NOVEMBER, 1941

THE ULSTER MEDICAL JOURNAL

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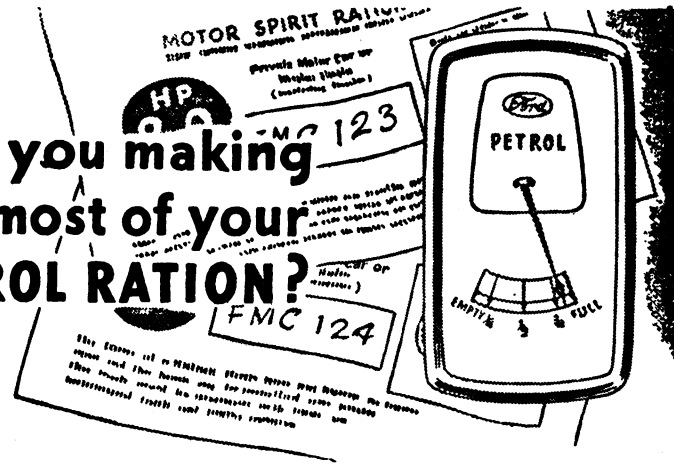

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References: British Medical Journal, November 16th, 1940, page 679.
The Lancet, November 16th, 1940, page 627.
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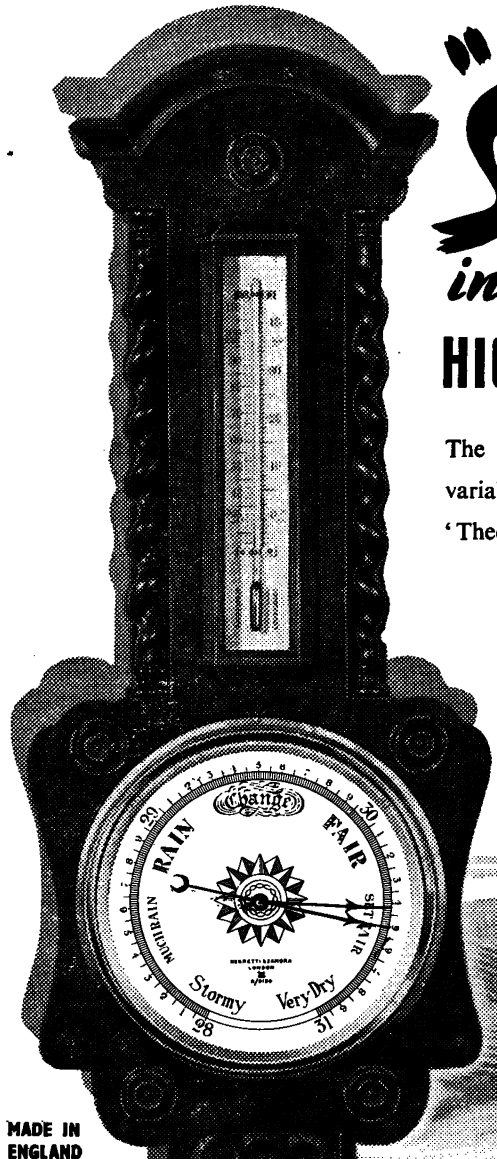

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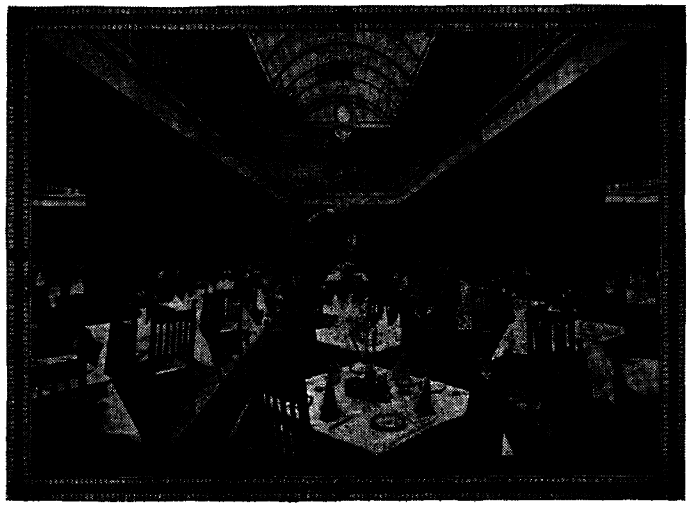

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THE
ULSTER MEDICAL JOURNAL

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Vol. X 1st NOVEMBER, 1941 No. 2

Generalised Neurofibromatosis

By J. H. BIGGART, M.D., D.SC.

Professor of Pathology, Queen's University, Belfast

WHILST neurofibromatosis, in more or less localised form, is not infrequently seen, most of our knowledge concerning the histological structure of the nodules is gained from biopsy specimens. The following case, illustrating as it does the widespread distribution of the process and the nature of the associated lesions, seemed, therefore, worthy of report.

CLINICAL HISTORY.

The patient, a man of fifty years of age, was admitted to the Royal Infirmary, Edinburgh, complaining of pain in his left foot and some weakness in the right arm. A week prior to admission he had noticed that the middle toe of the left foot was swollen and painful. The swelling evidently arose in relationship to a corn. The pain and swelling increased, and the skin finally broke down with the discharge of pus.

The weakness in the right arm had developed during the past eight months. It was accompanied by a feeling of numbness and tingling. These symptoms had become much more marked in the two months prior to admission.

On examination, the middle toe of the left foot was found to be the site of a marked infective necrosis with a thick purulent discharge. The adjacent toes were also red and swollen.

There was a well-marked generalised von Recklinghausen's disease, the whole body being covered by multiple pedunculated and pigmented nodules. There was weakness of the muscles of the right upper limb, most marked in the flexor group of muscles, but present also in the extensors. Wasting and weakness of the small muscles of the thumb and the first dorsal interroseous muscle were present. There was little appreciable loss of sensation in the affected arm. A hard immovable swelling was found in the right side of the neck. This was pyramidal in shape, the apex reaching as high as the cricoid cartilage, whilst the base continued into the thorax underneath the clavicle.

Other examinations were essentially negative. The blood-pressure was 150/90. The infected toe was amputated, but the patient died fourteen days later from a rapid terminal enlargement of the tumour in the neck. The clinical impression was, that one of the nodules of the von Recklinghausen's disease had become sarcomatous.

AUTOPSY (437/36).

The body is that of a well-developed male of middle age. Scattered diffusely over the trunk are numerous small tumours, averaging one to two mm. in diameter. Some of these are pedunculated; some sessile. All of them are of subcutaneous origin. A few similar nodules are also found on the proximal parts of the limbs, but their distribution is irregular and cannot be said to follow the course of any particular nerve or nerves. Associated with these tumours there is a marked degree of pigmentation. This is not diffuse, but appears in small patches resembling freckles. These are not present over the face or hands. In the right supra-clavicular triangle there is a large firm mass, which displaces the trachea towards the left.

Body cavities.—The pericardial sac is normal. The apex of the right lung is slightly adherent to, but easily separated from, the tumour, which extends down into the mediastinum as far as the level of the bifurcation of the trachea. There is a fibrino-purulent exudate over the base of the right lung. The peritoneal cavity contains no free fluid.

Heart.—This is of normal size. The epicardium is thin and translucent. There are no lesions in the right auricle or ventricle. The left ventricular wall shows no hypertrophy. The endocardium is thin and the valves normal. Atheromatous changes are marked in the coronary arteries.

Lungs.—There is a fibrino-purulent exudate over the diaphragmatic surface of the left lung. This lung is heavier than normal, and on section numerous small peribronchiolar areas of consolidation, which are becoming confluent, are found. The dark colour of these areas suggests that there may have been some aspiration of gastric contents. The lining of the bronchi is œdematous and congested. The bronchial glands are not appreciably enlarged.

The right lung shows a smooth pleura, which is lightly adherent to the tumour mass in the region of the apex. The upper and middle lobes are crepitant. The lower lobe shows a patchy consolidation similar in type to that seen in the left lung. The peribronchial lymph-nodes show congestion, but no metastases.

Spleen.—This is of normal size. The capsule is thin. On section, the malpighian corpuscles are distinct. The pulp is red, but not softened. The vessels are thickened, and the trabeculæ prominent.

Liver.—This is of normal size and shape. There are no glands in the hilum, though the gastro-hepatic ligament is greatly thickened. The common bile-duct is patent and the gall-bladder normal. On section, the liver lobules are quite distinct, though their central zones are pale. The bile-ducts and radicles of the portal vein are patent.

Pancreas.—The ducts are patent. On section, the acinar tissue appears normal.

There are two small nodules about three mm. in diameter attached to branches of the sympathetic nerves on the posterior surface of the head of the gland.

Stomach and Intestine.—The stomach shows nothing of note. At the duodeno-jejunal junction there is a small (four mm.) nodule on the serous surface. The terminal ileum shows several adhesions and much thickening of the mesentery. There is some black pigmentation of this area. A large inguinal hernial pouch is found with similar pigmentation, and the findings suggest that there has been a herniation of the terminal ileum with subsequent reduction and fibrotic repair of the damaged mesentery.

Genito-urinary system.—The kidneys are of normal size. The capsule strips easily, leaving a smooth homogeneous surface. On section, the organs appear acutely congested, but the cortex is of normal width and the striæ regular. The medulla shows nothing of note. The pelvis is normal. There is some atheroma of the renal arteries. The ureters and bladder show no lesions.

Adrenals.—These appear to be of normal size. Both together weigh 17 gm. On section, the cortex is of normal width, and appears well filled with lipoid. The medulla of the right gland is of normal size. That of the left gland shows a small circumscribed nodule, 3 mm. in diameter. On this side also there is an elongated tumour, 2.5 cm. by 0.7 cm. by 0.5 cm., on a branch of the sympathetic plexus running to the gland.

Neck organs.—There is a large piece of orange impacted in the œsophagus at the level of the bifurcation of the trachea. At this level there is some narrowing of the lumen from pressure by the adjacent neoplasm.

The trachea is congested, but does not appear compressed, though it is displaced well towards the left.

The thyroid is rather small. On section, it appears to be a normal colloid-containing gland. The parathyroids are not enlarged.

The mass in the right supra-clavicular region extends downwards into the upper part of the right thorax and medially into the mediastinum (fig. 1). It can be easily separated from the surrounding structures. The trunks of the right brachial plexus appear to emerge from its lateral surface, whilst the three lower cervical roots enter into its medial surface. The phrenic nerve appears to course through the centre of the tumour, and, whilst thickened, can be quite easily dissected from the tumour (fig. 1). On section, the tumour appears as a white, hard, structureless mass with a few scattered areas of necrosis. The external jugular vein which courses over its anterior surface shows a recent thrombosis. The carotid artery runs for some distance through the tumour, but is apparently normal.

The aorta shows only a little atheroma.

Brain.—The dura is normal. The meninges over the left temporal lobe are thickened and rather opaque. The brain appears large. The vessels at the base are dilated, but not atheromatous. There are no tumours on the acoustic or optic nerves. Section following fixation shows nothing of note.

Hypophysis.—This is of normal size, but slightly compressed by arterio-sclerotic internal carotid arteries. On section, nothing abnormal is noted.

Spinal cord.—Naked-eye examination of the spinal cord shows no lesions. A small circumscribed nodule is present on one of the roots of the cauda equina. Apart from this, the dorsal nerve-roots appear quite normal in their course inside the dura. Several of the dorsal-root ganglia, however, in the lumbar region are enlarged to three or four times their normal size, and from these greatly thickened nerves arise.

