critical review of the factors which influence infection, morbidity, and mortality, including an account of the vulnerable age-groups, of the decline of mortality in Western Europe and North America, and of the changing picture regarding infection. An interesting section compares tuberculosis mortality figures in certain countries with mortality rates from all causes and from other respiratory diseases. The implications of these findings concerning the accuracy of tuberculosis statistics is discussed. A full section deals with the new statistical method of studying mortality by the follow-up of the "cohort" of each decade throughout age-periods from infancy to old age.

New work on variations in virulence of the tubercle bacillus is described, but no evidence is brought forward that there has been a decline in virulence since the first studies at the beginning of the century.

The author appears to regard exogenous superinfection as relatively unimportant, regarding all manifestations of disease as essentially the products of unhealed primary lesions. The conditions of primary infection and the size of the infecting dose are considered to be of importance : "There appears to be reasonable grounds for believing that the variations in the number of infecting bacilli which lie within the range of possibility in natural infection in man are sufficient to exert an influence upon the progress of infection."

There is a well-documented account of environmental factors, considered critically in relation to the various countries studied and their economic, social, and cultural position. Climate, population density, employment and occupation, housing, nutrition, and social life and habits are fully dealt with in separate sections.

The last part of the book deals with the measures which are being taken throughout the world to evaluate the tuberculosis problem and to bring the disease under control. There is a full account of BCG vaccination and a description of the rapid extension of this method of prevention in many lands. Throughout the work the vast mass of information has been skilfully handled and presented attractively.

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# Neo-Natal Anæsthesia

By HAROLD JEFFERSON, M.B., D.A.

Visiting Anæsthetist, Belfast City Hospital

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## *A Paper read to the Ulster Medical Society*

As the ages of the patients concerned may vary from a matter of hours to weeks, and, as feeding difficulties have usually rendered them underweight, it will be appreciated that these factors, when reinforced perhaps by vomiting and increased liability to respiratory infection, make anæsthesia in infants one of the major problems to be faced in anæsthetic practice.

When operation is not a matter of urgency, as for instance in hare-lip, then operation should, if possible, only be undertaken in mild weather, preferably during the summer months, when the incidence of respiratory infection is at its lowest level.

With regard to pre-operative care, the babies should be nursed in a warm sunny cubicle, and, if possible, have a separate nursing staff. The chest, nose, throat, and ears are carefully checked, and any abnormal physical signs, such as a rise in temperature or naso-pharyngeal infection are absolute contra-indications to operation, except in emergency cases. If dehydration is marked, an intravenous drip should be running. In this respect hyaluronidase has proved quite useful for hypodermoclysis.

In connection with diet, if feeding is not impossible, due to the condition present, the baby continues with its normal diet until the last feed before operation. The last feed is diluted with an equal quantity of 10 per cent. glucose and only half of the usual quantity is given. An interval of two to three hours elapses before operation. A similar feed is given after operation. The glucose insures that the liver will be well bolstered against the toxic effects of the anæsthetic.

In the cases of hare-lip the infants will, of course, have been treated for anæmia and brought up to weight by careful feeding.

Premedication consists of atropine alone, as any respiratory depression is dangerous, the dose being 1-200th to 1-150th grain, depending on the size of the infant. With the bigger infants .07 gramme of sodium soneryl may be given if the infant is very restless. Where obstruction in the gut is present a small stomach tube should be in position.

The safest anæsthetic agent in neo-natal work appears to be ether, delivered with a liberal supply of oxygen. Ethyl chloride should never be used, as it is a potent cardio-respiratory depressant.

The ether, when given with nitrous oxide and oxygen and *followed* by  $\frac{1}{2}$  per cent. novocaine infiltration of the abdominal wall gives, in our view, the best available anæsthetic, both from the baby's and the surgeon's point of view.

*Clothing during operation is important* :—It is essential that these babies must not become too hot during operation. A baby's heat regulating mechanism is both highly unstable and sensitive, and high temperatures in babies are easily provoked. The heat-regulating centre is depressed during anæsthesia, but recovers rapidly at its conclusion. With the application of excessive external heat, the baby's temperature may rise to 105° or 108° Fahrenheit. In addition, atropine raises the metabolic rate and tends to abolish sweating. For these reasons, therefore, it is better to have the baby on the cool side rather than to be too warm. It should be stripped down before induction and simply covered with a linen sheet or towel. Mackintosh sheeting should never be used.

When we consider the actual technique of anæsthesia, there are three requisites for the ideal administration.

The first requisite is liberal oxygenation through a perfect airway, thereby ensuring the absence of anoxia, protection against the toxic effects of the anæsthetic agent, and the avoidance of congestion and bleeding.

The second requisite is the avoidance of any form of increased mechanical effort. This covers both phases of respiration and will be due to obstruction of the airway.

Thirdly, excessive building up of carbon dioxide is to be avoided, as this is a toxic substance, causing additional respiratory effort, a raised blood pressure, and increased bleeding.

To turn for a moment to the induction and maintenance of the anæsthetic, we have given up the use of the "rag and bottle ether" method, for, although it is very good, we consider it is too clumsy to use whilst working so close to the surgeons, and, besides, the vapours the baby will inhale are much too chilly and add to the risk of respiratory infection. The method now adopted is to use the Boyle machine with the semi-closed attachment and the rebreathing bag closed off. The rubber mask is at all times held about one inch from the face, so that there is no rebreathing, and the baby breathes anæsthetic mixture and air. The smallest rubber mask made has a capacity of 90 c.c. and if the mask were held close to the face this would be a great addition of dead space to a small tidal air of about 180 c.c.

The baby then is rendered unconscious with nitrous oxide and air and oxygen is added up to 40 per cent. with ether until a moderate depth of anæsthesia is reached. When this stage is reached the surgeon may now infiltrate skin and abdominal muscle with  $\frac{1}{2}$  to 1 per cent. novocaine and adrenalin. This local anæsthetic helps greatly in the relaxation of the abdominal wall and also limits hæmorrhage. We have now come to the conclusion that it is better to induce general anæsthesia from the start, as it is very difficult to induce general anæsthesia half way through an operation under local, when the infant has already had a pull on its mesentery, and is struggling violently to escape from the whole business. The local anæsthetic is therefore used as an adjunct to the general anæsthetic, and not vice versa, as with anæsthesia in adults.

If, as may happen during the induction for a hare-lip operation, the baby develops cardio-vascular collapse, becoming very white and shocked, it should be

returned to the ward and operation deferred for three months. This collapse is very liable to occur when ethyl chloride is used, and is not, in my opinion, in any way due to an idiosyncrasy on the part of the infant.

During the operation great care must be taken to see that the infant does not become too lightly anæsthetised, as this is probably the most common cause of trouble and actual disaster. One should always, if in doubt, err on the side of depth of anæsthesia, because if one is anæsthetising too timidly the anæsthetic will become light, vomiting or laryngeal spasm, or both, may occur, to be followed in an amazingly short space of time by the most frightening cyanosis, which the baby will tolerate very badly. The same sequence of events, in a delicately balanced nervous mechanism, is liable to occur if an airway is used in these infants. These airways are liable to cause copious salivation in an infant, leading to obstruction and perhaps again spasm of the larynx.

In the operation for hare-lip, usually undertaken at from eight to twelve weeks, the induction of anæsthesia is carried out in a similar manner to that mentioned and when a moderately deep plane of anæsthesia has been reached, a Magill endotracheal tube, size 0 or 00, is passed orally with the aid of a laryngoscope. Extreme gentleness is essential, and no bruising of the larynx or cords is permitted. If bruising does occur through passing a tube in light anæsthesia, it is probably safer to abandon the operation for some weeks, as laryngeal œdema and obstruction are almost certain to occur post-operatively.

It should be remembered, when considering how long the operation should continue in these infants, that the infant's larynx is extremely tiny, and that the passage of even an adequately-sized tube cuts the diameter of the trachea by about one-third. That is to say, some important degree of respiratory obstruction is present before the operation even begins.

The tubes used are either of stiff rubber, or are armoured with wire to prevent kinking. None of them is really satisfactory, as the rubber is too thick, or the tube kinks, and even when the armoured tube is stitched to the tongue or the gag, it tends to arch up and embarrass the surgeon.

The tube is brought out of the mouth and is connected by a T piece or a sidevent to the supply of anæsthetic. No rebreathing is permitted here either, and only the lightest anæsthesia is required during the operation.

The post-operative anæsthetic complications are: firstly, bronchitis or bronchopneumonia, with or without atelectasis—diffuse or massive; and secondly, in the case of hare-lip surgery, laryngeal œdema, which is recognised a few hours after operation by stridor, and may go on to gross obstruction necessitating tracheotomy. This latter condition must always be due to rough handling during intubation.

Anæsthesia has been described as a journey, starting with consciousness and ending in death, a halt being called in that journey when the required depth of anæsthesia is reached. If it is remembered when working with infants that this journey may be very short and that every known anæsthetic accident and disaster may occur without the slightest warning, then, by taking the most meticulous care, it is hoped that the anæsthetic morbidity and mortality rates will still improve.

# A Case of Ureterocele in an Infant

By MAURICE LAVERY, M.Sc., M.B., F.R.C.S., F.R.C.S.(E). and

R. R. DICKSON, M.B., F.R.C.S.

URETEROCELE is well known to genito-urinary surgeons, but it has attracted very little attention from pathologists or biologists. The usual description is that the primary condition is a pinhole ureteric orifice, with a dilated ureter above and a cystic projection into the bladder below, with the ureteric orifice on the surface of the cyst. Cystoscopically, the swelling expands and collapses according to the pressure in the bladder. A certain amount of movement in the cyst wall has been observed, and if this is a ureteric movement, it would seem to indicate that ureteric muscle is present in the wall. It may be unilateral or bilateral, and is often discovered accidentally, or mild obstructive renal symptoms have been present in some cases. It is treated by intravesical diathermy destruction of the wall. The size of the ureterocele varies, a common size is about an inch diameter. Occasionally the cystic swelling in the female has prolapsed through the urethra and appeared on the surface. A case of this type was described in the *British Journal of Surgery* (Volume 29), by Ian McPherson. This case was a female, aged 27. The case which we report is a young female infant with a severe congenital anomaly of the hand. This associated deformity seems to us to emphasize the congenital nature of this rare condition. It is worthy of note that the condition ureterocele is not mentioned in Thompson Walker's *Genito-urinary Surgery*, 1946, or in Hamilton, Boyd, and Mossman's *Human Embryology*, 1947.

## CASE REPORT

H. C.—Female, aged 8 months.

This was the second child, the first being born in June, 1947. The birth was normal full term. The mother stated that she fell at six months. The child was born with a congenital amputation of the left hand at the carpo-metacarpal joint level. Admitted to the Ava Children's Hospital, 18/8/49.

On the day of admission the mother noticed a dark-red swelling about half an inch in diameter in the vulva. This was replaced in the bladder by her own doctor, who referred the child to hospital for observation. On admission nothing abnormal was found, other than the deformed hand, but on the 20/8/49 a soft, fluctuant, purplish-red mass, one and a half inches in diameter, was found projecting from the urethra (fig. 1). This swelling had an expansile impulse when the child cried. The swelling was reduced into the bladder with difficulty and it immediately recurred. No ureteric orifice was discovered on the surface. The diagnosis of ureterocele was confirmed by aspiration of urine through a hypodermic needle.

On 21/8/49, without anaesthesia, the greater part of the projecting tissue was excised with the diathermy needle and the remainder returned to the bladder (fig. 2).

The excised portion showed evidence of vascular obstruction and no muscle fibre was demonstrated microscopically.

On 29/8/49 intravenous pyelography showed both kidneys functioning well. The left side was slightly dilated. There was a constant circular filling defect in the left side of the bladder base. Cystogram showed no abnormality in the bladder, but the opaque fluid passed up into a dilated left ureter.

On 11/9/49 cystoscopy showed a moderate degree of cystitis, slight intravesical bulging of the right ureter, and a more marked bulging of the left ureter.

The cystitis cleared up satisfactorily and the infant was discharged on 28/9/49, free from symptoms. We have to thank Dr. Claude Field for carrying out the difficult investigations on this infant.

#### REFERENCE

McPHERSON, I. : *Brit. J. Surg.*, 29, 294; 1942.

## REVIEWS

**OXIDATION-REDUCTION POTENTIALS IN BACTERIOLOGY AND BIO-CHEMISTRY.** By L. F. Hewitt, Ph.D., B.Sc., F.R.I.C. Sixth Edition. Pp. 215. Edinburgh : E. & S. Livingstone Ltd. 1950. 20s.

This work was originally published in 1931 by the London County Council as a two-shilling booklet. Many biochemists and bacteriologists have been grateful to Dr. Hewitt for providing a relatively simple and clear introduction to the difficult subject of oxidation-reduction potentials, and its value is indicated by the fact that it has now reached the sixth edition. In this edition the publication has reached the status of a book. It is considerably enlarged, contains several new chapters, and has a substantial binding. Unfortunately, the price has increased tenfold as compared with the pre-war editions.

The earlier chapters remain an introduction to the study of oxidation-reduction potentials, including the meaning of the rH scale. Like Mansfield Clark, who originated this scale, the author recommends that the use of the scale be discontinued, as it is liable to cause confusion. In this connection it may be pointed out that in a new book "Multi-Enzyme Systems," by Malcolm Dixon, a strong case is made for wider use of the rH scale, as it has many advantages, notably the simple relationship between the rH value and free energy of a reaction.

The book covers a wider field than might be expected from the title. It deals with some of the techniques of pH measurement and with polarography, and contains chapters on bacteriological applications, and on chemotherapy and antibiotics.

The usefulness of the book is much increased by the provision of a thirty-page bibliography

D. C. H.

**A HISTORY OF OTO-LARYNGOLOGY.** By R. Scott Stevenson, M.D., F.R.C.S. (Ed.), and Douglas Guthrie, M.D., F.R.C.S.(Ed.). Pp. 155 with index. Edinburgh : E. & S. Livingstone. 1949. 17s. 6d.

A most excellent small book of 137 pages, written in an easy manner, with a minimum of irrelevant matter, and dealing with the history and evolution of the speciality of oto-laryngology in a very complete manner, free from undue technicality. It deals very adequately with the famous men who have played an important part in the progress of this branch of surgery, and with the gradual improvement, and widening of technique, and knowledge up to the present day.

The illustrations are excellent, and the liberal sprinkling of portraits makes many of the outstanding names become more nearly personalities.

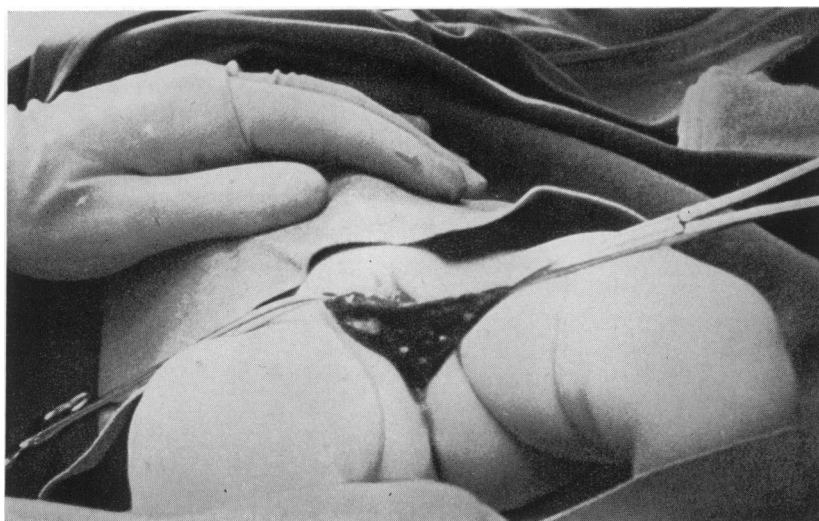
F. A. MacL.

A CASE OF URETEROCELE IN AN INFANT



**Fig. 1**  
Showing the prolapsed ureterocele.

A CASE OF URETEROCELE IN AN INFANT



**Fig. 2**

Condition after puncture of the ureterocele, showing distension of prolapsed tissue.



The excised portion showed evidence of vascular obstruction and no muscle fibre was demonstrated microscopically.

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The earlier chapters remain an introduction to the study of oxidation-reduction potentials, including the meaning of the rH scale. Like Mansfield Clark, who originated this scale, the author recommends that the use of the scale be discontinued, as it is liable to cause confusion. In this connection it may be pointed out that in a new book "Multi-Enzyme Systems," by Malcolm Dixon, a strong case is made for wider use of the rH scale, as it has many advantages, notably the simple relationship between the rH value and free energy of a reaction.

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D. C. H.

**A HISTORY OF OTO-LARYNGOLOGY.** By R. Scott Stevenson, M.D., F.R.C.S. (Ed.), and Douglas Guthrie, M.D., F.R.C.S.(Ed.). Pp. 155 with index. Edinburgh : E. & S. Livingstone. 1949. 17s. 6d.

A most excellent small book of 137 pages, written in an easy manner, with a minimum of irrelevant matter, and dealing with the history and evolution of the speciality of oto-laryngology in a very complete manner, free from undue technicality. It deals very adequately with the famous men who have played an important part in the progress of this branch of surgery, and with the gradual improvement, and widening of technique, and knowledge up to the present day.

The illustrations are excellent, and the liberal sprinkling of portraits makes many of the outstanding names become more nearly personalities.

F. A. MacL.

The excised portion showed evidence of vascular obstruction and no muscle fibre was demonstrated microscopically.

On 29/8/49 intravenous pyelography showed both kidneys functioning well. The left side was slightly dilated. There was a constant circular filling defect in the left side of the bladder base. Cystogram showed no abnormality in the bladder, but the opaque fluid passed up into a dilated left ureter.

On 11/9/49 cystoscopy showed a moderate degree of cystitis, slight intravesical bulging of the right ureter, and a more marked bulging of the left ureter.

The cystitis cleared up satisfactorily and the infant was discharged on 28/9/49, free from symptoms. We have to thank Dr. Claude Field for carrying out the difficult investigations on this infant.

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## REVIEWS

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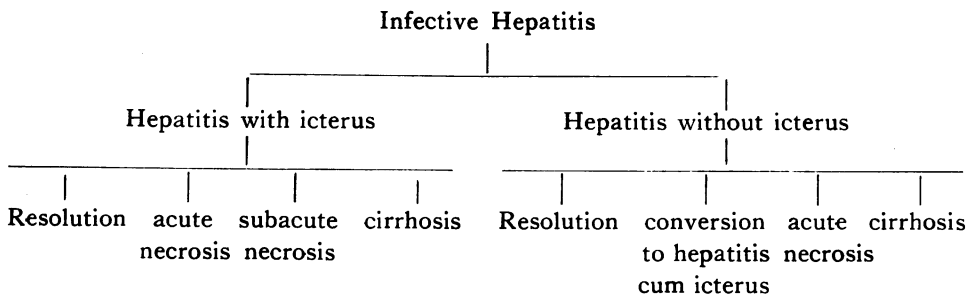


the inoculation of Brazilians in 1940, and American troops in 1942 with yellow fever vaccine prepared with human serum. Outbreaks of jaundice were reported in England about the same time, following the injection of mumps convalescent serum, yellow fever vaccine, and pooled human serum. It can thus be seen that homologous serum jaundice may occur after any of the following :—

1. Serum, plasma, and blood transfusions.
2. Protective inoculations in which serum, lymph, or some other human product is used.
3. Injection with a syringe which has not been properly sterilised, following its use in another patient.
4. Blood sampling.

It is now known that both infectious hepatitis and homologous serum jaundice are due to infection with a virus. The same virus is not responsible for both diseases and there are several important distinctions between them. The virus of infective hepatitis is present in the blood and fæces and is usually transmitted by the oral route, although it may be transmitted by the injection of infected blood products. Its incubation period is about thirty days. The virus of homologous serum jaundice is transmitted parenterally by infected blood products and the incubation period is about one hundred days. It has been found in the blood of apparently healthy individuals. Infection by one virus does not protect against infection by the other.

The clinical features of both diseases are well known and very similar; digestive disturbances, jaundice, and fever. There is, however, no doubt that many cases occur without jaundice, and that there are many variations in the clinical course. This has been summarised by Damodaran, as follows :—



Opportunities for the study of the pathology of hepatitis were few until the world war. Cases which died were of the fulminating variety and were called yellow atrophy. This being considered by most authorities to be unrelated to infective hepatitis, although as long ago as 1912 Cockayne came to the conclusion that catarrhal jaundice and acute yellow atrophy could be due to the same cause. Later, autopsies performed on soldiers suffering from catarrhal jaundice who died from trauma and the performance of liver biopsies has added greatly to our knowledge of the pathology of infective hepatitis in its non-fatal form.

In 1943 Dible, Michael, and Sherlock reported on biopsy material from fifty-six cases of epidemic hepatitis, arsenotherapy jaundice, and serum jaundice. They

found that the changes in the liver were related to the severity and duration of the disease. The main changes are those of hepatic cell degeneration, particularly in the centre of the lobule, and leucocytic and histiocytic reaction and infiltration, which is most marked in the region of the portal tracts. In mild cases the periportal cell accumulation predominates the picture, while in severe cases there is more marked hepatic cell necrosis and widespread leucocytic infiltration. Cases were seen in the stages of retrogression, necrosis, nodular hyperplasia, and cirrhosis.

We now come to an attempt to evaluate the incidence of cirrhosis, following hepatitis. Jones and Minot (1923) described two cases of cirrhosis following catarrhal jaundice. Cullinan (1936) mentions catarrhal jaundice as a possible cause in twenty cases of subacute hepatic necrosis. This view was also supported by Bergstrand in Scandinavia and also by Watson and Hoffbauer. More recently the question has been investigated more thoroughly due to the stimulus of the large epidemics of hepatitis which occurred during the late war. Sherlock (1948) studied nine cases by aspiration biopsy. Six of these had had infective hepatitis, and three serum jaundice following arsenotherapy for syphilis. Three of these cases had well-marked cirrhosis histologically, but no signs or symptoms and biochemical tests were normal. Four cases had cirrhosis with portal circulatory obstruction. Two cases had cirrhosis and hepatocellular failure, from which one died. Trotter (1945) describes four cases of cirrhosis following an outbreak of hepatitis in a diabetic clinic. Two of these patients died 300 and 110 days after the onset of jaundice. Fearnley (1947) describes the case of a soldier who died from cirrhosis six years after catarrhal jaundice. He had evidence of liver failure, hæmatemesis and marked ascites before death. Wyllie and Edmunds (1949) describe six fatal cases of hepatitis in children. Four of these cases died within a few weeks of the onset of hepatitis from liver cirrhosis, while the two who survived for periods of 5½ years and 16 months had well-marked cirrhosis.

The question of cirrhosis following hepatitis can be further investigated by considering the number of cases of cirrhosis which have an antecedent history of jaundice. The following series are considered.

Bloomfield—41 cases—10 per cent. antecedent jaundice.

Ratroff and Patek—386 cases—6.5 per cent. antecedent jaundice.

Eppinger—269 males—14 per cent. antecedent jaundice.

107 females—12 per cent. antecedent jaundice.

Howood and Watson studied the histories of one hundred cases of cirrhosis and one hundred control cases and found the following:—

100 cases of cirrhosis—17 per cent. had had clear-cut infective hepatitis.

100 controls—3 per cent. had had possible attacks of hepatitis.

We must then come to the conclusion that, while infective hepatitis and homologous serum jaundice are essentially non-fatal diseases with a mortality of under 0.5 per cent., and although in the majority of cases complete resolution of the disease occurs, there is also, I feel, no doubt that in some cases the disease is progressive. It may progress over a matter of weeks or months to death from sub-

acute hepatitis or cirrhosis, this being more common in those cases where the jaundice relapses and convalescence is prolonged.

A few cases have also been reported where, after an interval of several years of normal health, clinical cirrhosis has supervened, and, while the evidence that the cirrhosis in these cases is due to antecedent hepatitis is not absolute, it can certainly be considered as highly suggestive.

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#### REVIEW

THE CYTOLOGY AND LIFE-HISTORY OF BACTERIA. By K. A. Bisset.  
Pp. 136. Illustrations 43. Edinburgh : E. & S. Livingstone Ltd. 1950. 18s. 6d.

THE study of the bacterial cell for its own sake has had comparatively little time devoted to it. The relationship of bacteria to disease which dominated the scene in the early days postulated many problems, and the energies of workers were necessarily devoted to solving them.

The detailed and intensive study of the bacterial cell as a cell was and is limited both by its minute size and the techniques available.

Dr. Bisset's aim in this book is "to attract the attention of other workers in this and related fields to the importance of the conception of the bacterial cell as a living cell with the same function and structure as other living cells." Theories previously accepted may be re-examined in the light of the evidence here produced. All aspects of morphology are discussed, but the more controversial points, such as the presence or absence of nuclei in the bacterial cell and the existence of a life cycle, have much attention. Evidence is advanced for the sexual reproduction of bacteria and for the occurrence of a life cycle in groups, such as the myxobacteria and streptomycis, as well as the streptobacillus moniliformis.

While it is improbable that the more conservative workers will accept the evidence adduced for these occurrences, it is likely that the book will shake some out of an uncritical acceptance of text-book theory and may stimulate further work along these lines. It is primarily a work for the specialists in microbiology and kindred fields.

The book is beautifully produced, the diagrammatic illustrations are particularly clear and numerous, and the photographs, which are remarkably good, do exemplify the difficulty of being precise about the detailed internal structure of these tiny cells.