Peripheral nervous system.—The right brachial plexus as it emerges from the tumour already described, is found to consist of trunks, which are four to five times their normal diameter (fig. 1). They feel extremely hard. This increase in size is continued into all the branches of the plexus. On the nerve to the teres major (fig. 1) is a large fusiform swelling, 6 x 3 x 3 cm., which on section is seen to be composed of a rather hard but translucent tissue. The left brachial plexus is not so markedly enlarged, but there are definite fusiform enlargements on the median trunk.

The lingual and hypoglossal nerves show a diffuse thickening.

The sacral plexus is very definitely enlarged, the various roots feeling varicose, and on section showing a rather uneven but diffuse thickening. The right sciatic nerve is immense. As it is followed down the leg, it shows a large fusiform swelling. Here the nerve is 3 cm. thick and 5 cm. broad. This increase in diameter is continued into its branches. Longtitudinal and transverse sections of these parts of the nerve which appear merely diffusely thickened, show many small hard translucent nodules occurring in the course of its component nerve bundles.

The left sciatic nerve appears only slightly enlarged. A few firm nodules can be felt in its substance, and these become more obvious in the posterior tibial nerve. On a branch of the external popliteal nerve, about three inches above the external malleolus, is a large fusiform swelling, 8 x 4 x 3 cm. (fig. 3). In structure it resembles that found on the nerve to the teres major.

Practically all the peripheral nerves show irregularity in their contour. Sometimes it is a diffuse increase in size. In others it is definitely nodular, and the nodule may affect the whole or part of the diameter of the nerve, or of individual nerve bundles. Occasionally the mass is large, as in the case of the large fusiform tumours described above.

Autonomic nervous system.—This is affected in like manner and to the same degree as the peripheral nervous system.

The right recurrent laryngeal nerve and some of the branches of the vagus are three times their normal diameter. The left thoracic chain appears relatively normal. The right shows a large fusiform swelling at the level of the sixth thoracic segment (fig. 3), and there is enlargement of the seventh thoracic ganglion. The cœliac ganglion is slightly enlarged, and its branches to the viscera are everywhere prominent, some of them measuring 3 mm. in diameter. The thickening and varicosity of the plexus in the gastro-hepatic omentum has led to an apparent thickening of this structure. The nerves to the adrenals are prominent, and one branch to the left adrenal shows the fusiform swelling (2.5 x 0.5 x 0.5 cm.) already noted. The hypogastric and internal iliac plexuses show no apparent deformity,

but all their branches are more readily identified and may be a little thicker than normal.

Microscopical Examination.

In all, over sixty blocks of the various tissues have been examined by various methods, but detailed descriptions will only be given of those sections which show changes pertinent to the disease process in the nervous tissues. The patient died from a broncho-pneumonia, which was evidently the result of the aspiration of gastric contents. The parenchymatous tissues of the heart, liver, pancreas, and kidneys show little of note. Thyroid, testes, parathyroids, and hypophysis are normal.

Adrenals.—The right adrenal shows no lesions. Some of the small perivascular branches of the autonomic nervous system show an intense cellularity, which appears to be due to a proliferation of the neurilemmal cells. The left adrenal appears slightly enlarged. The section shows two small nodules of entirely different structure, which extend from the region of the central vein through the medulla and cortex to the periadrenal fat. One of these, the more central, is composed of large polygonal cells resembling those of the normal adrenal medulla, but three to four times as large. These large cells have a loose alveolar arrangement, the only stroma being a meshwork of capillaries. Some of these cells contain a melanin pigment; others show various forms of intracytoplasmic inclusion bodies, which in most instances are strongly acidophilic. All of these cells give a positive chromaffin reaction. The nuclei are large and vesicular, and in some instances show much resemblance to the nuclei of ganglion cells. The tumour projects into the adrenal vein, and fills about two-thirds of its lumen. This intravascular projection is still covered by the vascular endothelium, and there is no thrombosis. The tumour appears to have the characteristics of a chromaffinoma (phæochromocytoma).

The other nodule (3 mm. in diameter) is composed of whorls of axis cylinders, separated by fine bands of fibrous tissue fibrils running parallel to the axis cylinders. In the nodule one finds a few scattered ganglion cells, some of which are binucleated.

Peripheral nerves.—The large tumour in the right supra-clavicular region is composed of interlacing bands of large spindle-shaped cells with oval nuclei (fig. 6). These nuclei have a vesicular pattern of chromatin, and mitotic figures are common. The tumour is found to be infiltrating many of the smaller blood-vessels. It is worthy of note, however, that no metastases were found in the lungs, brain, liver, vertebral column, or skull. Silver stains show a large amount of reticulum. The cells themselves have long bipolar cytoplasmic processes. Areas of necrosis, secondary to the vascular occlusion, are common.

The tumour involves the trunks of the brachial plexus. These trunks are infiltrated by the tumour-cells. There is no spread along the perineural lymphatics. Instead, the tumour seems to spread directly along the nerve-tissue itself, and adjacent small nerve-twigs are almost completely replaced by this intraneural sarcomatous growth.

Histological examination of other nerves reveals a process which can best be

described in general form. Nerves showing no actual growth are seen to be generally thickened. This thickening is due to a laying down of fine fibrils of fibrous tissue parallel to the course of the myelin sheaths—a fibrosis very comparable to the gliosis which occurs in the tracts of the spinal cord in a plaque of disseminated sclerosis, and which might well be described as an "isomorphic fibrosis." This fibrous tissue bears a very intimate relationship to the essential nervous tissue, forming, as it were, a sheath around each individual myelin sheath. In such diffusely thickened nerves the perineurium appears quite normal, and it is obvious that the new formation of fibrous tissue does not arise from the perineurium, but from one of the more intimate sheaths of the nerve-fibre. In other areas the process may be apparent as a nodule on the course of the nerve. In the less complicated of such nodules one merely finds a local exaggeration of the more diffuse change described above. There is more fibrous tissue around the nerve fibres, and in Weigert-Pal preparations the myelin sheaths are found widely separated by this new fibrous formation, but they still course unimpaired through the nodule, and show their normal appearance when they finally emerge distal to it. In the larger nodules, however, such as those found on the right sciatic, left external peroneal, etc., whilst some of the myelin sheaths can still be stained, others have disappeared. Axis cylinder stains show that the myelin-sheath stains give an erroneous impression of the number of nerve-fibres coursing through the tumour. It would appear that some naked axis cylinders persist, even after destruction of the myelin sheaths. These larger nodules show a hyaline change in the fibrous tissue around the vessels, and a curious granular groundwork, which does not give a positive reaction with mucicarmine, between the bundles of reticulum.

This description fits most of the histological changes seen in the nodules. However, it is apparent from the description that we are dealing with the end-result of a proliferative lesion, and accordingly careful search was made for foci of active progression. Such were found in a nerve in the vicinity of the left adrenal, and in a branch of the left brachial plexus. Individual nerve-fibres in the more chronic lesions also showed phases of activity. In these areas the cellular reaction appears to be limited to individual nerve fibres. Surrounding one myelin sheath, or occupying the space where a myelin sheath had been, one finds a proliferation of three or four neurilemmal cells apparently forming a syncytium (fig. 8). Around these is a fibrous-tissue sheath. This sheath seems to become thicker and fibrous septa appear between the proliferating intra-sheath cells. Even in the active areas it is difficult to find any evidence of cellular activity in the interstitium of the nerve. In these active areas the perineurium seems to play no part in the proliferative process.

In the larger and older nodules a curious syncytium is found surrounding the bundles of reticulum. This cellular syncytium, however, is not an intrinsic part of the process. It is not constant. It is not seen during the active proliferative phase.

The skin, or rather subcutaneous nodules, show a histological appearance which is more difficult of analysis. Nerve-fibres can be traced into these growths. For

the most part, however, they consist of interlacing bands of cells, the origin of which cannot be so clearly seen as in the nodules occurring in the larger nerve-trunks. In some instances microscopical lesions surround the sweat glands, and suggest an origin from the autonomic nerve supply of these structures (fig. 5).

Dorsal-root ganglia.—The process resembles in all details that already described. There is a multiplication of the capsular cells, and in a few instances these have completely replaced the ganglion cells.

Autonomic nervous system.—The nodules and diffuse thickenings found in the autonomic nervous system duplicate in structure those found on the somatic nerves. Histological examination shows that the neurofibromatous change is extremely widespread. Apart from the gross changes noted at autopsy, small perivascular nerves in the liver, pancreas, renal pelvis, and lungs show similar neurilemmal proliferations and intraneural fibrosis.

Brain.—There is some ependymitis, and some periaqueductal gliosis, but neither of these changes appears of neoplastic character. No changes comparable to those seen in tuberous sclerosis, and no glial nodes are found. The cranial nerve-roots show no lesions.

Spinal cord.—The nodule already noted on the cauda equina shows much the same histological structure as that found in the nodules on the peripheral nerves. Axis cylinders are present, and there are none of the histological characters of the more usual neurinoma. Nothing abnormal is seen in the cord-substance.

Anatomical diagnosis.—

Septic gangrene of toe.
Generalised neurofibromatosis of peripheral and autonomic nervous systems.
Chromaffinoma of left adrenal : ganglioneuroma of left adrenal.
Sarcomatous change in neurofibromatous nodule in right supra-clavicular region.
Pressure upon trachea and œsophagus.
Aspiration pneumonia.

Discussion.

The present case furnishes an additional record of the association of generalised neurofibromatosis with other anomalies of the nervous system. The histological studies seem to help in the elucidation of the pathogenesis of the neoplastic process. Verocay (1910) first pointed out that tumours of the peripheral nerves were not due to a simple connective tissue hyperplasia. The palisading of the nuclei, however, which he emphasizes is a well-recognised histological feature of the solitary neurinoma, but is by no means characteristic of von Recklinghausen's disease, though small areas of this type of tissue in the nodules may undoubtedly occur. Masson (1932) has produced experimentally proliferations of the neurilemmal cells, which show many of the characteristics of the neurinoma, and which seem to have convinced this investigator that the neurilemmal cell is the cell-type of these tumours, and also that this cell may produce a type of argyrophilic reticulum. However, the application of Verocay's views to neurofibromatosis has not met with

general acceptance (Kaufman, Stout), and so in the analysis of the present case attention has been concentrated on those histological features which might support or refute the essential neural origin of the tumours. As will be noted from the microscopical description of the lesions, active cellular proliferation occurs in the early stages inside the membrane surrounding each myelin sheath. At this stage no other cellular activity is encountered. The perineurium remains normal; there is no cellular activity in the fibrous septa of the nerve-bundles, and no cellular activity, which might be interpreted as endoneurial, outside the neurilemmal sheath. The proliferating neurilemmal cells preserve their syncytial character. In this stage the myelin sheath may disappear, but there is usually preservation of the axis cylinder. Connective tissue fibrils then appear between the proliferated nuclei, and these seem always to be laid down parallel to the long axis of the neurilemmal cells, and therefore parallel to the original nerve-fibre. In some instances the syncytial cytoplasm of the proliferating neurilemmal cells is seen divided, as it were, into compartments by these fibrils. This process seems to proceed until the nerve-fibre, with or without its myelin sheath, is completely surrounded by a fine bundle of parallel reticulum fibres, which appear to be still enclosed in the original but somewhat thickened neurilemmal sheath. In this process there seems occasionally to be some lengthening and tortuosity of the original nerve-fibre, for even on transverse section a few myelin sheaths may be found running transversely for various distances. An accentuation of this lengthening and tortuosity results in the appearance of the plexiform type of tumour.