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# Naso-Pharyngeal Fibroma

By KENNEDY HUNTER M.B., F.R.C.S.(EDIN.), and  
HUGH G. BIGGART, M.B., F.R.C.S.(EDIN.)

W. McC., age 17 years. This patient attended E.N.T. Extern and was admitted to the Royal Victoria Hospital on the 16th September, 1949.

He complained of nasal obstruction for one year which was noticed first on the right side and later on the left. Four months before admission he had a severe epistaxis, during which he lost a considerable amount of blood. He was treated six months previously for meningitis and was in Purdysburn Fever Hospital for three weeks. He also complained of a certain degree of deafness.

On examination, he was a healthy looking male subject.

*Throat.*—Tonsils were large, no infection.

*Pharynx.*—There were several large veins visible on the posterior pharyngeal wall going up into the naso-pharynx.

*Nose.*—The septum was deviated to the right. There was hypertrophic rhinitis and muco-pus present in both nostrils.

## POSTERIOR RHINOSCOPY.

The naso-pharynx was filled by a large tumour which was blocking both choanæ and on palpation bled profusely.

*X-ray.*—There was a soft tissue mass approximately 2 cms. in diameter projecting forwards and filling up the naso-pharynx. There was considerable erosion of the roof of the naso-pharynx with extension of the soft tissue into the sphenoidal air sinus. The right frontal sinus showed loss of translucency, and mucosal thickening was present in both antra.

A biopsy of the tumour was carried out and this was attended by very severe hæmorrhage, which required almost an hour to stem.

In view of the difficulties of surgery in this case a course of deep X-ray therapy was given, but no change in the size of the tumour or the patient's symptoms was noted. It was finally decided that surgical removal should be attempted. Pre-operatively the patient was blood grouped and a transfusion started on the table.

## OPERATION.

The uvula and soft palate were split in the midline and retraction carried out. This gave a moderate exposure of the tumour. The tumour was incised with a cutting diathermy current and bleeding became extremely severe. It was removed by blunt dissection. The tumour was very adherent to the surrounding walls.

It was found to have eroded the floor of the sphenoidal sinus and exposed the basi-occiput. It extended into the nose and invaded the free edge of the hard palate.



During this procedure the patient lost about three pints of blood, which were replaced by continual transfusion during the operation.

A large pack soaked in thrombin-topical was inserted in the post-nasal space and the following day this was removed under morphia without further bleeding.

Post-operatively the patient developed a mild right basal pneumonia, but from this and the operation recovery was uneventful and complete. Hearing returned to normal and the nasal airway is now completely free.

#### PATHOLOGY.

*Macroscopically.*—The tumour was almost three inches long, one and a quarter inches wide, and yellowish in colour.

*Microscopically.*—The tumour presented the histological appearance of a myxomatous fibroma showing stellate cells in a mucinous background with few fibres and well formed blood vessels.

#### DISCUSSION.

Benign tumours of the naso-pharynx or any non-malignant tumefaction are enlargement of tissues encroaching upon the normal lumen and by so doing interfering with its normal relations and functions. Many types of tissue have been reported in these growths. Excluding adenoidal hypertrophy a fairly complete list would include :—

fibroma, hæmatoma, neuro-fibroma, lipoma, xanthoma, dermoid, teratoma, chondroma, and several types of cystic and polypoid structures.

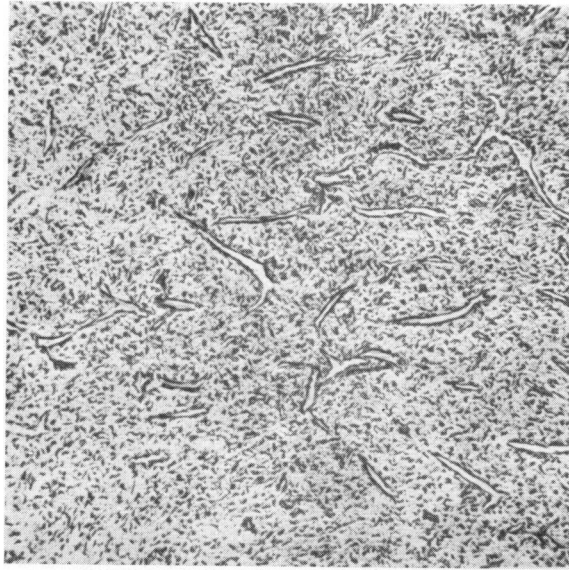
Of these, the most common is the fibroma. The origin of these fibromata is obscure but it is thought they arise from the periosteum of the bony structures making up the walls of the naso-pharynx. They are said to develop a few years prior to puberty and have a tendency to degenerate in the middle 20's. Many observers doubt the latter part of this statement.

Pathologically, they usually show numerous blood vessels with much dense fibrous tissue and a few cells. In earlier growths cellular deposits may be numerous. Treatment is extremely difficult, but final resort to surgery is usually necessary, as deep X-ray therapy has little effect.

Symptoms of epistaxis or naso-pharyngeal bleeding, which may be severe, deafness, tinnitus and nasal obstruction with erosion of bone and improper sinus ventilation, are described.

This case, then, produced the typical symptoms of such a tumour with the added feature of meningitis, probably due to infection through base of skull.

NASO-PHARYNGEAL FIBROMA



Microscopically, tumour shows numerous stellate cells with few fibres. Many large vascular spaces are seen in this field.



Photograph of Tumour.

# Ulster Neuro-Psychiatric Society

First Meeting held 18th November, 1949

## SCIENTIFIC BUSINESS

DR. TERENCE FULTON showed the following case.

Kippel-Feil Syndrome, and associated congenital abnormalities.

S.Y. Male. Aged 35.

*History.*—As a child the patient had high-arched feet, requiring special shoes. He was short-sighted, had to sit at the front of the classroom, and had a larger head than his contemporaries. During his teens he had attacks of vomiting and severe frontal headache, lasting for one or two days every month or so. No headaches since then.

He had no disturbance of gait until about ten years ago, when the (R) foot began to drag on the ground, so that he wore away the outer aspect of the sole. Five to six years ago the (L) foot was affected, and since then stiffness of the legs has gradually increased without remission. During the past three to four years he has had some difficulty in starting the act of micturition.

*Education.*—He left school at the age of 14, being in the seventh standard, and was considered a bright student.

*Family History.*—Irrelevant.

## EXAMINATION

*Skeletal Abnormalities.*—

1. "Mushroom" skull. Short neck, low hair-line, and limited, but painless, lateral flexion and rotation movements.
2. Cervical lordosis, and scoliosis to the (L), with secondary thoracic scoliosis to the (R). (Facial asymmetry is probably due to the cervical scoliosis.)
3. Thin distal segments of the arms and legs, the hands being narrow and small.
4. Bilateral pes cavus and "Friedreich toes."

*Neurological Abnormalities.*—

1. Bilateral primary optic atrophy, with generally constricted visual fields, and a superior temporal quadrant defect in the field of the (R) eye.
2. Bilateral horizontal nystagmus, greater to the (L) than the (R).
3. Bilateral congenital ptosis, greater on the (L) than the (R), with compensatory overaction of the frontalis muscles.
4. Exaggerated jaw jerk. Poorly executed movements of the tongue, and bilateral pyramidal signs in the legs (L) > (R), associated with spastic gait. The pyramidal signs in the arms are much less in extent, and are greater on the (L) than on the (R). Bladder function is involved by the pyramidal lesions.
5. Minimal cerebellar signs in the (L) hand.

*Other Abnormalities.*—

1. Abnormally small testes and poverty of secondary sex hair.
2. Severe myopia.

INVESTIGATIONS

1. *Lumbar Puncture.*—Initial pressure 100 mm. of fluid, and an excessive rise and rapid fall were observed during coughing and abdominal compression; though on jugular compression, fluid rose quickly, the fall was slow and incomplete. C.S.F. was normal, except for 50 mg. per cent. of protein.
2. *X-rays.*—
  - (a) *Skull.*—General enlargement of the vault, thinning of the bone, and marked convolitional markings, with erosion of the posterior clinoid processes. No platybasia. Findings indicate increased intracranial pressure.
  - (b) *Cervical Spine.*—Complete failure of segmentation of C 2-3 and partial failure of C 5-6, involving only the bodies. Associated C 6 spina bifida. The appearances are those of Kippel-Feil deformity.
  - (c) *Lumbar Myelogram.*—The contrast medium was arrested at the level of the foramen magnum, due to what is probably tonsillar herniation.
  - (d) *E.E.G.*—Non-specific abnormal out-bursts of theta activity, probably of deep-seated origin.

COMMENTARY

The skeletal abnormalities of the Kippel-Feil syndrome are apparent, both on clinical and radiological examination. The appearance of the head in association with this deformity, suggested that the neurological signs might, in part, be explained by basilar impression. This, however, was excluded by the radiological examination, and it was also evident that the pyramidal lesion must be placed above the level of the upper pons, because of the exaggerated jaw jerk. Such a lesion would not be produced by an uncomplicated platybasia, for, in this condition, the site of highest compression is in the pons, and involvement of the medulla and upper cervical cord are usually much greater in degree, with resulting lower cranial nerve signs. While hydrocephalus may occur in patients with platybasia, and give rise to hypothalamo-hypophyseal signs, more usually when hydrocephalus arises, the patient experiences symptoms of rapidly increasing intracranial pressure.

The C.S.F. manometrics at lumbar puncture indicated a partial block, and this was confirmed by the lumbar myelogram, an "extramedullary" type of arrest of the contrast medium occurring at the level of the foramen magnum. The myelographic appearances were consistent with the presence of Arnold's, or the Arnold-Chiari malformation, but it was felt, on clinical grounds, that the cerebellar manifestations, which, according to List (*Arch. Neurol. and Psych.*, 1945) are presenting signs, were relatively insignificant. The high pyramidal signs can be explained by the effect on the cerebral cortex of a chronic hydrocephalus, the result of the Arnold-Chiari malformation, but their asymmetry requires the presence of other lesions in the pyramidal pathways.

The myelographic appearances of mild tonsillar coning might well be the result of the chronic hydrocephalus, and not its cause. If there were already, however,

a minor degree of Arnold's malformation, then the resulting hydrocephalus would tend to accentuate the deformity by pushing down further into the upper part of the vertebral canal, the congenitally displaced cerebellar tonsils, and neighbouring verus.

The association of spina bifida and the Arnold-Chiari malformation is common, and a cervical spina bifida is an occasional accompaniment of the Kippel-Feil syndrome. Accompanying such a vertebral deformity there may be a syringomyelia, but in this patient there was no evidence of such widespread and characteristic sensory changes as one would expect if this were so.

In conclusion, it was felt that, while in many ways the Arnold-Chiari malformation would best explain the physical signs, it would be necessary to have further information with regard to the ventricular system, in order to exclude a lesion at a higher level producing the hydrocephalus and the myelographic appearances as a secondary effect.

*Mr. Calvert* said that he thought the condition was due to an Arnold-Chiari malformation in combination with the Kippel-Feil syndrome, despite the minimum of cerebellar signs, but felt that unless it could be proved that the neurological signs and symptoms had been rapidly progressing, decompression was not indicated at present. Air pictures might be dangerous, and one would have to be prepared to go on to do a decompression.

*Dr. Millar* suggested that the condition might be explained on the basis of a Kippel-Feil malformation and Friedreich's ataxia. This would explain the optic atrophy, pyramidal cerebellar signs, kypho-scoliosis, and the bilateral pes cavus.

*Dr. Fulton* said that Friedreich's ataxia had been considered in the diagnosis, but that though it would explain many of the signs, it took no account of the chronic hydrocephalus, which itself would produce optic atrophy and certain of the other signs.

## 2—ATHETOSIS

DR. R. S. ALLISON gave a brief historical review of the subject and of the differentiation of athetosis from other types of involuntary movements. The anatomical localisation of the lesions and the pathways involved were discussed. Three illustrative cases were then shown.

The first case (pseudo-athetosis) was demonstrated for the purpose of comparison : a youth, aged 17, with astereognosis of the left hand. The involuntary movements in the fingers which occurred when the eyes were closed and the arm outstretched lacked any spastic element and were groping or searching in character.

In the second and third cases of true choreo-athetosis, the involuntary movements affected the face and arm on one side in the second case and the hand alone in the third. Their grotesque and purposeless character was evident, as was also the tonic spasm in the parts involved, whether they were at rest or attempting a voluntary act. In both cases symptoms had been present since birth or early childhood.

*C. P., now aged 21,* showed continuous writhing movements of the right face and hand and had a right-sided hemiparesis. The motor weakness and involuntary

movements had been more pronounced when he was a child. Since he began work at 14-15 the disability had been less. He walked with only a slight limp and was steadily employed as a watchman. His dexterity in the use of the right arm was demonstrated, though finger movements, of course, were impossible. Speech function was unimpaired, though the utterance was affected to some extent by involuntary movements of the right side of the face. He had derived benefit from the continuous use of phenobarbitone. He was not considered suitable for operative treatment. Apart from other circumstances, the right arm was too useful to warrant any possible interference with its chief motor function.