In all this period of activity our histological observations tend to agree with those of Masson, that it is the neurilemmal cells, and these alone, which proliferate. However, the nature of the material renders it difficult to be certain that the reticulum is formed by these cells, and that it may not be laid down by the endoneurium in response to the activity of the neurilemmal cells. Doubts as to the exact nature of the endoneurium have recently been raised by Masson (1932). This author states that the structure which has been called endoneurium, may be divided by histological analysis into two systems. Firstly, there is an interstitial connective tissue-fibre lying in the angle where three nerve-fibres meet. This system cannot be held responsible for the sheath-like proliferation of connective tissue-fibrils. Secondly, there is a fine fibrillar sheath around each individual nerve-fibre—the Plenk-Laidlaw sheath. This sheath, which consists of delicate interlacing fibrils, is closely applied to the neurilemmal cells, and Masson's studies seem to indicate that it is part of the neurilemmal sheath rather than a distinct entity. In neurofibromatosis, therefore, whilst the cellular activity appears confined to the neurilemmal cells, the actual multiplication of fibrils occurs in connection with this Plenk-Laidlaw sheath.

In the older nodules the proliferated cells disappear, and one finds relatively acellular bundles of reticulum, in which, however, it is sometimes possible to demonstrate the original axis cylinder. In these too one finds a curious finely granular ground-substance between the bundles of fibrils. The nature of this substance and its derivation cannot be settled from histological examination

alone. It does not give a positive reaction with mucicarmine. Running through this groundwork, and surrounding the individual fasciculi, is a cellular syncytium. The cells composing this syncytium appear to be of endoneurial origin. The cells of the normal endoneurium show the same anastomotic prolongations and the same thin membrane of cytoplasm around their nuclei. These anastomosing cells, however, cannot be regarded as an essential part of the process. They tend to be most prominent in those areas where the cellular activity of the neurilemmal sheath has ceased : they may only be formed in one part of the nodule; and they are not obvious in the early proliferative stages of the process.

It would appear, therefore, that in neurofibromatosis the active process is on the part of the neurilemmal cells, analogous to the process which Verocay suggested, and Masson seems to have demonstrated in respect of the neurinoma. The different histological appearances of the two types of growth—the neurofibroma and the neurinoma—seems to be due to the fact that, whereas in the neurinoma there are no myelin sheaths or axis cylinders and the cellular proliferation is independent of these structures, in the neurofibroma the cellular activity occurs in relationship to individual nerve-fibres, which form a scaffolding for the cellular proliferation and new tissue formation, and so modify the ultimate histological architecture of the tumour.

Whilst the histological study of this case, therefore, suggests the importance of the schwann cells, the nature of the associated lesions also favours this suggestion. It is now more or less accepted that the neurilemmal cells develop from the neuroectodermic crest, and hence have ultimately the same origin as the ganglion cells and the cells of the adrenal medulla. Hence the association of the chromaffinoma and the ganglioneuroma in this case would support the idea that von Recklinghausen's disease has its origin in some congenital abnormality of the neuroectodermic crest. The association between neurofibromatosis and tumours of the adrenal medulla has also been noted by Zuzuki (1909), Kawashima (1911), and others. Such an idea is further supported by the occasional occurrence of lesions in the central nervous system—gliomas of the optic chiasm (Bailey) bilateral acoustic neurinomas. In a few cases the central lesion has been tuberous sclerosis (Orzewski and Howicki, Bassoe and Nuzum), and even in patients free from the stigmata of this process, small glial nodes may be found scattered throughout the brain and spinal cord. It might be suggested, therefore, that some congenital abnormality of the neuroectoderm may result in a closely-related variety of lesions—tuberous sclerosis, central, or peripheral von Recklinghausen's disease. These lesions may occasionally occur together, or only one manifestation of the defective anlage may be present.

The other points of interest arising in the present case are : (1) the widespread involvement of the autonomic nervous system, (2) the chromaffinoma, and (3) the malignant transformation of the cervical nodule. The case as a whole duplicates in these points that described by Herxheimer and Roth (1914). Their case showed multiple tumours of the skin, tumours of the intercostal and lumbar nerves, a large pelvic sarcoma, a chromaffinoma of the adrenal medulla, and multiple subserous

nodules on the jejunum. Widespread involvement of the autonomic nervous system has been noted by Czerny, Harbitz, and Verocay.

Chromaffinoma of the adrenal is frequently associated with paroxysmal hypertension, but the clinical record in this case gives no indication of this having occurred, though the period of observation is too short to be conclusive. The curious growth of this tumour into the adrenal vein without metastases has also been noted by Manasse (1893).

Malignant transformation of a nodule is not infrequent. This may be manifested by an increase in cellularity of the tumour without any marked changes in its histological pattern. Such active areas are found in the present case in some of the splanchnic nerves, in the left brachial plexus, etc., and in the event of biopsy, one would have hesitated to pronounce them benign. In his recent review of these tumours, Stout (1935) states that they rarely metastasize, though they tend to recur locally. The extensive involvement of the nerve trunks, however, by the proliferative process renders the idea attractive that the subsequent growth near the site of removal may be, not a recurrence in the usual sense of the term, but a recrudesence of cellular activity in the same or in an adjacent nerve. From the nature of the process it is obviously impossible to remove all the affected tissue, for as is seen in this case, the cellular proliferation may extend as far as the muscle spindle, or the terminal nerve-twigs around the sweat-glands or the blood-vessels. The possibility of multiple sites of cellular activity may also be held to influence treatment. Habermann, for example, reported a case in which primary malignant growths were found in the sciatic, ulnar, and occipital nerves.

The malignant tumour in the right supraclavicular region, however, is not merely a rapidly-growing nodule. Here the histological picture is that of a spindle-cell sarcoma, and it is surprising, in view of the extensive invasion of the blood-vessels, that no metastases were found. However, in a series collected by Stout of ninety-one cases of malignant change in von Recklinghausen's disease, only eighteen (twenty per cent.) showed metastases. In the present case it was impossible to demonstrate any axis cylinders or myelin sheaths in the sarcoma—this finding agreeing with the clinical history of the recent development of paralysis in the arm-muscles. It would seem, therefore, that the occurrence of motor-paresis in a patient with neurofibromatosis is at least suggestive of malignant change, for even large benign nodules on the course of a nerve interfere little, if at all, with motor function, though there may be some sensory loss.

Finally, it may be noted that the nodules may be stimulated to active growth by conditions which are associated with changes in the endocrine organs. Thus in pregnancy, latent nodules may become apparent, and others rapidly increase in size. With the cessation of the puerperium, growth diminishes, but may reappear in a subsequent pregnancy. Of interest in this connection is the recent observation by Ferguson (1935), that the blood of patients suffering from von Recklinghausen's disease, neurosarcoma, and malignant melanoma contains the hormone intermedin —the melanophoric hormone of the hypophysis.

SUMMARY.

(1) A case of generalised von Recklinghausen's disease is reported.

(2) The generalised subcutaneous tumours were associated with tumours on the deep nerves, marked changes on the autonomic nervous system, neurosarcoma in the right brachial plexus, chromaffinoma and ganglio-neuroma of the adrenal.

BIBLIOGRAPHY.

1. BASSOE, P. AND NUZUM, F. (1915): *Jour. Nerv. and Mental Disease,* xlii, 785.
2. COPELAND (1935): *Amer. Jour. Cancer,* xxiv, 751.
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4. FERGUSON: Quoted by Copeland.
5. HERXHEIMER AND ROTH (1914): *Zeigler Beit.,* lviii, 319.
6. HARBITZ, F. (1909): *Arch. Int. Med.,* iii, 32.
7. KAUFMANN: *Pathology,* vol. iii (translated by Reiman), London (1929).
8. KAWASHIMA (1911): *Virch. Arch.,* cciii, 66.
9. MANASSE (1893): *Virch. Arch.,* cxxxiii, 391.
10. MASSON (a) (1932): *Amer. Jour. Path.,* viii, 367 and 389; (b) (1935): *Bull. Soc. franc. de Dermat.,* xlii, 1278.
11. ORZECHOWSKI AND NOWICKI (1912): *Seit. f. d. ges. Neur. u. Psych.,* xi, 237.
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13. VEROCAY (1910): *Zeigler Beit.,* xlviii, 1.
14. ZUZUKI (1909): *Berlin. Klin. Wochenschr.,* xxxvi, 1644.

Simmonds' Disease

By J. H. BIGGART,

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THE description by Paulesco in 1907 of the symptoms developing in dogs after extirpation of the anterior pituitary was followed in 1914 by Simmonds' now classic paper demonstrating the clinical picture in man which he correlated with destruction of the anterior pituitary by disease. In recent years such cases have attracted much attention, and over two hundred have been reported in the literature, of which few more than one-third have been verified by post-mortem study. In an extensive review in 1933, Silver found forty-one cases so confirmed, in 1936 Howard and Rhea mention a total of forty-seven proven cases, whilst in 1938 Lisser and Ascarilla collected sixty-nine. Sheehan (1939) calculates that in each ten thousand of the population there are about two severe and seven lesser examples of hypopituitarism, but post-morten findings have so far failed to reveal this high incidence. Unfortunately, in many recent papers some confusion has arisen between the clinical picture of Simmonds' Disease and anorexia nervosa, and it has become more than ever necessary to insist upon the post-mortem verification of the diagnosis whilst we await the development of some clinical observation or biological test which will more clearly separate these two conditions.

In view of the still relative rarity of confirmed cases, and in order that any

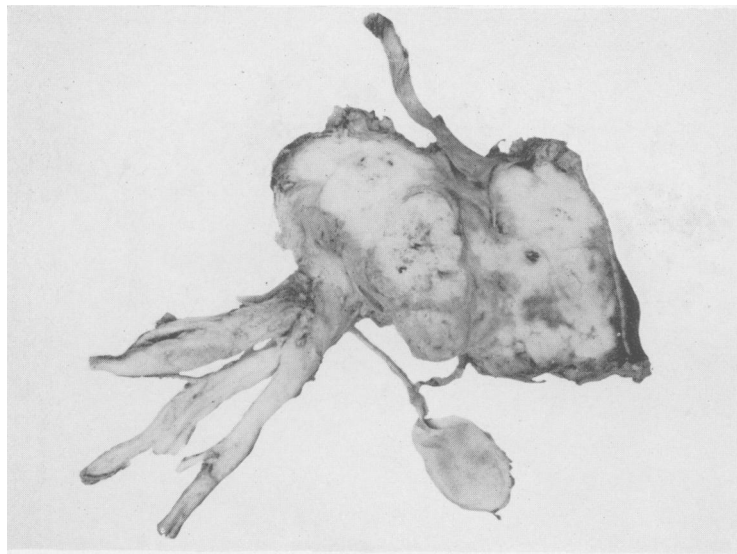

Fig. 1

Tumour of right supraclavicular region, showing involvement of brachial plexus. Note the thickening of the brachial plexus, and the swelling on the nerve to the Teres major. One-third natural size.