*W. McA.*, now aged 23, was brought forward as a suitable candidate for neurosurgery. The patient was right-handed, and in this case it was only the left hand which was involved. The patient was willing to exchange the involuntary movements for a paralysed limb if necessary. These were almost entirely confined to the hand, and the patient did what he could to conceal the movements by carrying the arm at the side, so that the hand lay behind the left buttock. All his activities, including dressing, were conducted with the right arm. He was employed as a clerk. Psychiatric history revealed no evidence of any abnormal mental traits; on the contrary, he had a well-integrated personality, a stable temperament, and was above the average in intelligence. I.Q. 118 on the Wechler-Bellevue scale.

Dr. Allison suggested that in this case the technique employed by Paul C. Bucy of Chicago, with whom he had discussed the case, should be employed. The representation of the left upper extremity should be extirpated, so as to include the precentral gyrus to the depth of the Rolandic fissure, and forward for about one centimetre anterior to the precentral sulcus. All the grey matter should be excised, but no more white matter than was necessary, and posteriorly it was recommended that the excision would have to be almost two centimetres deep.

A discussion followed, in which Dr. Lothian suggested that perhaps it would be safer and simpler to amputate the offending hand; this view was supported by Mr. Connolly and Dr. Millar. Mr. Connolly thought that the cortical excision may be difficult and dangerous. This view, however, was not taken by Mr. Calvert.

#### SECOND MEETING, HELD 27th JANUARY, 1950

MR. R. J. LUKE gave a short paper on the methods and value of activation in electroencephalography.

In this context, activation is defined as a process which results in demonstrating an abnormality which was previously unrecognised, or less obvious, in the EEG.

A brief survey of the use of hyperpnea, hydration, photic stimulation, cardiazol, and sleep were given. It was concluded that, although the value of hyperpnea is limited, its ease of application makes it worth while. Photic stimulation is also easy to apply and it will probably become more important as its resources are more fully explored. When both these techniques fail, cardiazol may be used. Cardiazol may be combined with photic stimulation to give a more potent activator than either alone. Hydration and sleep are both laborious techniques requiring considerable time, and their efficacy is too low to make them of value for general use.

However, sleep is a useful technique to apply to children. Abnormal EEGs have been recorded in about seventy-five per cent. of epileptics, but the proportion of diagnostic resting records is very much lower. Using these auxiliary techniques, the proportion of diagnostic records can be greatly increased.

Dr. Milliken, Dr. Millar, and Dr. Thompson discussed this paper.

DR. H. H. STEWART discussed the Danish drug tetraethylthiuram disulphide (antabus) in the treatment of chronic alcoholism. This drug by itself causes no ill effects, but in the presence of alcohol a very severe reaction takes place. This reaction is said to be due to the accumulation of acetaldehyde in the blood due to the interaction of alcohol and antabus. Fatalities from the use of this drug have been reported from Denmark and other places, but in all cases the post-mortem usually absolved the drug. After treating six cases of chronic alcoholism with this drug, a severe reaction occurred in the seventh, in which a hemiplegia with aphasia resulted. Gradual recovery took place. The motor aphasia being the last thing to clear up. A super-added functional condition was also described, which cleared up completely.

The conclusion was that antabus was a dangerous drug and that the ill effects were far outweighed by the advantages of its use. It was suggested that until the control of acetaldehyde in the blood was perfected the drug was unsuitable for routine clinical use.

#### THIRD MEETING, HELD ON 17th FEBRUARY, 1950

DR. P. J. O'MALLEY demonstrated an interesting case of right-sided hemianopia. A married woman of 38 who awoke one year ago to find that she was unable to see to the right. She had mild constitutional symptoms for two weeks, including giddiness, lassitude, mild headache, and fever, but no other neurological signs. The hemianopia made a partial recovery. The differential diagnosis was discussed, and it was decided that it probably had a vascular basis involving the occipital lobe. She had a small congenital nævus near the right eye.

DR. R. S. ALLISON presented a case of a woman with a "sacred hand." A Mrs. F. J., aged 45, of good intelligence and good previous work record, who, following the birth of her only child seven years ago, developed unpleasant thoughts concerning Purdysburn Mental Hospital, where she had been a patient on seven occasions. These thoughts she tried to ward off or suppress by repetition of pleasant thoughts, compulsive movements, and other obsessional acts. When aged 14 years, her mother, who was ill, asked her to cool her brow with her hand. This she did after considerable thought as to which hand she would use. The left hand was the one chosen and thus, ever since, she has tried to protect it from the "world" and unpleasant or dirty tasks.

When she is very ill the left side of her body may become totally neglected; also the home and her family. She was mildly depressed, but showed little tension or concern about her illness.

Controversy ranged from obsessional neurosis, with agitation and depression requiring leucotomy, to the possibility of hysteria. The case remains under observation.

DR. D. DAWSON presented the history of a man, A. N., aged 37, who was at present in hospital suffering from a mild reactive depression, the cause of which was due to unsatisfactory sexual relationships with his wife, who, having seven children, did not desire any more. They slept in different rooms, but he visited her in a fugue state and at times was violent. On account of this he had asked to be sterilised, as he thought that his wife had had enough operations. He felt that if the fear of pregnancy were removed, harmony might be re-established in the home.

It was decided that the fugue state was probably hysterical. The Society discussed the legal and psychiatric complications which might issue from male sterilization.

#### FOURTH MEETING, HELD ON 24th MARCH, 1950

MISS E. BUTTFIELD (late speech therapist to Bangour head injury and plastic unit) was introduced by Miss Mitchell. Miss Buttfield read a very interesting paper on "Rehabilitation of the Adult Dysphasic Patient."

The team connected with the rehabilitation of dysphasic patients, which functions at Edinburgh Royal Infirmary and Bangour Hospital, West Lothian, was described. The team consists of neuro-surgeons, neurologist, psychiatrist, psychologist, nursing staff, social worker, physio-therapists, occupational-therapists, physical training instructor, and speech therapist. The work was instigated by Professor Norman Dott and is directed by him.

Principles of re-education—compensation, substitution, and direct retraining were outlined.

It was suggested that a 'sound-film' was the only medium whereby methods of speech re-education could be fully explained.

The importance of the Ministry of Labour officials co-operating in the re-settlement of dysphasics was emphasised.

Three cases were discussed :—

*Case I.*—A man, aged 30 years, with the diagnosis of a vascular accident developed on the basis of chronic ulcerative colitis. Admitted to Bangour two years later with a complete expressive aphasia (no spontaneous recovery in the interim), and a right hemiplegia. He had limited silent reading ability and agraphia. On discharge from hospital a year later he had a vocabulary of some seventy words, but on four occasions only had produced a word that he had not been taught. There was some recovery of function alongside direct retraining in silent reading comprehension and calculation. He learnt to weave whilst in hospital and is now established as a 'home' worker, with a loom provided by the Ministry of Labour.



*Case II.*—A Sgt.-Air Gunner, R.A.F., was involved in a car crash sustaining multiple head wounds. Sixty days later he was transferred to Bangour with a severe predominantly receptive dysphasia, some field defects in the right eye, and the left eye nearly blind owing to direct injury to the optic nerve. His expression was limited to a few colloquial phrases; these were not mutilated nor was there any jargon. He could name no common objects. He was alexic and asymbolic, agraphic, and acalculic. This man was treated for twenty-one months chiefly as an out-patient. His expressive speech function recovered almost completely, but repetition was limited to three to four words. He learned to write rapidly, providing it was a 'known' word; other words had to be experimented with, using mainly kinæsthesia until they seemed correct. Reading speed remained at oral reading level and all words had to be subvocalised. Calculation was taught entirely by concrete form. His resettlement was firstly to a M. of L. Industrial Rehabilitation Centre and from there as an assistant to a market gardener.

*Case III.*—A lorry driver, age 32, with severe mixed dysphasia following thrombosis of the left middle cerebral artery. He was admitted to B.I.U. six months later. Physical signs were very slight weakness and moderate proprioceptive loss in right limbs. He was very obviously impaired in non-language functions. His progress after eight months language re-education was disappointing. He could only speak in three to four word sentences and quite often resorted to writing, which was at a slightly higher level, rather than embarrass himself by producing a wrong word. His chief improvement was in his morale and appearance. From being untidy, dirty, and depressed, he became tidy and cheerful looking. He was resettled in light labouring.

This interesting paper was discussed at some length by Doctors Stewart, Allison, and O'Malley, and Miss Mitchell.

DR. TERENCE FULTON demonstrated an interesting case of Sturge-Kalischer-Weber syndrome:—

The patient (a man of 29) had an extensive facial nævus at birth, was difficult to resuscitate after delivery, and was difficult to feed as an infant. Following a generalised convulsion at the age of twenty-one months, he was unconscious for some considerable time, the head was retracted, and the (R) arm and face were subsequently paretic. He proved to be an uneducable child, reaching the milestones at an abnormally late age. He continued to have generalised convulsions, and following each of these his speech was grossly affected for several days. At the age of 15 it was noticed that his (R) leg was weak and that he limped.

On examination at the present time he has the following physical abnormalities:—

1. Extensive vascular nævus over both sides of the face, especially in the distribution of the first and second divisions of the trigeminal nerve, and involving also the related mucosæ. Some parts of the nævus are raised to form small, wart-like, soft excrescences.
2. Dementia.
3. Small head.

4. Almost congruous incomplete (R) homonymous hemianopia, with macular sparing, and a sense of "wholeness" of the environment.
5. Nystagmus on lateral deviation to the (L) more than the (R) and on central fixation.
6. Impaired convergence.
7. Severe (R) hemiplegia affecting the arm more than the face and legs, with hypotrophy of the (R) half of the body, and contractures.
8. Mild (R) hemi-hypæsthesia and hemi-hypalgesia, with a corresponding degree of loss of sensation transmitted by the posterior columns.

*Radiological Investigation* showed the tortuous branched and parallel lines of calcification over the (L) posterior, parietal, and occipital regions, typical of leptomeningeal venous angioma, while the (L) half of the vault of the skull was much smaller than the (R).

*E.E.G.* was helpful to the extent that the alpha rhythm on the (L) side was almost absent, that on the (R) being normal.

*Diagnosis.*—Sturge-Kalischer-Weber Syndrome.

#### COMMENTS

The signs and symptoms indicate widespread disturbance of function of the (L) cerebral cortex. The main interest of the case lies in the disturbance of visual function, incomplete hemianopia, being associated with greatly diminished alpha rhythm in the E.E.G. It is noteworthy, also, that the leptomeningeal calcification revealed by X-ray examination of the skull is mostly situated over the area of the occipital pole. There is no doubt, however, that the nævus extends much more widely than the area of calcification would suggest. The small, wart-like angiomatous excrescences in the face resemble somewhat adenoma sebaceum in appearance, and, in fact, the case was reported of the association of this condition with leptomeningeal nævus. A biopsy has still to be taken.

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Surgical technique is not dealt with in detail and many of the procedures are regarded as 'office' work but the scope of this is obviously greater in the U.S.A. than in this country.

Antiseptics and antibiotics are brought up to date, but this latter term cannot be expected to apply for very long.

The use of Proctocain and similar agents is not looked on with favour.

The various lesions are dealt with in separate sections, well illustrated by X-rays, photomicrographs and coloured photographs.

There are special sections on geriatrics and pædiatrics.

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C. J. A. W.

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# The Painful Shoulder

By R. J. W. WITHERS, M.D., M.CH.(BELF), F.R.C.S.(EDIN.).

from the Orthopædic and Fracture Clinic, Royal Victoria Hospital, Belfast

PAIN in the shoulder may result from lesions of a gross nature, such as fractures, dislocations, tuberculosis, bone tumours, and infective arthritis, and may also be due to conditions of the cervical spine, the neck, the apex of the lung and pleura, and obstruction of the cervico-axillary outlet. As well as this, pain may be felt in the shoulder from visceral disturbance, notably from heart and gall-bladder disease. When these abnormalities are excluded there still remains a well-defined group of conditions of the shoulder in which there is limitation or alteration in the range of movement, pain on attempted movement, sometimes muscular wasting, and usually no radiographic evidence of abnormality.

Certain morphological and anatomical considerations must be recalled to appreciate the problems of pathology, symptomatology, and treatment.