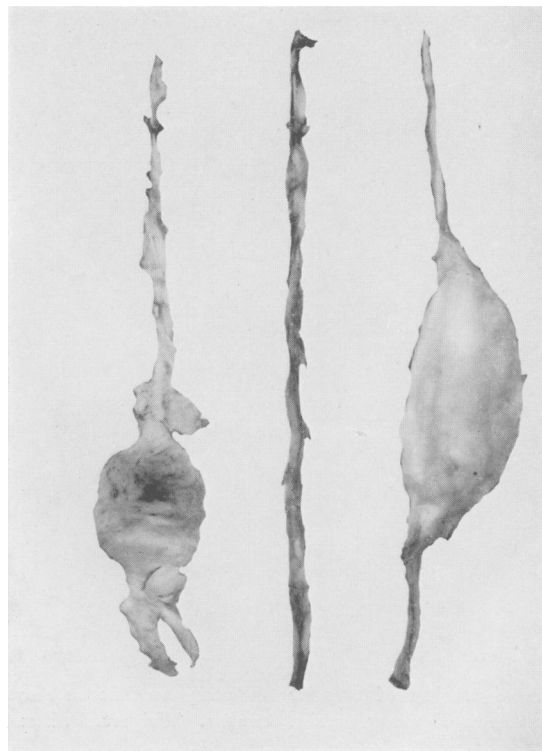

Fig. 2

To show the varied appearances found on the peripheral nerves. The illustration shows the right thoracic sympathetic chain, the ileo-inguinal nerve, and the left external peroneal nerve.

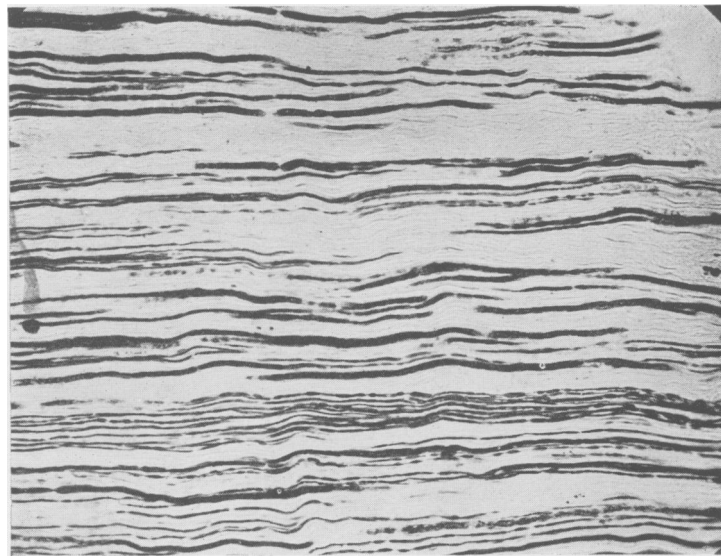

Fig. 3
Longtitudinal section of a nerve showing diffuse thickening. The myelin sheaths are separated by fibrous tissue lying parallel to the nerve fibres. Weigert. Pal. x 110.

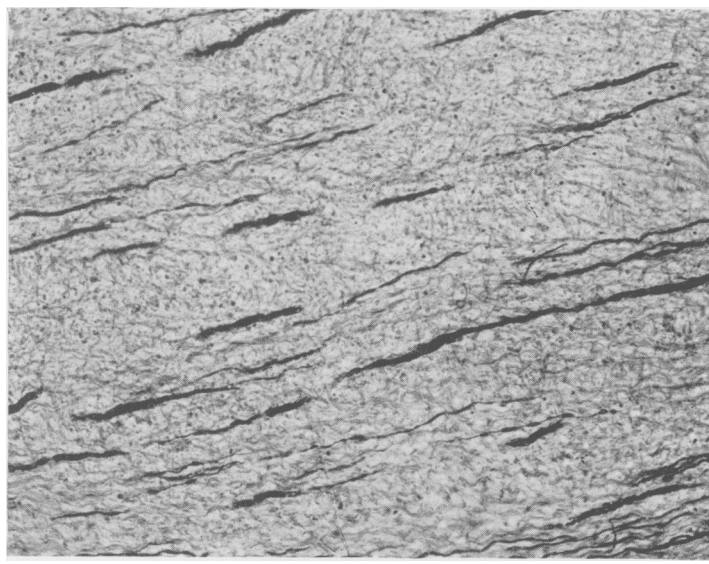

Fig. 4
Longtitudinal section of tumour on peripheral nerve showing the preservation of many myelin sheaths. Weigert. Pal. x 110.

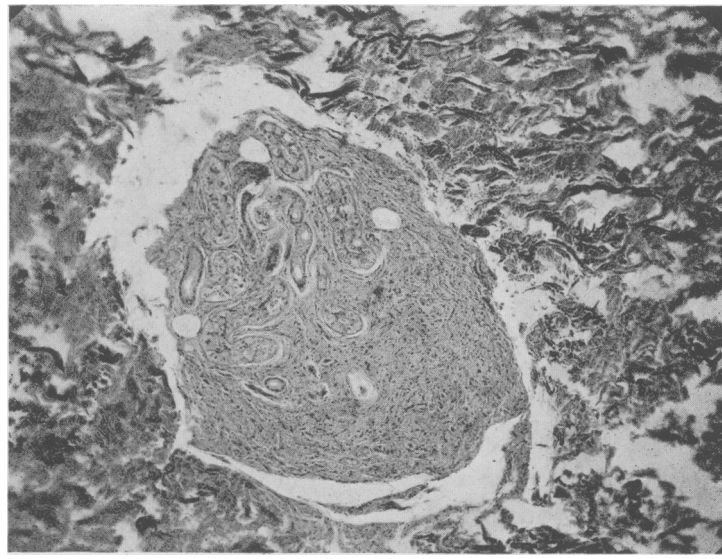

Fig. 5

Small neurofibromatous nodule surrounding sweat-glands. x 75.

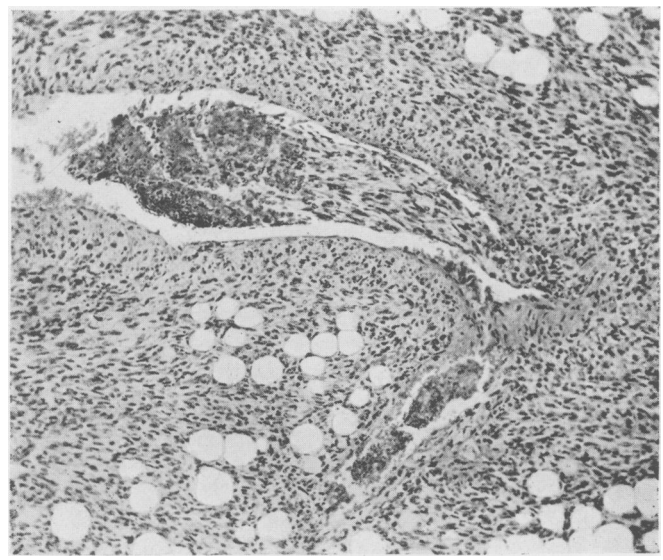

Fig. 6

Neurosarcoma. Section from right supra-clavicular tumour, showing invasion of a blood-vessel and adjacent fat. The tumour is composed of non-specific spindle cells.

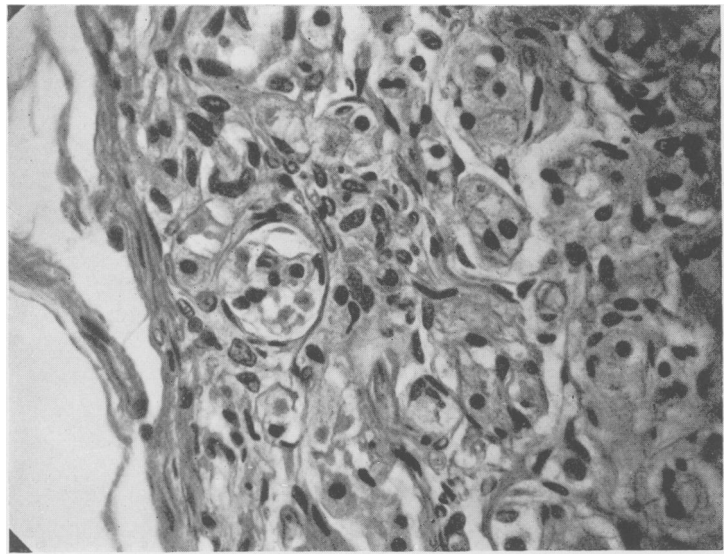

Fig. 7

Section from a cellular area from a nerve in the pancreas. Note the cellular proliferations in relation to individual nerve-fibres. x 600.

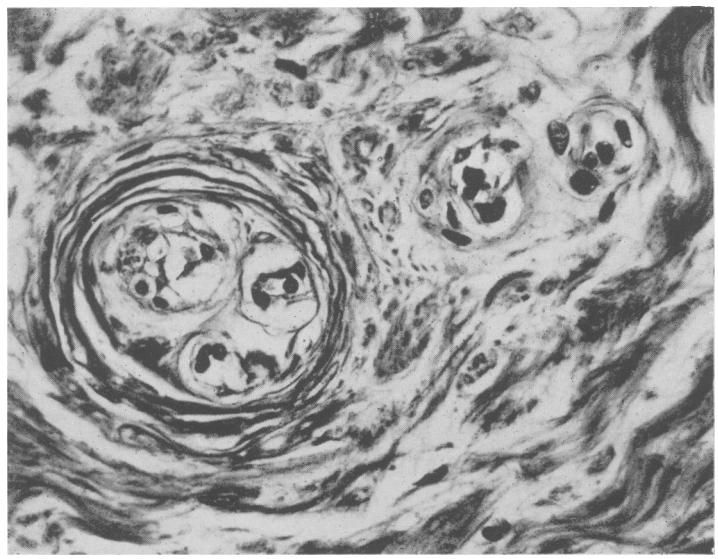

Fig. 8

Section from a tumour nodule, showing cellular proliferations in relation to surviving nerve-fibres. x 600.

SUMMARY.

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BIBLIOGRAPHY.

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9. MANASSE (1893): *Virch. Arch.,* cxxxiii, 391.
10. MASSON (a) (1932): *Amer. Jour. Path.,* viii, 367 and 389; (b) (1935): *Bull. Soc. franc. de Dermat.,* xlii, 1278.
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In view of the still relative rarity of confirmed cases, and in order that any

possible cases in Northern Ireland might be reviewed, the following cases seemed worthy of report.

CASE I.

The patient was a female aged 61 years. She had never been married, and had no children. Her menopause occurred at the age of 37 years. It was difficult to elucidate the menstrual history previous to this, but so far as it can be determined it was quite normal. Shortly after the menopause the patient noticed her skin becoming dry and scaly. She became nervous and subject to fits of depression and acute attacks of melancholia.

On admission to hospital she was found to be a thin, frail woman. Her skin was dry and scaly and had lost its elasticity. She had lost many of her teeth, and her hair was thin and dry. Her cardiac, respiratory, and urinary systems were normal. Her basal metabolic rate was −35. Blood-pressure was 126/80. Thyroid medication was given without effect. She developed broncho-pneumonia, which proved fatal.

At autopsy the cause of death was a confluent broncho-pneumonia. The following extracts from the autopsy report give the essential findings :—

Pituitary.—The sella turcica was enlarged to almost twice its natural size. The pituitary appeared greatly enlarged, measuring 2 cm. in antero-posterior diameter. On section, the gland was found to be replaced by a cyst, which occupied the site of both the anterior and posterior lobes. This cyst had a thin tough wall 1-2 mm. in diameter, and contained a glairy rather yellowish fluid. Histologically a few eosinophil cells embedded in the fibrous wall of the cyst near its anterior margin represented the anterior lobe. No posterior lobe tissue was found. The cyst was lined by a layer of columnar epithelium, which was ciliated. This epithelium rested upon a dense layer of fibrous tissue, which represented the destroyed glandular tissue. From the main cystic cavity, smaller epithelial lined diverticula projected into capsule. The pituitary stalk and its covering pars tuberalis were normal (fig. 1).

Thyroid.—This was small (weight 10 gm.). On section, the gland had a whitish appearance, and no colloid could be seen on macroscopical examination. Histologically there was a remarkable degree of atrophy. The stroma was infiltrated by lymphocytes (fig. 6).

Adrenals.—These were small, both together weighing 6.5 gm. On histological examination, the cortex was found to show a generalised atrophy. The medulla showed no definite change.

Ovaries and uterus.—Both ovaries and uterus were atrophic, but little emphasis can be placed on this finding owing to the age of the patient at the time of death.

The liver, spleen, and kidneys were reduced in size, but showed no lesions.

Summary.

Cyst of pituitary.
Atrophy of thyroid and adrenals.
Splanchnomicria.
Broncho-pneumonia.

CASE II.

The patient was a woman of 63 years. She was married at the age of 28 and

had two children, one at the age of 30, and the other at the age of 34. Both of these are alive and well. Following the birth of the second child, she never menstruated. There are no available details of the obstetric history in either confinement, but her sister states that at the second confinement the patient was dangerously ill and was attended by a consultant. About the age of 36 she began to suffer from fits of depression, to seek relief from these in alcohol and other drugs. Finally her mental symptoms became so marked that she was admitted to the asylum at the age of 40 years.

On admission, she was found to suffer from melancholia with remarkable outbursts of irritability. During these she swore violently, threw dishes, and had definite delusions of persecution. She appeared quite well nourished.

Two years after admission it was noted that she appeared somewhat myxœdematous, with dry scaly skin and some loss of hair. Thyroid medication had no effect. The basal metabolic rate was -30.

The urine showed albumen and a few casts. A few years before death she began to show some rigidity and tremor of both arms, and finally she was confined entirely to bed. Her blood-pressure at this time was 240/180. She died at the age of 63 from cardiac failure.

Autopsy.—The patient was a small, frail old woman with inelastic skin, complete absence of pubic and axillary hair, and thinning of the hair of the scalp. There was no abnormal pigmentation of the skin or mucous membranes. She was completely edentulous.

The chief findings were the following :—

Heart.—This was greatly enlarged. The endocardium was everywhere normal, and the enlargement was found to be due to hypertrophy of the left ventricle. There was marked atheroma of the coronary arteries and the aorta.

Kidneys.—These were reduced in size. The capsule stripped relatively easily, leaving a finely granular surface. On section, the cortex was found thinned. The cortical striæ were irregular and deformed by the presence of pale translucent areas of fibrous tissue.

Liver.—This was small, weighing only 2 lb. On section, it had a brownish tint. The lobules were small and indistinct. There was no cirrhosis. Bile-ducts and radicles of the portal vein were normal.

Spleen.—This weighed 3 oz. The capsule was thin. On section, no malpighian corpuscles could be distinguished. The trabeculæ were prominent.

Pancreas.—This was also reduced in size, and weighed only 40 gm. The ducts were patent, and on section the acinar tissue showed no abnormality.

Thyroid.—This was reduced in size and was hard and nodular. It weighed 14 gm. On section, a cyst, 2 cm. in diameter, was found in the upper pole of the right lobe. The remainder of the gland was pale, with much increase in the fibrous stroma.

Adrenals.—Together these weighed 5 gm. On section, the cortex was thin and lacking in lipoid.

Ovaries and uterus.—These were small and atrophic.

Pituitary.—This was small. On section, the reduction in size was found to be entirely due to destruction of the anterior lobe, which was cystic (fig. 3).

Brain.—There was a little fibrous thickening of the meninges, with some generalised atrophy of the convolutions. The basal vessels showed a marked degree of atheroma. Section of the fixed brain showed bilateral cystic infarcts in the basal ganglia, most marked in the globus pallidus.

Microscopical Examination.

Pituitary.—The posterior lobe, stalk, and pars tuberalis appeared normal. The anterior lobe had largely disappeared, being replaced by a cyst, into which projected several rounded knobs of tissue composed of anterior lobe cells. Over the upper surface of the gland there was a proliferative arachnoiditis.

Thyroid.—This showed a profuse generalised increase in fibrous tissue. The gland tissue was represented by groups of small acini, often without colloid, surrounded by collections of small lymphocytes.

Adrenals.—There was a marked degree of atrophy of the cortex, which showed numerous large fat spaces between the atrophic cells. The medulla showed no abnormality (fig. 7).

Kidneys.—Some of the glomeruli were replaced by hyaline fibrous tissue. Others showed fibrous adhesions between the glomerular tufts and Bowman's capsule. Others were atrophic. Many of the tubules were atrophic, while others appeared hypertrophic. There was a generalised increase in the fibrous tissue stroma.

Histological examination of the other organs showed atrophy of the parenchymatous tissue and arterio-sclerosis. The brain showed diffuse atrophy with reparative gliosis, which could be explained on the basis of the marked arterio-sclerosis.

Summary.

Cystic atrophy of the anterior pituitary.
Atrophy of all endocrine organs.
Absence of body hair.
Splanchnomicria.
Chronic glomerulo-nephritis.
Generalised arterio-sclerosis.
Cardiac hypertrophy.
Cerebral infarcts.

CASE III.

The patient, a woman of 51 years, was admitted to hospital as a case of myxœdema. She was married, and had one child born when she was forty years of age. The labour had been complicated by a retained placenta necessitating manual removal, and followed by a low-grade sepsis with a protracted convalescence. She had never menstruated subsequently. Following the pregnancy she became apathetic, and had gained in weight.

Examination showed a patient who looked older than her years. She had a myxœdematous appearance, with thinning of the hair of her scalp and absence of axillary hair. Her blood-pressure was 135/75. Basal metabolic rate was -30. Her teeth were bad, and during her stay in hospital she developed an abscess of the gum, which spread to her neck, and she finally died from broncho-pneumonia.

Autopsy.

Apart from the signs of toxic involvement of the organs, the following changes were found :—

Pituitary.—This was represented by a small greyish mass in the sella turcica. The stalk was normal, but it was impossible to define clearly the anterior and posterior lobes. On histological examination, the anterior pituitary was found almost completely replaced by fibrous tissue, only a few cells of anterior lobe tissue being found along the upper margin of the gland. The posterior lobe, though small, was not actually involved in the sclerotic process. The pars tuberalis could be seen on the anterior margin of the stalk.

Adrenals.—These were small, both together weighing only 5 gm. On histological examination, the cortex was found to be very atrophic, with large fat spaces replacing many of the cells. No lesion could be appreciated in the medulla.

Thyroid.—This was very atrophic. Microscopically, small atrophic acini were found lying in a stroma of fibrous tissue, which was densely infiltrated by lymphocytes.

The other organs showed some degree of atrophy.

Commentary.

In any extensive review of the reported cases of Simmonds' Disease some difficulties in its diagnosis become apparent. Either, as more especially in the continental literature, it is diagnosed much more frequently than is justified on post-mortem examination, or, as in the present three cases, the diagnosis is made in retrospect following the post-morten. It becomes obvious that in spite of the many recent reviews (Calder, Sliver, Howard and Rhea, Meyer, Lisser and Ascarilla), present criteria for its successful diagnosis may be insufficient.

The accepted clinical picture is loss of weight, usually stated to be a marked degree of emaciation, amenorrhœa in the female, loss of libido and potency in the male, asthenia, and a lowered basal metabolic rate. With these symptoms there is often an appearance of premature senility, loss of axillary and pubic hair, and not uncommonly some degree of mental change.

The condition most frequently confused with Simmonds' Disease is anorexia nervosa, and it is apparent from the literature that the latter is seen much more frequently. Simmonds' Disease is most frequently seen in women, but in women in the fourth, fifth, and sixth decades, who have previously borne children. Anorexia nervosa also occurs most frequently in women (ninety per cent., Ryle), but usually in girls or women below the age of thirty, who are unmarried and in whom the history of onset is not related to child-birth, but rather to some psychological stress.

The patient with Simmonds' Disease shows thinning and loss of axillary and pubic hair. In anorexia nervosa the female patients may show the development of the male distribution of pubic hair, whilst a strong growth of downy hair may be more widely scattered on the trunk and limbs. However, some thinning of the body hair is also occasionally seen, and minor degrees of change are probably of little or no value in the differential diagnosis.

Some degree of mental change is quite common in Simmonds' Disease, and is usually shown by listlessness, apathy, or even melancholia. Manic depressive episodes have also been described. Gull noted the remarkable energy often shown by the patient with anorexia nervosa, but once again there is much individual variation.

The response of the patient to treatment is perhaps the most useful diagnostic feature of the case of anorexia nervosa.

Finally, the patient with Simmonds' Disease need not show emaciation. In two of the present cases the patient had at death, or had shown previously, the clinical appearance of myxœdema which may well have prevented the consideration of Simmonds' Disease in the differential diagnosis. This fact, that the patient with destruction of the anterior pituitary may present the appearance of myxœdema, is not apparently generally realised. It has recently been stressed by Sheehan. In 1939, Castleman and Hertz report the case of a woman aged 48. When 38 years of age she had a miscarriage of five months in her first pregnancy. Within the next year she had a sudden onset of amenorrhœa. Her skin grew progressively drier and coarser. There was gradual loss of strength and enterprise and the development of a placid disposition. She continued to appear well nourished. Her blood-pressure was 160/100 mm. Hg., and basal metabolic rate 28. The clinical diagnosis was myxœdema. At post-mortem she was found to show fibrosis of the anterior pituitary with atrophy of the other endocrine glands. These authors have collected six similar cases from the literature, and the present Case III represents another example where the clinical picture of myxœdema has developed in association with fibrosis of the anterior pituitary. It is interesting that Case II at the time of her admission to hospital presented a similar myxœdematous appearance. Sheehan in his review of the literature states that a severe degree of emaciation is quite uncommon, and the present cases tend to support his contention. Where the presence of emaciation ceases to be the stimulus to the diagnosis, it is probable that more cases of Simmonds' Disease will be diagnosed ante-mortem, and that less confusion will arise with anorexia nervosa.

The pathogenesis of the disease is still not clear. As in the present Case I, the cause may be the destruction of the pituitary by a tumour, cyst, or granuloma. Most frequently the disease develops subsequent to child-birth. Sheehan, from an analysis of the cases of post-partum necrosis of the pituitary, believes that this lesion is the result of the effects of the shock due to hæmorrhage. Most of his cases have had a labour complicated by retained placenta, and it may be that the resulting low pressure allows thrombosis to occur in the engorged sinusoids of the pregnancy gland. However, whilst we agree that acute post-partum necrosis of the pituitary does occur in association with retained placenta and hæmorrhage, we are not convinced that the mechanism is so straighforward. Many patients die within a week after loss of blood and degrees of shock quite comparable in their severity to those seen in complicated deliveries. Yet necrosis of the pituitary has not been seen in such patients. Even in severe shock an attempt is made to maintain the blood-supply to the vital centres, and the pituitary must surely benefit

in this respect from its anatomical propinquity to the central nervous system. If the blood-supply is so poor as to permit intra-vascular clotting, this is therefore most likely to occur in other organs.

Examples of acute necrosis of the corpus luteum have also been occasionally encountered in the absence of any lesion of the pituitary. These have been associated with eclampsia.

It seems to us, therefore, that the shock theory of Sheehan does not adequately explain the mechanisms of post-partum necrosis of the pituitary. Perhaps the placenta may play a more important rôle than has so far been assigned to it. The fact that it is an endocrine gland which becomes necrotic is of some importance. Large doses of hormones have produced, experimentally, necrosis. Thus Severinghaus states that large doses of œstrin may produce small necroses in the anterior pituitary. Large doses of thyrotrophic hormone have been seen to produce large areas of necrosis in the liver. It is thus possible that the pituitary necrosis may have an endocrine origin.