## COMPARATIVE ANATOMY

In those mammals in which the fore-limb has been freed, the scapula shows gradual changes from the long narrow bone of the pronogrades to a progressively broader form with ever increasing size of the infra-spinous fossa in the orthogrades. Progressive size and extension of the acromion has occurred, indicating the increasing dominant position occupied by the deltoid in the function of the shoulder joint. In order to afford a strong levering effect, the deltoid insertion has undergone progressive distal migration on the humeral shaft. With regard to the muscles, the supraspinatus, whilst remaining relatively static in its morphology, has progressively decreased in relative importance to the total shoulder musculature. Indeed, it is not too much to say that in Man the supraspinatus muscle is well on the downward path of degeneration. The deltoid has increased in relative importance; for example, in the opossum it forms twenty per cent., whilst in Man it claims forty-one per cent. of the total shoulder musculature. The infra-spinatus is absent in the opossum, but in Man it forms  $7\frac{1}{2}$  per cent. of the entire muscle mass. The subdeltoid bursa makes its first appearance in the chimpanzee and it is clear that its function has to do with the freedom of rotation of the upper end of the humerus in relationship to the under-surface of the acromion. Without such rotation (external rotation) it would be impossible for Man to elevate his arm above 90 degrees, as impingement of the great tubercle of the humerus against the acromion process would, and indeed does, occur (Inman, Saunders, and Abbott, 1944).

## MOVEMENTS

The movement of full elevation of the arm includes motion at no less than four different joints—the sterno-clavicular joint is in motion in the early phase

of elevation; the acromio-clavicular joint towards the end of full elevation; whilst gleno-humeral and scapulo-thoracic movements are occurring throughout all phases. The participation of each of these four components in the entire movement of elevation is simultaneous and not successive. It was formerly taught that the movement of elevation was a simple matter—the deltoid contracted until the arm reached 90 degrees from the side, and then the serratus magnus, tilting the scapula forwards, effected the last 90 degrees of elevation. Later still, the supraspinatus was thought to effect the first 15-20 degrees of abduction, then the deltoid to 90 degrees, followed by the serratus magnus to 180 degrees. That this teaching was entirely false cannot be doubted in the present state of our knowledge. It is now clear that the movement of elevation of the arm occurs by the contraction of all the shoulder muscles at once, but, of course, each muscle showing a peak of contraction at certain phases of movement. Myographic studies, where, by placing electrodes directly into the substance of the various muscles, electrical action potentials can be picked out and magnified, show that this statement is true. For example, the supraspinatus contracts through all phases of elevation, but its peak of contraction is somewhere between 80-120 degrees. This muscle acts progressively with the deltoid throughout the entire range of movement. These myographic studies, well recorded in the work of Inman, Saunders, and Abbott, suggests that, with regard to gleno-humeral movement, the muscles concerned can be divided into two groups:—a deep set, consisting of the spinati and subscapularis, along with the teres major and minor, and a superficial set, essentially the deltoid. These two groups of muscles are separated by the subdeltoid bursa, and each has an important part to play. In the past, abduction of the shoulder has been considered in terms of the power of the deltoid. Now it seems clear that the depressor, fixator, and tensor actions of the deep muscles assume equal importance—without these, the deltoid is useless to move the arm away from the side. At the start of elevation the entire shoulder muscle mass is in action and the scapula seeks, in relationship to the humerus and to the chest wall, a precise position of stability.

This early phase is highly irregular and characteristic for each individual. During this phase the scapula is moving on the chest wall and the head of the humerus is being drawn into the glenoid and fixed there, so as to act as a hinge for further movement of elevation. The fixation is effected by the combined pull of the supraspinatus upwards, the infra-spinatus, tending to pull the head of the humerus backwards, downwards, and into the glenoid, and the subscapularis forwards, downwards, and into the glenoid.

Once this setting phase has been stabilised (and this occurs at about 30 degrees abduction or 60 degrees forward flexion) the relationship of scapular to humeral movement thereafter remains constant—for every 15 degrees of movement, 10 are scapulo-humeral and 5 are scapulo-thoracic. C. E. Beevor (1904), in his Croonian Lectures, laid down this axiom with regard to the brain:—that it knew nothing of the action of individual muscles, but only of movement. We can then see a central pattern of movement carried to the periphery; in other words,

elevation of the arm at the shoulder is a peripheral pattern controlled centrally, in which the brain does not dictate the action of any one muscle by itself, but does dictate the action of all the muscles acting together, albeit, each with differing power at a precise and critical phase of movement. From this it follows that the only way, in shoulder lesions, to gain full movement is to practise movement of the shoulder. It is obviously useless to attempt static contraction of any one muscle or even to encourage such static contraction by the use of the faradic current. Only by total movement can recovery of function, in lesions of the shoulder joint, be effected.

#### SHOULDER CAPSULE

In the adult the capsule of the shoulder joint is fused with the overlying deep muscles, whereas in infants it is separated from the deep muscles by areolar tissue, the tendons being short and the muscle fibres extending to within a few millimetres of their humeral attachment. With increasing age the tendons become longer, and in old age the supraspinatus tendon may extend as far proximally as the suprascapular notch. The degree of adhesion of the tendons to the capsule increases with age, and in older subjects the fusion is complete. These changes are more marked in the upper part of the capsule, so that in adult life the entire supraspinatus tendon and the upper parts of the infra-spinatus and subscapularis tendons are intimately fused with the capsule. This fused portion will be referred to hereafter as the musculo-tendinous cuff. The frequency with which rupture of this cuff is found at post-mortem examination was first emphasized by Codman (1931). More recently, Grant and Smith (1948) have demonstrated that degenerative changes in the cuff occur in arithmetical progression as age advances. In a large series of dissecting-room specimens, they found degenerative changes in no case up to 40 years; in 25 per cent. of subjects from 40-55 years; and after 75 years fully 50 per cent. showed lesions. It was presumed that many of these lesions were associated either with no symptoms at all or with minor symptoms during life. These degenerative changes ranged from minor superficial fibrillation of the musculo-tendinous cuff to complete rupture of it. On account of the fusion of the deep muscles with the capsule; the natural tendency, as age advances, to degenerative changes; and the special liability of direct injury to the musculo-tendinous cuff—it is contended that most cases of painful shoulder are due to pathological changes in the upper part of this peculiarly fused capsule.

#### CLINICAL GROUPS

Three most important lesions of the musculo-tendinous cuff will be considered; first, ruptures of the supraspinatus; secondly, supraspinatus tendinitis; and thirdly, inflammatory lesions of the cuff, that is to say, tendino-capsulitis, or, simply, capsulitis. Dissatisfaction must be expressed with the nomenclature of the painful shoulder. Such terms as peri-arthritis, bursitis, arthritis, rheumatism, neuritis, and pseudo-neuritis are all begging the question of pathology. If the pathology is in the musculo-tendinous cuff of the capsule, then surely the term "capsulitis" or "tendinitis" is sufficient for the majority of cases. The term "bursitis" is

condemned. The bursa is the "peritoneum" of the deep muscles of the shoulder and, like the peritoneum, it seldom is the site of primary pathology, although it often shares the pathology of the organs which it protects, namely, the capsule and deep tendons. There is no doubt that bursitis does occur when there is capsulitis or a traumatic lesion of the capsule, but, as a condition by itself, it must be excessively rare. Such terms as "neuritis," "pseudo-neuritis," and "rheumatism" mean nothing—they have been introduced by those impressed by the painful nature of the condition, but without thought of the pathology causing the pain.

#### 1. RUPTURES OF THE SUPRA-SPINATUS (*The Useless Arm Syndrome*).

The rupture may be complete or incomplete, involving neither the entire width nor depth of the tendon.

The term "supraspinatus" is used here for convenience, but it must be remembered that many of these tears do not confine themselves to the supraspinatus tendon, and extend anteriorly into the upper part of the subscapularis tendon and sometimes posteriorly into the infra-spinatus tendon, i.e., "cuff" ruptures are common findings. The typical case occurs in a man, usually over the age of 40, who, after a fall on the shoulder, or after attempting to lift a heavy weight, experiences sudden diffuse pain over the shoulder with inability to elevate the limb and inability to support the limb in the elevated position when so placed by the examiner. This syndrome is combined, however, with a normal range of passive movement (see fig. I, a-b). Sometimes there is tenderness over the great tuberosity of the humerus. In this type of case the disability is due to loss of the power of fixation of the humeral head in the glenoid by the deep muscles and the deltoid is therefore put at such a disadvantage that, although it is able to contract, as can be felt by the examining hand, it is unable to sustain true shoulder movement. When the arm is placed by the examiner above a right angle, in complete ruptures the inability to sustain the shoulder in this position causes the arm to fall uselessly to the side. This sign has been called by the Americans the "down it will go" sign. In cases of recent rupture the loss of fixator function may be no more than a reflex phenomenon produced by pain, because the infra-spinatus and subscapularis, often uninjured and much more important than the supraspinatus, are also thrown out of action. If this is accepted, it follows that clinically it may be impossible to distinguish between complete and incomplete ruptures. Two methods are of help in distinguishing between these two lesions:—

(a) After the injection of a few c.c. of 2 per cent. novocaine into the subdeltoid bursa, restoration of shoulder movement in minor and incomplete ruptures may be effected in a few minutes, but in complete ruptures no change will occur.

(b) Radiography of the shoulder after injecting 6-8 c.c. of Perabrodil 35 per cent. into the subdeltoid bursa can demonstrate whether a communication exists between the bursa and the cavity of the true shoulder joint (Axen, 1941). Normally these two cavities are completely separated by the upper part of the musculo-tendinous cuff, but in complete ruptures of this cuff the floor of the bursa and the roof of the joint cavity are opened up with a communication between the two (see fig. II, a-b—Bursograms). It is worthy of note that, in every positive case which

I have seen, perabrodil injected into the bursa causes severe pain to the patient, whereas, when no rupture is present and the perabrodil remains entirely in the bursa, the condition of the shoulder is painless.

## 2. SUPRA-SPINATUS TENDINITIS (*The Painful "Arc" Syndrome*).

Following a fall, or indeed sometimes spontaneously, a painful arc of movement during mid-elevation of the shoulder develops with weakness of the limb, which may be mild or may be so extreme that the patient is unable to elevate the arm against the resistance of two fingers. There is no actual limitation in the range of movement, and full shoulder movement is present. The symptoms are often mild to begin with, so that it may be many months before the patient seeks advice. In the past it has been thought that the disability is due to impingement of an irritated or inflamed supraspinatus tendon against the under-surface of the acromion, but in some cases where excision of the acromion has been carried out for rupture, the typical pain of supraspinatus tendinitis has persisted. So much bone had been removed that acromial pressure could not have been the cause. It may well be that there is another explanation and that not all cases of supraspinatus tendinitis produce pain by mechanical impingement against the acromion. In the myographic studies of the shoulder joint previously referred to, it has been noted that the peak of contraction of the supraspinatus is between 80-120 degrees of elevation. This muscle is contracting most powerfully in that particular phase of elevation, which, in the condition under consideration, is associated with pain. It is possible that active contraction of a supraspinatus tendon, irritable from inflammation or rupture and accentuated by the pull of the infra-spinatus and subscapularis at the focal point of their attachments, may be in itself sufficient to cause the typical pain (see fig. III).

I do not now believe that this group is a true tendinitis, since, following a series of explorations for this syndrome, I have found about seventy-five per cent. to be due to ruptures of the musculo-tendinous cuff. It would appear, therefore, that the so-called supraspinatus tendinitis syndrome is no more than an expression of spontaneous degeneration or of rupture of the musculo-tendinous cuff. The pull of the subscapularis and infra-spinatus, whilst irritating the degenerated or ruptured cuff at the focal point and causing pain, is sufficient in these cases to fix the humeral head and allow full gleno-humeral movement to occur, even though weakly, against resistance. In many cases the ruptures, often incomplete, may set up those processes of inflammation and repair which are curative and which have led to the operative findings of "tendinitis." Indeed, the fact that spontaneous recovery occurs in a big percentage of cases is sufficient evidence for this belief.

## 3. CAPSULITIS (*The Frozen Shoulder Syndrome*).

The Americans, with their peculiar aptitude for expressive nomenclature, have referred to this group as "the frozen shoulder." The literature abounds in works on "the frozen shoulder," the first paper on this subject being published in Paris in 1872 by Duplay. Indeed, the condition has sometimes been referred to as Duplay's Disease. The average age of patients is over 50 and males and females



are about equally affected. In sixty per cent. there is a history of trauma, but in the rest the onset is spontaneous. In this group there is pain, limitation of scapulo-humeral movement, weakness, and associated muscular wasting. At the onset the pain may be almost continuous, felt diffusely over the entire shoulder, and even with radiation down the arm to the fingers. It is worse in bed at night. Later the pain settles down and is only present when attempts at movement are made when it is typically found at or about the deltoid insertion. At this stage the patient finds that a certain return of movement has occurred. It is believed that the essential pathology in this group is inflammation of the capsule following injury, or from irritative causes within the musculo-tendinous cuff itself, such as calcium deposition or the degenerative process referred to above.

Neviaser (1945) explored ten human shoulders in order to demonstrate the underlying pathology, and he found that the capsule was thickened and adherent to the humeral head; when the joint was manipulated, the capsule separated from the head of the humerus in much the same way as an adhesive bandage can be torn from the skin. Sections of the musculo-tendinous cuff were taken and the pathology showed inflammatory and reparative changes. He adds, in his article, that he only did this to find out what the pathology was, and he stresses that he does not recommend this procedure as a method of treatment. If this view is accepted, it is clear that two stages in the pathological process of capsulitis can be recognised:—(a) An early stage of acute inflammation with inflammatory exudation into and around the capsule and limitation of shoulder movement due to muscle spasm which, therefore, relaxes under anæsthesia; (b) A later adhesive stage, when resolution of the inflammatory process has taken place, in which a replacement fibrosis occurs, with the formation of adhesions and limited movement even when the muscles are relaxed by anæsthesia.