The retained placenta, once the fœtus has become separated, may discharge its contained hormones into the circulation, and these in turn may act upon the anterior pituitary. Such an explanation is as equally logical as Sheehan's shock hypothesis from the facts which he has presented, and in addition would allow us to understand more easily the limitation of the complication to pregnancy. A quantitative estimation of the hormones circulating in the blood during the time of retention of the placenta would therefore be of interest in confirming or disproving this hypothesis.

The present three cases differ little in their clinical picture from those reported in the literature. Mental symptoms of some degree have been recorded in about fifty per cent. of the cases. There is, however, no standardised picture, and Wadsworth and McKeon point out that some of the mental symptoms may be due to the associated hypoglycæmia, whilst the common apathy, somnolence, depression, and slowing of mental processes may be pituitary in origin. In other patients the mental picture may well be due to some concomitant mental illness.

References.

Calder, R. M. : *Bull. Johns Hopkins,* 1932, l, 87.

Silver : *Arch. Int. Med.,* 1933, li, 175.

Howard, C. P. and Rhea, L. J. : *Internat. Clinics Philad.,* 1936.

Meyer, W. C. : *Deutsch. Arch. f. kein. Med.,* 1938, clxxxii, 351.

Lisser, H. and Ascarilla, R. F. : *Fr. A. Am. Physicians,* 1938, xxxii, 735.

Sheehan, H. L. : *Jour. Path. and Bact.,* 1937, xlv, 189.

Sheehan, H. L. : *Quart. Jour. Med.* (New Series), 1939, vol. viii, 277.

Castleman, B. and Hertz, S. : *Arch. Path.,* 1939, xxvii, 69.

Severinghaus, A. E. : "The Pituitary Gland," *Assoc. Research. Nerv. and Mental Dis.,* 1938, vol. xvii, p. 103.

Wadsworth and McKeon : *Arch. Neur. and Psych.,* 1941, xlvi, 277.

Simmonds, M. : *Deutsche Med. Wchnschr.,* 1914, xl, 322.

Paulesco, N. C. : *L. hypophyse du Cervaeu,* Paris Vigot Freres, 1908.

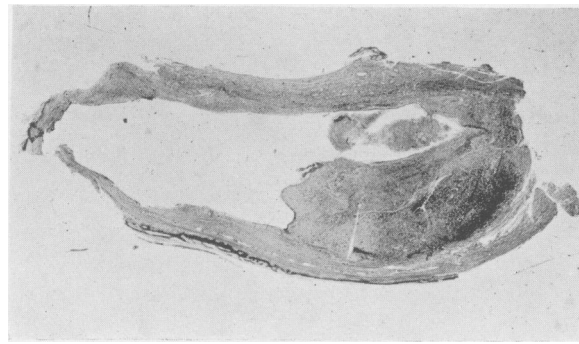

Fig. 1

Case 1: Antero-posterior section of pituitary gland, showing a cyst replacing both anterior and posterior lobes.

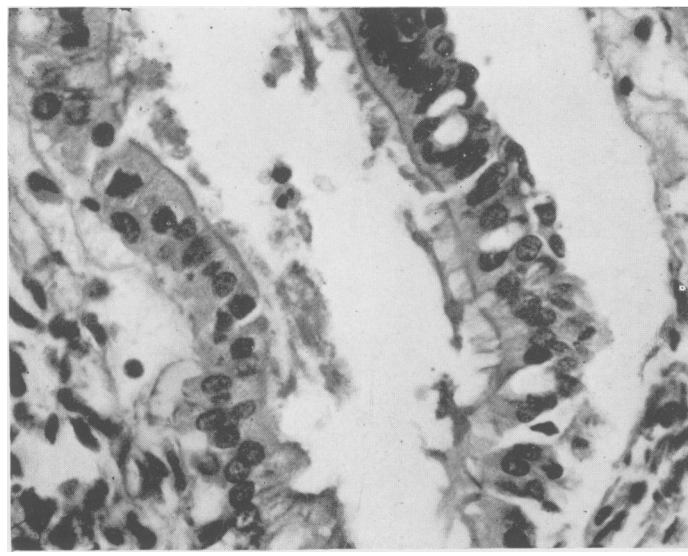

Fig. 2

Case 1: The section shows that the cyst is lined by high columnar ciliated epithelium.

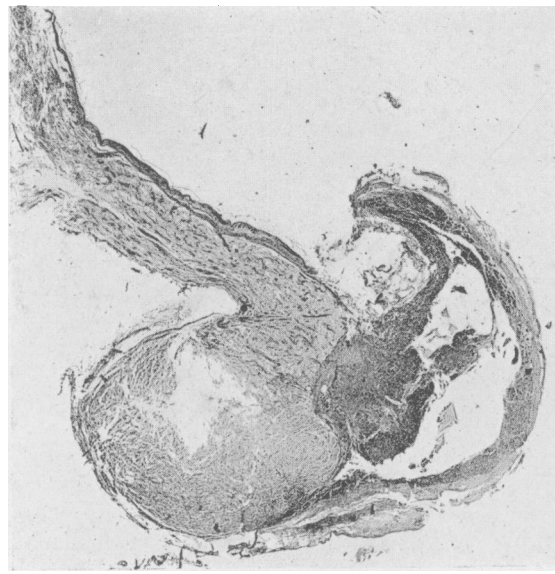

Fig. 3

Case 2: Section of pituitary gland, showing the cystic degeneration of the anterior lobe. The pars tuberalis and pars nervosa are normal.

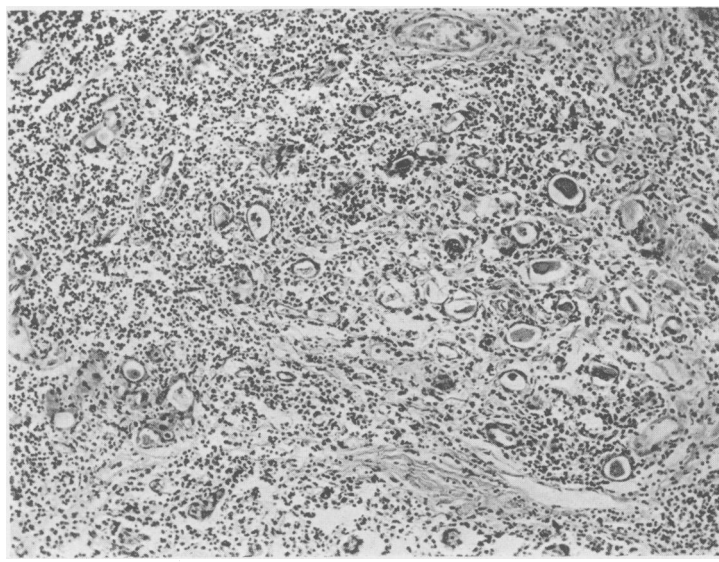

Fig. 4

Case 2: Section of thyroid to show the extreme degree of atrophy and the diffuse infiltration with lymphocytes. The thyroid in cases 1 and 3 is similar.

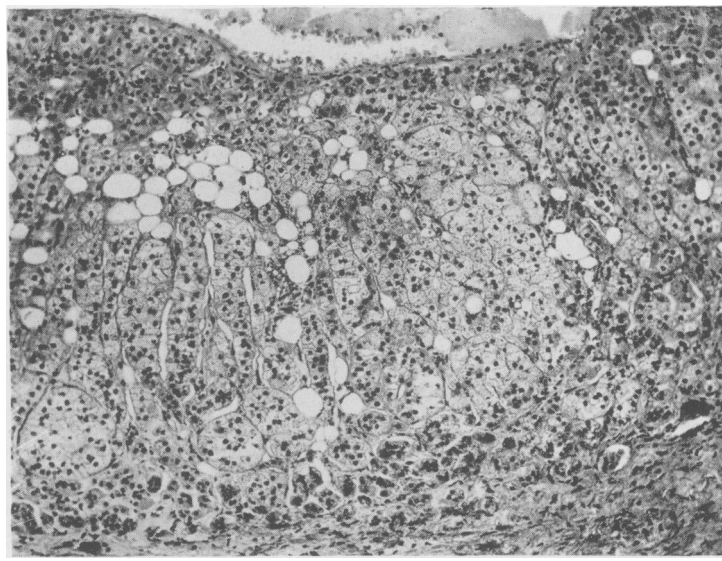

Fig. 5

Case 2: Section shows the atrophy of the adrenal cortex and its partial replacement by adipose tissue.

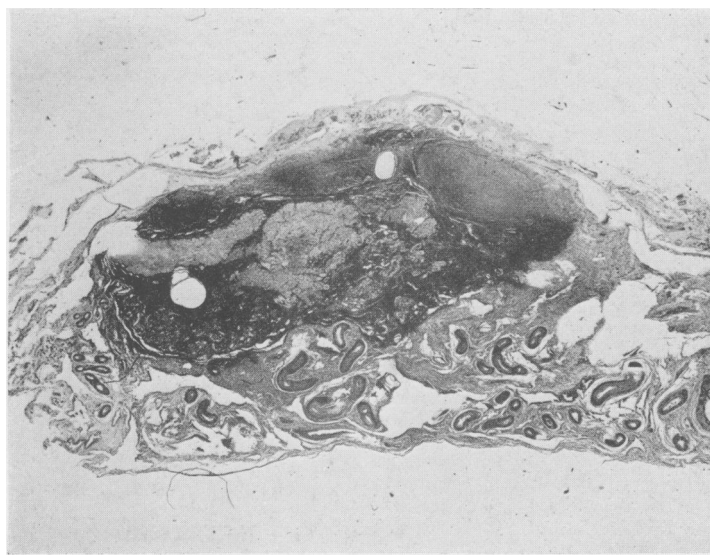

Fig. 6

Case 3: To show the ovarian atrophy. x 8.

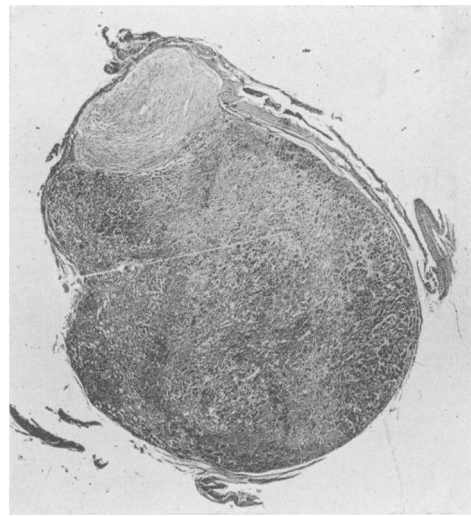

Fig. 7

Section of pituitary gland from a patient who died a week after delivery. The section shows necrosis of over two-thirds of the anterior lobe.

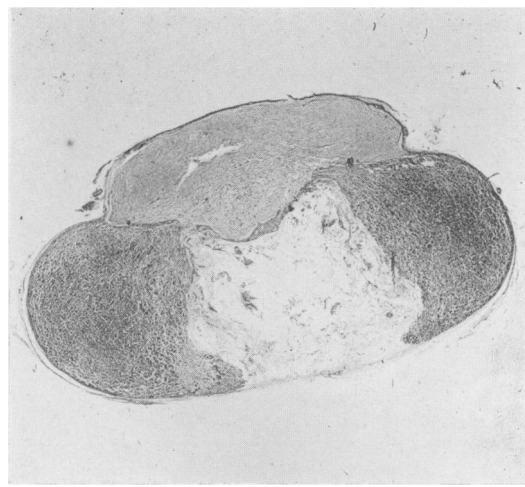

Fig. 8

Section of pituitary gland from a woman aged 37 who died two years after a complicated child-birth, showing partial cystic destruction of anterior lobe with no clinical symptoms of Simmonds' disease.