This concept of the pathology is borne out clinically, and it is suggested to examine every "frozen shoulder" under anæsthesia in order to test the range of movement and to find out whether the limitation is protective or adhesive. It must be stressed that up to the moment there is nothing one can do, during the acute phase, to help the patient recover movement rapidly. It is clear that, in such a tendinous and avascular structure as the shoulder cuff, the repair process will be slow and, therefore, the symptoms of which the patient complains will last for a long time. This is clinically true, as cases of "frozen shoulder" very often have symptoms up to a year or even longer.

Degeneration or rupture, especially central and incomplete rupture, of the tendinous cuff may be followed by calcium deposition. Such deposits, requiring X-ray examination for their discovery, produce symptoms which are no different from those already described under the "Painful Arc Syndrome" or the "Frozen Shoulder." They were found in only three per cent. of cases in the Royal Victoria Hospital. Their very presence is evidence of degeneration, but sometimes they may set up, by irritation, a repair process sufficient to effect cure, and, at others, may rupture into the subdeltoid bursa with relief to the patient.

Biceps tendon degenerations occur usually as part of the general degenerative changes in the shoulder cuff and will not be considered further.

*Frequency of the three groups.*—Out of one hundred personal cases :—

12—“Useless Arm Syndrome”

27—“Painful Arc Syndrome”

61—“Frozen Shoulder Syndrome”

#### TREATMENT

##### RUPTURES OF THE SUPRASPINATUS TENDON (*Useless Arm Syndrome*).

Conservative measures are adopted in the first instance and the arm is put at complete rest with the shoulder abducted to 60 degrees, with about 60 degrees forward flexion, and external rotation about 30 degrees. This can be effected in an abduction splint, but, as these splints are of standard size, it is difficult to apply one closely enough to the arm and chest for the average patient's comfort. It is better to use, in the majority of cases, a thoraco-brachial plaster of paris cast. After a period of four weeks the lid of the plaster is removed and, if the patient can elevate the arm from the splint and hold it elevated, then it is considered that healing of the lesion is occurring, or, indeed, has occurred. After removal of the splint, shoulder exercises are practised and encouraged by heat. Where no recovery of elevation has occurred in four weeks, operation is advised. The operation is really an exploration of the upper part of the shoulder and therefore adequate exposure is necessary. This is carried out through a shoulder-strap incision with the patient lying on his good side, and, as a first step, the acromion process is excised. Usually the subdeltoid bursa is opened into at this stage of the operation, but, if not, it is deliberately incised, and the upper part of the musculo-tendinous cuff exposed and explored. The rupture is usually found within a short distance of the attachment of the cuff to the great tubercle of the humerus and only if a rupture is present can the articular surface of the humeral head be seen. In some cases, almost complete avulsion of the whole musculo-tendinous cuff may be found, and in others the rupture is of small degree and usually assumes the appearance of a triangular-shaped or L-shaped rent which may extend for some distance into the substance of the supraspinatus, or may have a longitudinal extension either into the infra-spinatus behind or subscapularis in front. In other cases the rupture is on the humeral side of the cuff only and can only be demonstrated by incising the bursal floor—it usually presents as a “wrinkle” in the bursal floor.

When I started operating on ruptures of the supraspinatus, I always attempted to bring down to the great tubercle, and suture it there, the medial end of the rupture. I was very unimpressed by the end results of such treatment and, indeed, I often felt that the operation had not been worth while, as it either failed to cure the patient, or in some cases actually made the condition worse. After realising, however, that the pull of the subscapularis or the infra-spinatus may cause longitudinal extension of the tear to extend proximally, I contented myself with freshening the edges of the rent and side-to-side suture. The results from this method of repair have been much more encouraging. It should not be forgotten,

even though an apparently effective repair has been carried out, that the prognosis in complete traumatic rupture of the supraspinatus still remains far from good. The operative repair often sets up a "frozen shoulder" and it may be twelve months before one knows what the end result is to be. This is practically always true in massive ruptures or avulsions, where the outlook is particularly poor. Indeed, in a fairly recent series of eighteen operations, only in three was a perfect functioning shoulder obtained. The rest were unsatisfactory—in five, movement was good, but there was pain and weakness of the shoulder thereafter, whilst in ten the condition was unaltered.

#### SUPRASPINATUS TENDINITIS (*Painful "Arc" Syndrome*).

Conservative measures are suggested in the majority of such cases, as there is a tendency, after a period of nine to twelve months, for many of the cases to undergo spontaneous recovery. Operative treatment is therefore only advised for the worst cases and for those in whom conservative measures have failed after a reasonable period of time.

Repeated injections of local anæsthetic into the bursa are sometimes helpful, but never curative. It may well be that this treatment allows the patient to manipulate his own shoulder painlessly, and if a few adhesions are present between the musculo-tendinous cuff and either the acromion or the humeral head, tearing of such adhesions may effect cure. In this group I am not satisfied that physiotherapy, in any of its forms, has any part to play. In those cases in which operation is called for, the technique adopted is exactly the same in the first instance as has been described for ruptures of the supraspinatus tendon, viz., excision of the acromion.

In a recent series, in which operations were done, it was found that a minority were due to a localised capsulitis related to the area of the supraspinatus tendon, but the majority of the lesions were actually ruptures, incomplete or complete, many of which were clearly spontaneous ruptures in a thin, attenuated, and obviously degenerated musculo-tendinous cuff. A note of warning should be added: it is sufficient in the group under consideration to be satisfied with acromiectomy and exploration and nothing more where an already degenerated supraspinatus tendon is found with or without a rupture. Indeed, I have made the condition very much worse in several cases by neglecting this rule. Whatever may be the explanation, when the acromion is excised, the end result from such cases of resistant type is reasonably good—the pain disappears rapidly, though, of course, weakness of the shoulder is usually a permanent feature.

#### CAPSULITIS (*"Frozen Shoulder" Syndrome*).

It is important to divide these cases into their sub-groups of "irritative" or "adhesive" lesions, and, as previously pointed out, this is best done by examination under anæsthesia.

(a) In *irritative capsulitis*, under anæsthesia, movement returns passively to normal, whilst in adhesive capsulitis movement is still restricted. In irritative capsulitis treatment is primarily by rest. Some years ago I used the abduction

splint, but no evidence has been found that a splint has any advantage over a sling. Indeed, many patients treated in a splint were forced to discard it as it increased their pain. During the acute phase rest is all-important. The patient can be encouraged by the application of heat, but in a not inconsiderable percentage heat seems to irritate and aggravate the symptoms. If complete rest is given to the shoulder, however, the pain gradually settles down, and, as soon as the condition becomes bearable, gentle active exercises for the shoulder are then practised with the patient at first supine. On the average, return of function can be expected in about three and a half months. If more vigorous measures are adopted, the inflammatory process does not get a chance to settle down and symptoms may persist for many months longer than necessary. The best that can be done in this early phase is not really to "treat" at all, but to encourage with wise words, with analgesics, and with a sling. To treat by physiotherapeutic measures and by manipulation may only increase the symptoms. This suggestion does not lessen the difficulties where "treatment" is expected or demanded by the patient.

During this painful stage, injection of the supra-scapular nerve at the upper border of the scapula has been found, in a proportion of cases, to be of some help. It is not claimed that it could possibly effect any change in the underlying pathology, but it may lessen the pain by blocking the sensory impulses from the shoulder. After the acute irritative phase has settled down there will be, in a certain number of cases (and especially where the inflammation has been severe), replacement fibrosis in the capsule, so that the condition passes gradually into the second sub-division of "adhesive capsulitis."

(b) In *adhesive capsulitis* patients usually have had their symptoms for many months; indeed, if a painful and stiff shoulder has been present for more than four months the chances are that it is already in the adhesive stage. Under anaesthesia no change in shoulder movement occurs and this is the type of case which responds well to manipulation of the shoulder. In this group pain is much less severe and only occurs after attempts at movement. Since lateral rotation of the humerus is necessary for full abduction, the manipulation to be carried out first of all is lateral rotation of the humerus; indeed, in many cases manipulation in this direction only is sufficient to free the shoulder. During manipulation the adhesions can be felt and sometimes heard breaking down. Following manipulation, the arm is supported in a sling and active exercises practised from the beginning. Extreme gentleness in manipulation must be stressed. The "snaps" of a vigorous manipulation may be a ruptured tendon or a fractured neck of the humerus.

A small percentage of cases of capsulitis never recover, and it is presumed that in them the degenerative changes in the musculo-tendinous cuff are of such an extreme degree that reversibility of the pathological process cannot occur. This percentage is small, however, as, in a recent series of my own, it only occurred in three cases out of sixty-one, i.e., about five per cent. In the same series "crepitus" on movements of the shoulder was an almost constant finding, even years after full recovery had occurred. Often the patients were unaware of it and its significance is hard to assess.

## SUMMARY

1. It is suggested that the great majority of cases of painful shoulder are due to pathological or traumatic lesions of the fused musculo-tendinous cuff of the upper part of the shoulder capsule. Other causes do occur, but they are uncommon.
2. Conservative treatment of all lesions is stressed to begin with.
3. Surgery should only be advised in complete ruptures of the supraspinatus and in those other conditions of the shoulder which have resisted conservative measures. The essential part of the operation is removal of the acromion and exploration of the musculo-tendinous cuff. Suture of ruptures, cutting of adhesions, etc., etc., depend on the exploratory findings.
4. It should be remembered that ruptures of the cuff may cause no symptoms, may lead to complete loss of shoulder function, or may cause a painful arc of movement during mid-elevation, but the clinical state depends on whether or not the condition of the cuff is causing pain, and whether or not the other deep muscles of the shoulder can compensate in their tensor and fixator actions for the loss of action of the supraspinatus.
5. Even in cases of capsulitis it is thought that the condition is pre-disposed to by the same degenerative changes in the musculo-tendinous cuff which pre-dispose not only to rupture, but also to so-called supraspinatus tendinitis. If this is true the *rupture* syndrome, the *tendinitis* syndrome, and the *capsulitis* syndrome are all different expressions and different degrees of the same underlying degenerative changes.
6. The management of the "frozen shoulder," whether loss of movement is protective or adhesive, calls for time and patience, but the ultimate outlook is good. If the pathological concepts outlined above are accepted, it is doubtful whether physiotherapy (apart from active exercises and manipulation) has any part to play at all.
7. From a clinical point of view, it is helpful to think of the painful shoulder in terms of the useless arm, the painful arc, and the frozen shoulder syndromes. These are expressions no doubt objectionable to the pathologist or the anatomist, but are of help to the practitioner in sorting his cases into some useful form, so that helpful advice can be given the sufferers of shoulder pain in regard to treatment and prognosis.

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## REVIEW

A COURSE IN PRACTICAL THERAPEUTICS. By Rehfuss, Albrecht, and Price. Baillière, Tindall & Cox. 82s. 6d.

This monumental work comes mainly from the Jefferson Medical College, Philadelphia. It is a book of over eight hundred pages and will find its place in medical literature as a work of reference rather than a text-book for students.

The arrangement is very modern, every step in the treatment of any disease is clearly set out in tabulated form and there is nothing vague or indefinite in this book. Some seventy plates give at a glance the clinical picture, ætiology, symptoms, differential diagnosis, nursing care, and treatment.

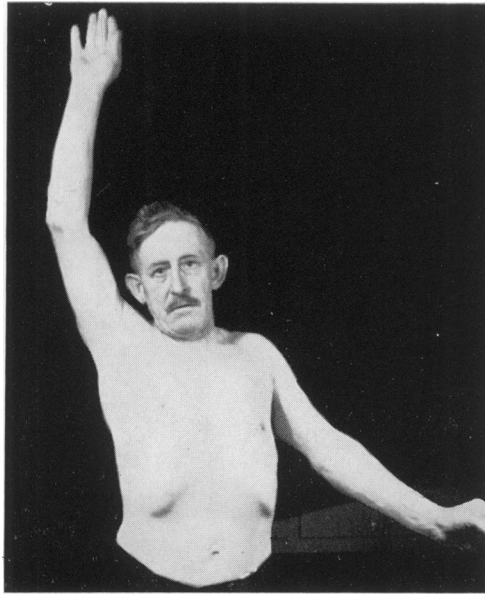
Accessory therapeutics measures all described in complete detail and it is helpful to find measures such as Buerger's Exercises are adequately illustrated and the time spent in each position is clearly indicated.

Proprietary preparations are an important feature of present-day medicines. While many of these remedies are of great merit, it is not always easy for the physician to discriminate between the rival claims of enthusiastic manufacturers. Professor Rehfuss and his colleagues mention all the important British and American proprietaries, and their assessment of the values of their preparations has every appearance of fairness and wisdom.

The book is probably the most important work on practical therapeutics of this century and is warmly recommended. The English edition, published by Messrs. Baillière, Tindall & Cox at 82s. 6d., is a credit to this famous firm.

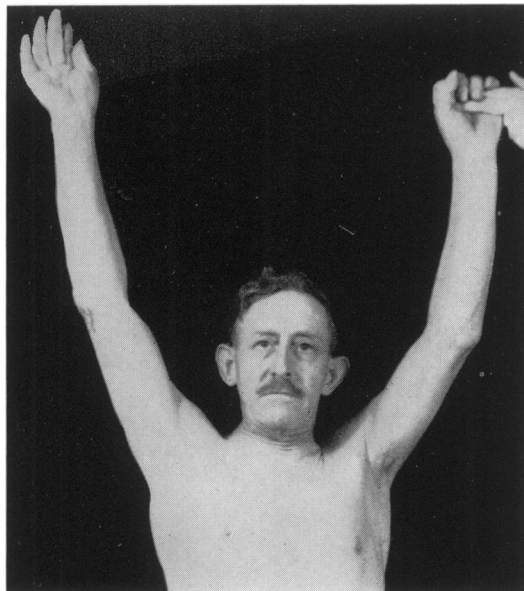
T. A. K.

THE PAINFUL SHOULDER



**Fig. 1 (a)**

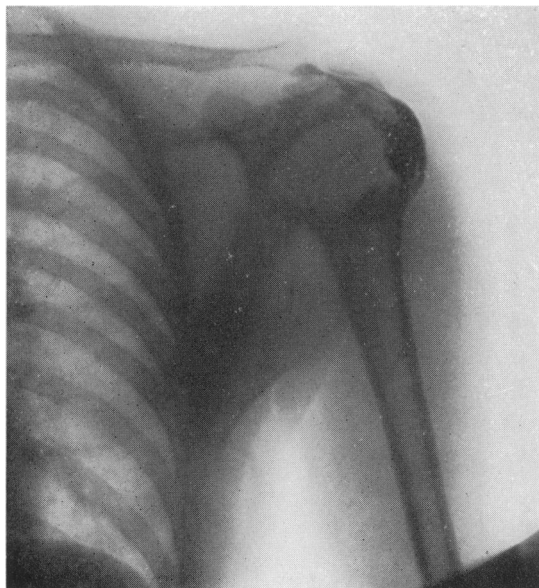
Rupture of the supra-spinatus tendon. Shewing the patient's inability to elevate the arm whilst the opposite arm has full movement.



**Fig. 1 (b)**

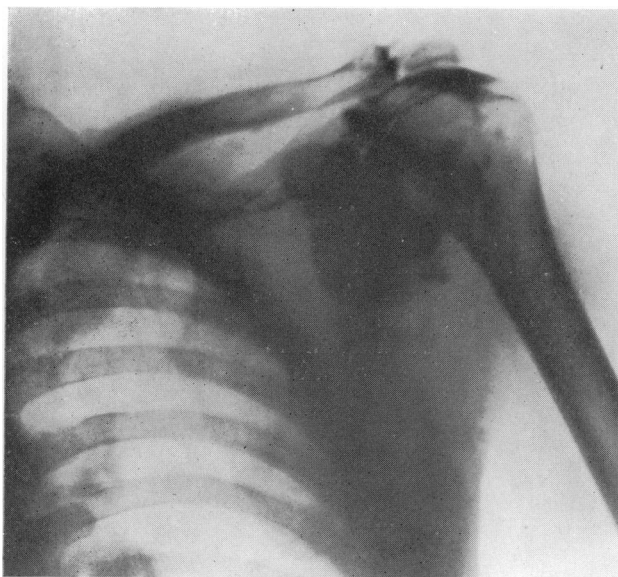
Rupture of the supra-spinatus tendon. Shewing that a full **passive** range of elevation of the arm is possible.

## THE PAINFUL SHOULDER



**Fig. II (a)**

Normal bursogram. Perabrodil has outlined the sub-deltoid bursa, only the joint cavity remaining clear.



**Fig. II (b)**

Bursogram in a case of ruptured supra-spinatus. Note that most of the perabrodil has escaped from the sub-deltoid bursa and has filled up the shoulder joint cavity.



THE PAINFUL SHOULDER

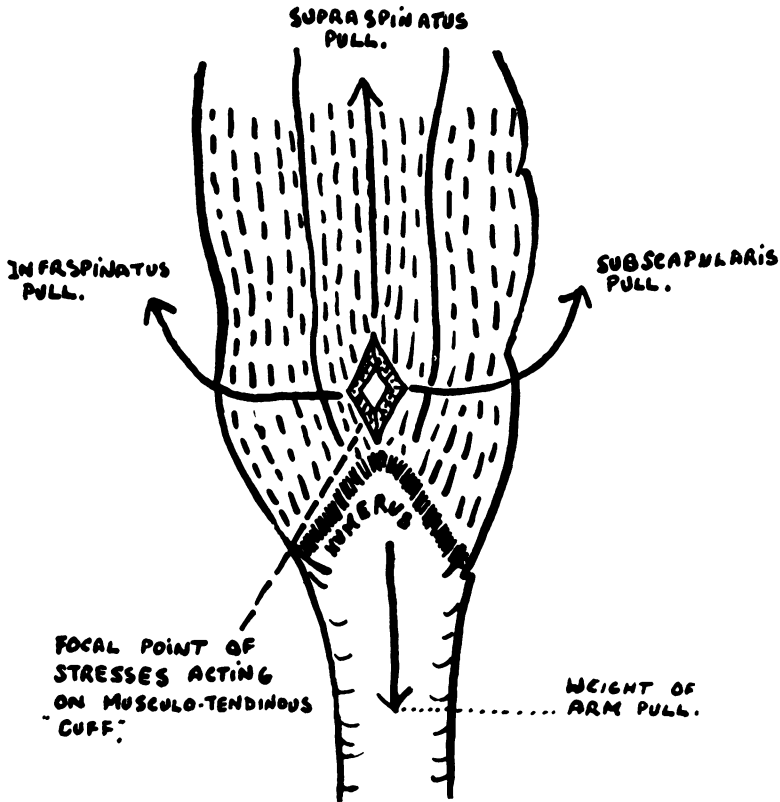


Fig. III

Diagrammatic representation of the deep shoulder muscles seen from above. The focal point of stress of the musculo-tendinous "cuff" is indicated and the transverse arrows shew how the pull of the subscapularis and the infra-spinatus may irritate a "cuff" lesion or cause extension of a "cuff" rupture. (Modified from Jones, L.: Arch. Surg., 49, 390; 1944.)

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T. A. K.

## REVIEW

THE ORIGIN OF MEDICAL TERMS. By Henry Alan Skinner, M.B., F.R.C.S.C.

Pp. viii + 379. London : Baillière, Tindall & Cox. 1949. 54s. net.

STEDMAN'S MEDICAL DICTIONARY. Edited by Norman Burke Taylor, M.D., F.R.S.C., F.R.C.S.(Edin.), etc. Pp. xlv + 1361. Fully illustrated.

Seventeenth Edition. London : Baillière, Tindall & Cox. 1950. 65s.

THESE books are closely related in many ways and it is not only suitable, but also appropriate, to review them together.

The first named book was written by the Professor of Anatomy in the University of Western Ontario, and it is intended for the use of medical students in their pre-clinical years.

Professor Skinner regrets the poor general education of most students entering medicine and hopes to make up for this in some degree by putting into their hands a book giving the meaning, significance, and history of medical terms used in the basic sciences of anatomy, physiology, biochemistry, and pathology. This is a very wide field and the author is deserving of congratulations and gratitude for the way in which he has fulfilled his task.

Unlike the editor of Stedman, he has used, in general, English spelling rather than American, but on occasions he uses American spelling if he prefers it.

Irish readers of the book will take pleasure in finding in it famous Irish medical names, such as Adams, Cheyne, Corrigan, Colles, Graves, Houston, and Montgomery, but this pleasure is a little diminished by the reflection that the most recent Irishman mentioned died seventy years ago.

For a reason that is not clear, modern bacteriological nomenclature is not used, although the modern name of some organisms is given as an alternative, for example, *bacillus abortus* is also given as *brucella abortus*.

Stedman's medical dictionary is an old friend and, if proof of its popularity be sought, it will be evidenced by the appearance of this, the seventeenth edition.

Scientific writers are often accused of using jargon, and to this accusation a plea of guilty might well be accompanied by the current retort, "So what?" When a new science is being elaborated, or an old one developed, it often occurs that there is no existing word in the language to describe the phenomena under consideration, and the writer is obliged to borrow from another language whose vocabulary is more comprehensive, or else string together native words and give the resulting compound a new and technical meaning. This is the origin of jargon. The Greeks, being early in the field of physical and metaphysical speculation and research, had an extensive vocabulary which was later used by the Romans. It was inevitable that when science began to be studied and the results of scientific observation written down in the various languages of western Europe, the already extensive Græco-Roman scientific vocabulary should be taken over almost unchanged into English, French, German, Italian, Spanish, and the rest. In Germany in the 1930s the Nazi demand for a purely Teutonic culture led to a movement away from the Græco-Roman stream of words, so that, to give one example, "mucosa" became "schleimhaut" (slime skin).

The vocabulary of medicine is a formidable one and all who use it, whether in teaching, or writing, or testifying in courts of law, or in the consulting-room, or at the bedside, have need of a reference book such as Stedman, which, besides being a dictionary in the ordinary sense, is also an encyclopædia of medical history and biography, and there is something of interest and value on every page.

It should be noted that the present editor is also connected with the University of Western Ontario, but, unlike his colleague, Professor Skinner, he has in most instances given the American spelling first and the English spelling second, so that we have "hemorrhage," "color," etc.

The bibliographer will regret that there is no mention of the date of the first edition and that space was not found to reprint the preface to it.

H. G. C.

## MONTHLY OBSTETRIC MEETINGS

A MEETING was held on the 25th September, 1949, and Mr. H. L. Hardy Greer took the chair. The statistics for the previous quarter in respect of the Jubilee Maternity Hospital and the Royal Maternity Hospital were presented and a general discussion followed.

At a meeting held on the 26th October, 1949, Mr. H. L. Hardy Greer took the chair and Dr. B. M. Corkill gave a review of the cases of multiple pregnancy delivered in the Royal Maternity Hospital during the last ten years. There were 376 cases in this review, an incidence of 1 in 42 births. Racial characteristics, heredity, increasing age, and increasing parity are said to influence the incidence of multiple pregnancy, but no conclusions could be drawn on these points in this series. There was a high incidence of premature labour (80 per cent.), pre-eclamptic toxæmia (45 per cent.), anæmia (9 per cent.), and hydramnios (6 per cent.). Two cases of locked twins occurred in this series, in spite of its extreme rarity. Three mothers died, an incidence of 0.8 per cent., and the total foetal loss was 15.7 per cent. The speaker, in conclusion, gave means by which the foetal mortality could be further reduced.

A meeting was held on the 23rd November, and Professor Macafee took the chair. Mr. W. Campbell described a case of Roberts' pelvis, which had been caused by an injury in childhood. There was complete absence of the right sacral ala leading to gross contraction of the pelvis and necessitating elective Cæsarean section. Dr. John Watson discussed the maternal deaths occurring in the Royal Maternity Hospital during the years 1927-36 and 1939-48. There were 141 cases during the first decade and 119 cases during the second decade. Puerperal sepsis represented the greatest single cause in the first decade, but had dropped to fourth place in the second decade. Its place had been taken by shock and postpartum hæmorrhage, but this, in turn, appeared likely to be superseded by heart disease as a cause of maternal deaths. The speaker tried to find the primary avoidable factor in each case during the second decade and, where possible, apportioned the blame to the obstetrician, the patient's doctor, or the patient herself. He considered that the incidence of maternal deaths could still be greatly reduced by improved obstetrical skill and education, teamwork, utilisation of the blood transfusion service, and a fuller employment of the advances in anæsthetic technique.

At a meeting held on the 4th January, 1950, Mr. J. A. Price took the chair and Professor Macafee opened a discussion on hydramnios. He took as his standard any case where a suspicion of hydramnios had led to an X-ray examination. He reviewed 10,902 hospital cases and 1,119 private cases with an incidence of hydramnios of 1.2 per cent. and 1.4 per cent. respectively. In cases of hydramnios the gross foetal mortality was 58.1 per cent. in the hospital series and 52.9 per cent. in the private series. There were fifty-four cases of hydramnios (36 per cent. of series) in which

X-ray showed a gross foetal abnormality, usually anencephaly. Taussig claims that anencephaly is always associated with hydramnios, but search of the hospital records showed nine cases of anencephaly without hydramnios. Even when the X-ray appeared normal, hydramnios carried a serious prognosis for the baby—in seventy-eight cases the foetal mortality was 33 per cent. and in five of these cases an abnormal baby survived. There was an increased incidence of pre-eclamptic toxæmia in cases of hydramnios. Uterine inertia was a hazard in labour and led to an abnormally high forceps and Cæsarean section rate. The speaker concluded with a reference to the treatment of hydramnios and to the prognosis in future pregnancies after the delivery of an abnormal baby.