The Child, the Community, and the General Practitioner

By CHARLOTTE EMMA WARNER, M.D.

THE day is past when the ruling idea of public health administration was dumps, drains, and germs, or even food, water, and housing. More and more, preventive medicine stresses individual resistance as the key to community protection. The maintenance and improvement of environment take their places as means to an end, and that end individual good health. It is clear from the writings of the great British and American public health administrators that they aim at no less than the handing over to the community of every individual, at the age when he is expected to take part in its activities, as a whole man, a sound mind in a sound body, a unit in that city of bone, which the old Irish, justly, as is seen to-day, considered stronger than one of stone.

If the whole man is the end, he is no less the beginning of public health organization. Institutions, like machines, are made by men, and without men in charge of them, are spoil for time or a more malicious enemy. "For men," said a Greek, using the word connoting men of vigour, "and not mere forts and ships, are the stuff of a nation."

Now, physiologist and psychologist agree at least in this, that power to influence the health or wholeness of a human being varies inversely with his years, and wanes more swiftly than they advance. It is hardly too much to state that a man can be marred, if not made, in the few years between conception and the start of school education. Read, for example, the laments recurring in report after report of school medical officers in Great Britain, over the "damaged goods"—irretrievably damaged goods—which, as school entrants, year by year reach their care.

On this showing, the potential or basic or essential health of a community is the sum of the health of its individual mothers and young children. Responsibility for the medical care of it is, in Great Britain, shared by all three divisions of the profession, viz. : general practice, the public health service, and consultant practice, which, whether or not it is formally associated with a medical school appointment is, in effect, teaching practice.

Is this care giving the best results possible, even within the present social and economic framework of society?

A negative answer, so far as Great Britain is concerned, has lately been given to this question in a review of it by a highly competent authority. In the "Lancet" issues of 4th, 11th, 18th May, and 1st June last, appeared a serial contribution under the title : "Death in the First Month and the First Year." The author is Charles McNeil, Edward Clark professor in the University of Edinburgh, of *not,* be it noted, diseases of children, but of child life and health. None of the three branches of the profession escapes Dr. McNeil's criticism, but in the main that stresses not so much shortcomings of any, as lack of contact between all three.

Much of the detail of his case and more of his suggestions for reform are inapplicable in our area. But neither our statistics nor Dr. Kidd's review, published

in the "British Medical Journal" and in the Journal of this Society, of our public health conditions would justify denial of the substance of the charge—lack of continuity in the care of young children, with resulting damage to the adult citizen and therefore to the public health.

If this be accepted as true, it is less excusable than across the channel, where childhood is so often cut off from green fields or seashore by leagues of street, and from sunlight by industrial smoke. Here, too, contacts between doctors in different branches of the profession and at different levels in each are many and friendly, and there is still such a survival as the family practitioner, to whom the heredity and environment, or, as it is called in South Down, "seed, breed, and way of doing," of every family in his district may be matter of personal knowledge. The argument cuts both ways. The simpler conditions that make the defect less excusable make it correspondingly easier to remedy. For instance, only one of our incoherent public health services, viz., the dispensary service, concerns itself with the country child of pre-school age, and that service is part-time by general practitioners. The obvious remedy is improvement of contact between teaching and general practice, which in turn could not fail to hasten reform of dispensary practice.

The object of this paper is to show that experience, which there is no reason to think is peculiar to one practice, supports the charge that the North of Ireland child does suffer from lack of continuity in medical care, and further, that improvement can begin, not in some problematic post-war future, but at any time from now on.

(1) In the case of the woman with child, the principle of routine supervision of the presumably healthy, as against ad hoc attendance on the sick, is established. More or less ante-natal care is the rule in most private practices. Even if the woman is to be transferred for her confinement to another practice, some previous contact is made between the two doctors concerned. It is when the infant is two or three weeks old that the first break occurs, Dr. McNeil points out in how critical a period. The mother has just left the shelter of clinic or best bedroom, and begun to cope with domestic work and worry. It is in the child's first month of post-natal life that neo-natal effects, i.e., congenital and inherited tendencies and the carry-over from intra-natal stress, are at their height.

Here is an example of what may happen to him and his mother—for they are still, though in lessening degree, a unit—at this time. A mother, hitherto known only by sight or not at all, comes to the doctor with an ailing baby. She may have moved into the district since his birth, or she may have married into it and returned to her mother's house and the care of a medical relative or of her childhood's doctor for her confinement, or she may have attended a consultant or clinic for ante-natal advice, and later been delivered as an in-patient. In any case, according to her own account and the objective evidence of the baby's condition, she has left the care of one doctor without having impressed on her the necessity for keeping her child under observation by any other nearer at hand. She has not even understood that a feed which was a feast at ten days is a famine at six weeks. She is sometimes not even sure of the baby's birth-weight. Asked what kind of a "time" she had,

she often says she cannot remember, "as she was under chloroform." Even had she not been, her answer as an indication of the degree of birth-stress suffered by the child, would be untrustworthy, for it is in non-technical terms, and coloured by temperament. In the circumstances, treatment inevitably resolves itself into alteration of a regime prescribed elsewhere. The mother demurs: "But Dr. A. (who delivered her) is such a good doctor." All that she retails of his care of her, and perhaps independent knowledge as well, justifies her opinion. So Dr. B. starts treatment under a handicap, and, more important, so does the baby, without any further advantage to Dr. A. Everybody loses at this game.

It would be a simple matter to bridge this first gap. Already it is the custom of city maternity hospitals to refer city children born in their practice to the care of welfare clinics run by the hospital or by the local authority. The least that any hospital or obstetrician should do is to give every mother, on parting from her, written directions, not merely for the feed in use at the time, but for systematic increase in strength, and also the strongest and least mistakable warning against otherwise altering or adding to it, or, in the case of breast-feeding, against weaning on any but medical advice. But better still would be to impress on her the necessity of choosing immediately some doctor in her own neighbourhood and putting her child in his care, and with that end to hand her a card addressed to Dr. Dash, and containing a brief history—something like this: "Mrs. John Smith, primipara delivered by Dr. A., in the Blank Clinic, 1st October, 1940. Pregnancy normal. Duration of labour, 26 hours. Delivery by easy forceps. Puerperium normal. Male child in good condition at birth. Weight, 8 lb.; on discharge 8½ lb. Feed on discharge, breast. Condition on discharge, both satisfactory."

(2) But one and the same doctor may have been in charge throughout, and yet the child be no better off later. Dr. C., having, with all the judgment and skill to be expected from a pupil of an Irish midwifery school, delivered a living mother of a living child, may put the latter on the bed and never look at it again, except at its arm when he vaccinates it, till the stormy night when he is hauled out of bed to treat it for larnygismus, or the chastening day when he meets it at a children's party, with a developing squint, or bowing of the tibiæ. The mother who is a primipara is to-day in the majority. Even when she is not, there is often a gap between births long enough to let her forget how she brought up her first. She is in constant need of advice, and, failing instructions from her doctor to bring her child for inspection at stated intervals, she hesitates to trouble him, and seeks it elsewhere. If she is well-off, her adviser may be her trained children's nurse, impressively badged and uniformed, and liable to make the baby, like herself, the victim of the "system" under which she was trained, and any modification of which has come in the course of years to seem a sacrilege. The intelligentsia surround themselves with hand-books and are muddled by multitude of systems, and the poor by the multitudinous want of system got by consulting one "neighbour woman" after another. But the chief guide and friend of the country mother is the district midwife. Of all public servants she is the most devoted and self-sacrificing, and the most evilly treated. She has recently had a short period of post-graduate

pædiatric training, but she is not often a fully-trained nurse, nor, any more than the average nurse outside the children's hospitals, has she the root of the matter in her. This root is realisation of the overwhelming importance in infant health of feeding and the part, so negligible as to be nil, played by drugs, including aperients, in the treatment even of the sick infant. The nurse, with this root in her, will not prescribe or materially alter a feeding-regime without medical advice. Dr. McNeil has something to say of the training of nurses. But in our area, the nurse's practice depends, apart from qualities personal to herself, on her original training and on the practice of the doctors for whom she works. As the standard of medical care of children rises, so will hers, and it is to be hoped her conditions with it.

Equally out of touch with medical control are the child-welfare schemes, at any rate those of which the writer has personal knowledge, run in some country towns in connection with district nursing associations, which thereby benefit by grants from public funds.

If all these are blind guides, the part of open-eyed decoy is played by wholesale advertisement of patent foods. As generally used, they are at best an expensive substitution of starch for starch, at worst, a harmful addition of starch to starch, in the weanling's diet, which, even under supervision, is apt to contain too much. Bread, potatoes, and wheat or oatmeal porridge give all the starch needed, have other food values, and are balanced by the butter, milk, or other fat which it is custom to give with them. They are home-grown. There are also social and psychological reasons in their favour as part of the household menu. The child who gets "the run of the house," if the house is a good one, does well. The weanling foal or lamb eats grass, puppies and kittens the leavings from their mothers' dinner, getting thereby not only a balanced ration, but also some social virtues.

A composite fee is the custom for the care of pregnancy, labour, and puerperium. The offer of one in return for routine care of the child would therefore not be without precedent. Every general practitioner who delivers a child should look at it, naked as it was born, at increasing intervals during its pre-school years, certainly till primary dentition is complete. The child of healthy stock, whose feeding and management is under medical care till that age, as a rule troubles the family doctor little afterwards, apart from accident and the exanthemata. Every argument in favour of ante-natal care, except the public attention concentrated on that, holds good for post-natal. It gives better results at less cost to interview a jolly baby at convenient times and places than to treat a peevish one for eczema, recurrent respiratory disease, or any other of the illnesses associated with unwise feeding or management. The child who falls victim to inevitable accident or illness is less likely to resent familiarity from a doctor he knows as a play-fellow. A general practice comes to include its own child-welfare clinic, where even those who were students when the stress was more on disease than on health, and less on the infant than on the older child, become familiar with the wide variations of the normal, and its ill-defined passage into the pathological. It was to meet this need for continuous care of normal childhood that the British Child-Welfare Service came into being. Grim indeed were the conditions of child life in great cities at the

beginning of the century. Statistics give little idea of their horror, nor of the corresponding conditions of industrial private practice, and of the ineptitude of English pædriatric teaching at the time. It was natural, but deplorable, that the State went outside the ranks of the general practitioners for the staffing of the new clinics; natural, but deplorable, that the general practitioners opposed their introduction as yet another "encroachment." The history of the British Welfare Service is a warning. Child-welfare is still a blank sheet in front of the general practitioner in the rural north of Ireland. Self-interest, no less than the community's, would make him endorse it as his own, fulfilling his proper function as doctor and physician. A doctor is by derivation a teacher a physician a naturalist, and a student of nature. A literal translation of the words into action is the one barrier against "encroachment" by the quack and the official.

(3) Dr. McNeil points to the third gap, viz., that between teaching and general practice, in the following passage: "We cannot hope to change our climate, and it will not be easy to rebuild our houses, but we can do much to improve our domestic habits, and, above all else, we have doctors and nurses, who, *at present untrained and incompetent in this early problem of dietetics, can be taught to master it,* and to establish good digestion and nutrition in the first month, and so provide the strongest safeguard against infection."

The sting in this gets home to both general practitioners and teachers, but the words "can be taught" seem to hold out hope for both. Actually, Dr. McNeil later seems to throw over the general practitioner as past all but praying for, for all his suggestions for reform apply to doctors in training rather than those already in practice.