A meeting was held on the 25th January, Mr. H. L. Hardy Greer taking the chair. Mr. G. Boyd described a case of malignant hypertension. The patient, a young primigravida, had a family history of hypertension. After a twenty-week miscarriage she had eight Jacksonian fits on the eighteenth puerperal day. Thrombosis of a cerebral vein was suspected, but persistent hypertension and albuminuria, with marked retinopathy, pointed the way to the correct diagnosis. The patient showed some improvement after bilateral lumbar sympathectomy. The speaker discussed the differential diagnosis of puerperal fits with particular reference to eclampsia.

Dr. J. Watson described a case of gross cervical œdema occurring in a primigravida at thirty-seven weeks, and suggested that it might have been due to the wearing of very tight corsets in an effort to conceal pregnancy. The œdema resolved after rest in bed and the patient later had an uncomplicated delivery.

Dr. F. G. Grant described a case of annular avulsion of the cervix. This occurred in a young primigravida after a labour of seventy-six hours. The patient had had moderate contractions throughout labour and the head was deeply engaged in the pelvis. The cervix failed to dilate beyond two fingers, and while preparations were being made for Cæsarean section the patient delivered herself unexpectedly. It was then found that this had occurred because a ring of cervix one inch in thickness had been torn away—the ring surrounded the external os which was still only two fingers dilated. There was no hæmorrhage from the torn cervix and the patient made an uneventful recovery.

Dr. G. B. Gibson described a case of massive cervical fibroid, which gave rise to dystocia in labour. This was mistaken for a placenta prævia until the baby was extracted through the centre of it with great difficulty during a lower segment Cæsarean section. The capsule was resutured initially and a hysterectomy was performed later in the puerperium.

At a meeting held on the 22nd February Mr. H. L. Hardy Greer took the chair and Dr. R. S. Allison discussed some of the nervous diseases associated with pregnancy. He pointed out that disseminated sclerosis was not necessarily a rapidly progressive malady. There was no evidence that pregnancy influenced the progress of the disease in its early stages—in late cases daily ambulation was essential because bed rest caused rapid progression of the paraplegia. The speaker also described the pathology, diagnosis, and treatment of cerebral venous throm-

basis and emphasised that its incidence was considerable. This condition was characterised by puerperal headache, fits, and transient coma, followed by focal neurological signs. Treatment was by sedatives and heparin—if the superior longitudinal sinus were involved, daily lumbar puncture was of value.

## REVIEW

THE CHILD IN HEALTH AND DISEASE. By C. R. Grulee and R. C. Eley.  
Pp. 1,006. Baillière, Tindall & Cox. 66s.

THIS volume follows the now usual practice of making use of a number of expert contributors on the different branches of the subject, the whole edited and co-ordinated to present a balanced view of the subject as a whole. In this case there are over fifty contributors, and the editors state in their introduction that the aim of the book is to emphasize the practical application of the study of pædiatrics at the bedside, rather than the purely scientific advances in the subject.

With nineteen sections and ninety-one chapters there is inevitably some unevenness, and a tendency to overlap and repetition, but the editors' aims have, in general, been well realised. It is doubtful, however, whether this book is suitable for students, as they suggest; certainly few students in British medical schools would have the time or inclination to read the 1,006 pages which it contains.

The range and size of the work prevents any detailed discussion of the individual sections. In the introductory section on "general considerations" one finds a short chapter on pædiatric pathology, which is valuable to the general pædiatrician, though, presumably, too elementary to be of value to pathologists. There is a useful chapter on E.N.T. procedures, and a short section on surgical pædiatrics, including good advice on preparing the child for an operation. (Unfortunately by the time the child comes to the pre-operative stage it is usually too late to advise the parents.)

As in most American pædiatric text-books, a rather larger age group is covered than in British practice, and a section on adolescence includes a chapter on gynæcological disorders in adolescents, with the comment that these patients are most often courteously referred from the pædiatrician to the gynæcologist, and, equally courteously, from the gynæcologist to the pædiatrician.

There are a few statements which are rather unorthodox in various parts of the book; in the chapter on cerebral palsy it is assumed that the increased incidence of this condition in premature infants cannot be due to birth trauma, because of their small size and easy delivery. In discussing the ætiology of cirrhosis in childhood, the rather surprising figure of sixteen per cent. due to chronic alcoholism is quoted. Intracranial hæmorrhage is blamed for one-third of neonatal deaths, a much higher figure than usually found in this country. It is interesting to note that B.C.G. vaccination is considered to be of doubtful value, but great reliance is placed on mass miniature radiography for the control of tuberculosis. In the section on aspiration pneumonia, which lays valuable stress on the need to avoid oily nasal preparations and the danger of forcing cod-liver oil into a struggling infant, it is stated that vegetable oils are more irritating than mineral oils, which is contrary to general teaching and practice (e.g. the use of iodised poppyseed oil for bronchography).

The chapter on intestinal parasites is spoiled by the lack of correlation between illustrations and text, the references in the text having no connection with the numbered figures quoted; also the illustration of ova has no key to identify them.

Despite these minor failings, the book is, in general, at a high standard of sound practical teaching, and will be helpful to postgraduate students of child health and to practising pædiatricians. Unfortunately, devaluation has made it a rather expensive addition to the practitioner's library.

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basis and emphasised that its incidence was considerable. This condition was characterised by puerperal headache, fits, and transient coma, followed by focal neurological signs. Treatment was by sedatives and heparin—if the superior longitudinal sinus were involved, daily lumbar puncture was of value.

## REVIEW

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## THE NORTHERN IRELAND COMMITTEE OF THE NUFFIELD PROVINCIAL HOSPITALS' TRUST

THE first meeting took place recently in Belfast of the Northern Ireland Committee of the Nuffield Provincial Hospitals Trust. It will be recalled that in 1942 there was constituted the Northern Ireland Regional Hospitals Council, under the chairmanship of Mr. D. Lindsay Keir (now Sir David Lindsay Keir). This Council was formed under the ægis of the Nuffield Provincial Hospitals Trust, and it will be remembered mainly for its "Survey of the Hospitals Services of Northern Ireland," which was published in 1945, and, following that, its "Plan for the Hospital Services for Northern Ireland, published in 1946 as "The Red Book." Many tributes have been paid in Parliament and elsewhere to the assistance these publications afforded to those responsible for framing the present Hospitals Service.

With the passing of the Health Services Act (Northern Ireland), 1948, it was believed that the Council, under its then Constitution, had fulfilled its main purpose. It was felt, however, that there was a place for an independent voluntary body, fully representative of all the health and social services, and, accordingly, the final act of the Regional Hospitals Council was to draw up a new Constitution, providing for the formation of a new committee, to be known as the Northern Ireland Committee of the Nuffield Provincial Hospitals Trust.

Under its Constitution, this Committee has, for its purpose :—

- (a) To assist the co-ordination on an area basis of hospital services.
- (b) To initiate and promote research which aims at the raising of the standard of hospital services throughout the area.
- (c) To advise on any proposals which serve the foregoing purposes.

Further, its functions are to advise and co-operate with the statutory and other bodies on such matters as may seem conducive to the improvement of the hospital, ancillary, or auxiliary services within the area, and also to initiate, promote, and encourage, whether independently or in consultation with appropriate bodies, research on causation, prevention, and treatment of disease, and for the improvement of the hospital, ancillary, and auxiliary services. It aims to achieve in every possible way the raising of the standard of service to the patient throughout the area.

The first meeting of this Committee was mainly concerned with business of a formal character, but preliminary discussion took place on several projects which might ultimately tend to fulfil the fundamental aims of the Committee.

It was decided that the existence of the Committee should be made public, so that any person or body of persons interested might have an opportunity of submitting for the consideration of the Committee proposals coming within its purposes and functions.



## REVIEWS

THE PARATHYROID GLANDS AND METABOLIC BONE DISEASE. By Fuller Albright, A.B., M.D., and Edward C. Recfenstein, jun., A.B., M.D., F.A.C.P. Pp. XXVI + 393 with 157 figures. London : Baillière, Tindall & Cox. 44s. net.

THIS work is the product of twenty-four years research in the metabolic ward of the Massachusetts General Hospital. The book opens with an account of the parathyroid glands from the physiological and clinical aspects, and, after considering the mode of action of vitamin D, devotes more than half its space to a study of metabolic bone disease, particularly osteogenesis, osteoporosis, osteomalacia, osteitis fibrosa, and osteitis deformans.

The action of the parathyroids is correlated with the structure and chemistry of bone. The authors belong to the school of thought which holds that primary function of the parathyroid hormone is in relation to the control of excretion of phosphate by the kidney, and that the changes in bone are secondary to the resulting fall in the serum phosphorus. Good clinical accounts of hypo- and hyper-parathyroidism are given, those on the latter being especially full. Primary hyper-parathyroidism is divided into four groups, depending on the presence or absence of co-existent bone disease and/or kidney disease.

In discussing the bone changes they prefer to call von Recklinghausen's disease "osteitis fibrosa generalisata" instead of "cystica." The bone tumours sometimes found in this disease, and called by Hunter and Turnbull "osteoclastomata," are, in the authors' opinion, equally suitably called "osteoblastomata." They point out that, "whereas every case of epulis is certainly not hyper-parathyroidism, this diagnosis must be carefully considered in every case." In the treatment of overactivity of the glands emphasis is laid on the danger of parathyroid poisoning, and its prevention by keeping the patient on a low calcium intake, even in the presence of osteoporosis. The authors' conceptions at times cut across current thought and practice, e.g., in the case of a schoolboy of 14 who was admitted with a fracture of the femur through a solitary cyst; this boy did badly after operation, and, as he had a high serum calcium with extreme demineralization of the immobilized parts of his skeleton, a diagnosis of hyperparathyroidism was made by the surgeon in charge; Albright explained the clinical course in this case as: (1) cyst; (2) fracture; (3) immobilization; (4) osteoporosis of disuse; and (5) hypercalcaemia due to the rapidity of the osteoporosis; and, accordingly, he recommended the boy to bear weight on the broken leg, with the result that the blood calcium fell from 14.4 to 11.3 mg. per 100 c.c. in a month. Similarly, they explain the hypercalcaemia of multiple myeloma and metastatic malignancy by inferring that the dissolution of the bone salts into the blood stream occurs more rapidly than the kidney can excrete them.

In the operative treatment they make a number of interesting points. The operation should only be undertaken by a surgeon who has made a special study of the appearance and location of normal parathyroids. Of their first sixty cases, eleven had tumours in the anterior mediastinum and five in the posterior mediastinum. Their first case had his tumour in the anterior mediastinum and was removed at the seventh exploration.

In an interesting chapter on the mode of action of vitamin D and dihydrotachysterol (A.T. 10), they conclude that vitamin D, in addition to increasing calcium absorption from the gut, has a second action in increasing the urinary phosphate output. A.T. 10 has an action similar to calciferol, and its phosphate excretion effect is greater than its calcium absorption effect and therefore it more closely resembles the action of parathyroid extract and, indeed, has proved of therapeutic value in hypo-parathyroidism.

The metabolic bone diseases are considered first generally and then in detail as regards X-ray appearances, histology, and chemistry, with special references to osteoporosis and osteomalacia. The authors make the interesting claim that one and probably two of the three famous cases

described by von Recklinghausen in 1891 were not examples of osteitis fibrosa generalisata and hence of hyperparathyroidism, but were suffering from "polyostotic fibrous dysplasia." The book concludes with an account of Paget's disease, with an ingenious, if not very satisfactory, attempt to explain its pathological physiology. It, however, has some useful points in treatment, one or two glasses of milk daily and large doses of calciferol, but in cases of Paget's disease who are immobilized with fractures or confined to bed, should have a low calcium phosphorus and vitamin D intake and a high water intake.

The book is well produced, with many useful diagrams, X-rays, and histological photographs, though a few are not clearly reproduced. Enough has been written to show that the book is a happy union of biochemistry and clinical medicine brought about by many years careful thought and much industry.

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**DISEASES OF THE NERVOUS SYSTEM.** By F. M. R. Walshe, M.D., D.Sc., F.R.C.P., F.R.S. Sixth Edition. Edinburgh: E. & S. Livingstone Ltd. 17s. 6d.

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The book is comprehensive, and, while there may be vagueness about the value of treatment mentioned, there is a list of references at the end of each chapter. Some of the therapeutics, too, are merely historical.

There are many illustrations, some coloured, and the large, clear print allows instruction to be acquired pleasantly.

H. A.

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