But it is precisely with these latter that this paper is solely concerned. There are three ways by which they can keep in touch with teaching, viz., through consultations, private and hospital; through medical societies and the journals, and by formal post-graduate courses.

It has been shown above that infant-feeding is apt to be taken for granted as a natural process—a large assumption in present semi-civilisation—and therefore not calling for medical attention. When difficulties arise in the course of it, and the family doctor *is* called in, the same assumption lowers the number of consultations asked for by him, when these difficulties prove obstinate. An indication of this is the experience that it is less often the family doctor than the distracted parents who refer affected children to hospital or another doctor. Yet, to tell a mother that the quality, as distinct from the quantity, of her milk does not suit her baby and therefore she must wean, or to accept, without patient trial, the report that cow's milk cannot be digested, are surely as serious statements as to tell an adult with apparent anæmia that he is suffering from the pernicious form, or that he has diabetes, because his urine contains sugar. An immediate and marked improvement in contact between teacher and practitioner would result if neither of these decisions were ever made hastily, or finally without specialist advice. This lack of personal contact is all the more regrettable because a number—perhaps the greater—of practitioners are of the make of St. Thomas in this, that they can believe, but not without being

made to see. They learn more readily from the spoken than the written word, from the seen than the quoted example. Medical meetings and the journals therefore count for little in their post-graduate education. Even if these counted for more, it is questionable whether they give to the young child all the space his importance in the family and the community deserves. Of the space granted, perhaps the pure scientist might deign to give more. The scientist is apt to sigh over the general practitioner, or spit at him, like the great epidemiologist who not long ago told us it was possible to be honest or a general practitioner, and the latter to yawn in retaliation. Yet they stand or could stand to each other as the zoologist or archæologist to the field naturalist. The physiologist, for example, is content to give the caloric and biochemical requirements for normal growth and nutrition, leaving the details of administration to the practitioner. The latter is then gloriously free to check the scientist's basic data by observation of normal children, and the last shout in a scientific argument is left to the human baby.

The Northern Ireland Medical Benefits Scheme set an example, later followed elsewhere, by providing post-graduate courses for panel doctors. Since the first of these was given, a whole generation of children have arrived at school. Many of them have prematurely decayed primary teeth. Their permanent teeth—already damaged—are on the point of eruption. The owners of them will in time come on the panel. Later, much money will be spent by them, the state, and the approved societies on artificial dentures. On this score alone, would it not have been a sound investment to have devoted at least one lecture in every course to infant nutrition and feeding? Incidentally, when these unnecessarily and irretrievably damaged citizens come on the panel as fourteen-year-old juvenile insured, their mouths will have been made at least clean by the work of the School Dental Service. How long will it be before some effort by bonus, penalty, or propaganda, is made to keep them clean?

The exact nature of the damage resulting from lack of continuity in care can be stated shortly. In far and away the greater number of cases it is frank starvation, quantitative in the earlier, qualitative possibly quantitative also, in the later mouths. This is regardless of the symptoms of which the mother complains, and also of her social class. The diagnosis is fortified by the easiest of therapeutic tests—full feeding. Let the physiologist explain why one starved infant gets diarrhœa, i.e., frequent small green stools, and another constipation, and the psychologist why one fades away unprotestingly, and the other cries almost continuously. The North of Ireland baby belongs in general to the latter class. Feeding according to schedules sent out with patent foods, as published in text-books, or taught in some clinics across the channel, fails to quiet him. As often as not, he comes yelling from the maternity clinic. He raises a number of questions, of which the least far-reaching is this: is the four-hourly feed of so many ounces of such a dilution as sound a standard during as at the end of the first month?

A young mother with a small and lively first-born son tried in vain to have this regime altered. The baby cried continuously. In order to allow the father to sleep, mother and baby went off to the back of the mountains to stay with an elderly

medical relative. The old man heard the story, from baby as well as mother, looked at the former, and then said: "Woman dear, give him his fill when he wants it." This she did, and some weeks later took a rapidly-improving baby home to his father. Some time later again, the latter rang up his mother's doctor to ask if it was quite natural for a two-months-old baby to sleep all night without crying. Some people are never satisfied.

McNeil says the artificially-fed child should be having whole milk at two months; Findlay, when at Glasgow, gave it from birth, and so, when the mother is unable to nurse, does many a South Down grandmother, with none but good results. Yet the feeding out of which came the following story is not uncommon. A girl at sixteen months showed bossed frontal bones, open fontanelle, rickety rosary, and markedly enlarged wrist epiphyses. She and her parents were of the large and generous build. She had been fed till the tenth month on milk dilutions with malted sugar and some orange juice. The rachitis was probably healed, as she was seen at the end of the summer and had been latterly on more generous diet. She was suffering from what appeared to be a popular eczema of the face. The mother, as usual in such cases, said: "I was so afraid of giving her too much." The answer, as usual, was "Only God and the baby know when it has had enough." "God may," said the father, "but that hussy never does." Fathers, being on the touch-line, often see more of the game. A surprisingly large volume may be taken, and taken greedily, when the feed is poor in quality—a common cause of vomiting in the small thin infant on too dilute a feed.

It is easy to memorise or keep a note of calories required per pound weight (50-70), the caloric value of milk (about 400 to the pint), and of sugar (about 15 to the dram). The necessary food constituents are known. What is more important to know by heart than weight-age scales or feeding schedules, is the sound, look, and behaviour of the well- and of the ill-fed baby, such natural phenomena as the deep sleep of the one, the light of the other, the vomiting of the lusty feeder at the over-flowing breast, the exhaustion or peevishness of the still unfed, tired of sucking at a breast fat but not full, or at a teat with too-small pores. This is lore such as no pædiatrician can teach, but every practitioner learns for himself. But these consultants have their uses. They sometimes save experience from being altogether too "dear-bought wit."

The whole man is by definition a sound mind in a sound body, and then something to him. Education at any age is through the functions most active at that age, sex-appeal at fifteen years, for instance, and food-appeal at so many months. There is the child who at that age tells its mother it will not, or cheats her into believing it cannot take milk. If it is a girl, she is liable to become one of those who come home just before it; if it is a boy, he is starting on the road to delinquency. The doctor who has failed to debunk him, shares the responsibility. If some of our magistrates and more of our anonymous writers to the press get their way, and we our deserts, who is to escape the whipping?

Since the greater number of country children are in the nominal care of the dispensary service, something has to be said about it. Neither ante-natal care of

mother and child, as accepted already, nor post-natal, as urged in this paper, is possible in the present conditions of the service, questionably even in outline, by the keenest youngster, in the least-crowded dispensary. That in itself is condemnation of the conditions. Reform should not wait on wholesale reorganisation of all the public health services. It has been suggested above that a raised standard of child-care outside the dispensary can compel reform within. Social inequalities under "plutocracy" are no longer in the background of working-class consciousness. To paraphrase an old joke—wise men will mend the dispensary system, or sorry men will be made to. But we, who have known as our colleagues or our medical attendants men like the Cromies of Clough or Warnock of Donegal, are open to a finer argument. We can plan no memorial worthier of them, no heritage more welcome to their young successors than a commission to build where they were left to patch.

In conclusion, it has not been possible in the time at disposal, to qualify as many statements or forestall as many objections as desirable. Moreover, the nature of the subject has forced concentration on the damaged minority to the exclusion of the sturdy majority. But however faultily it has been put, the question still stands: Why any damage? Nothing human reaches perfection; but why, of all damages, in a land which still enjoys a "roughness of plenty," need the chief damage be starvation?

References.

1. Douglas Hubble, Derbyshire Hospital for Sick Children, *Lancet* (October 12, 1940): "Management of baby must be direct concern of doctor; it should not be left to nurse."
2. *Lancet* (October 8, 1938), "Australasian News": "League of Nations statement that defects due to improper feeding in early life often irreparable."
3. Dr. F. M. Spencer, *New Zealand Med. Jour.* (August, 1938): "Most common fault in artificial feeding use of too dilute mixtures."
4. Dr. Kidd, *Infant Mortality Rates* (1934): Northern Ireland 70, England 59, Eire 63; *Registrar-General's Returns*: Northern Ireland (average 7 years to the end of 1939) 76; Professor McNeil (1937): Scotland 80, Glasgow 104, England and Wales 58, Greater London 54, France 65, Germany 64, Sweden 45, Göteberg 31, Holland 38, Amsterdam 30, U.S.A. 54, Australia 39, New Zealand 32.
5. Professor McNeil, *Lancet* (May 11, 1940): "Fact of our high infant death-rates cannot be questioned. It is also matter of fact, not opinion, that these high figures are largely due to a continued steady loss of babes after the first month. The explanation is offered that this is largely due to mismanagement of early feeding."
6. Spencer of Newcastle-on-Tyne, B.M.A. meeting, 1938.

The Treatment of War Injuries

By A Panel of Military and Civil Surgeons

TREATMENT OF FACIO-MAXILLARY INJURIES

I. LIFE-SAVING MEASURES: FIRST-AID.

(A) (i) *Prevention of Suffocation* caused by falling back of tongue, or blood in upper respiratory passages.

(a) Pull tongue forward.
(b) Hold head forward if walking.
(c) Carry face downwards if on stretcher.

(ii) *Arrest of Hæmorrhage.*

(a) Posture: sitting.
(b) Plugging.
(c) Digital pressure: get bleeding-point if possible between finger and thumb outside.

(iii) *Treatment of Shock.*

(a) Warmth.
(b) Fluid: feeding-cup or catheter. Catheter through nose.
(c) Relief of pain.

Preliminary Treatment.

(B) (i) Injection of A.T.S. 3 c.c., i.e., 3,000 units.

(ii) Irrigation warm soda bic. 4 per cent., or normal saline.

(iii) Removal of *completely* detached teeth, bone, or f.b's. Loosened teeth must not be touched unless dangerous.

(iv) Support of jaw, e.g., barrel bandage, elastoplast strapping.

(v) Four-tailed bandage must *not* be used. Apply sterile surface-dressing, preferably saline pack.

(vi) It may be necessary as an emergency measure to do laryngotomy or tracheotomy.

II. HOSPITAL TREATMENT.

(A) Surgical Aim: (i) Mitigation and obliteration of deformity.
(ii) Restoration of normal functional contour.

(B) Dental Aim: Maximum masticating efficiency dependent on:—

(a) Bony foundation.
(b) Retention of natural teeth in occlusion.

In order to attain the above, it is imperative that the surgeon and dental-surgeon work in close collaboration both before and during further treatment.

(C) Pre-operative Treatment.

(i) Revision of first-aid measures.

(ii) X-rays to show :—

(a) Number and direction of fractures.

(b) Location of f.b's.

(c) Teeth directly involved or likely to be involved.

(d) Presence of roots or fillings in fracture-line. X-ray films usually necessary :—A.P. and two laterals; 30° and 90° occipito-mental for nasal and maxillary; rotated views of lower jaws to include temporo-mandibular joints; dental films. Particular attention to regions of condyles.

(iii) Photographs if possible.

(D) OPERATION.

(1) *Choice of Anæsthetic.*—Ideal anæsthetic is endo-tracheal G.O.E. through nasal catheter, which may be preceded by pentothal induction. If technical difficulties render this impossible, e.g., gross destruction maxilla, continuous pentothal either directly or through saline drip if in use.

(2) *Time of Operation.*—Operate as early as is possible, compatible with shock, as primary healing may be obtained if sutured within twenty-four hours.

(3) *Operative Technique.*—(A) SURGICAL.

(i) Clean wounds with wet saline gauze, and irrigation and currette for f.b's. Soap and water may be used.

(ii) Excision of wounds unnecessary, except for removal of non-viable tissue or to make accurate suture of jagged edges possible.

(iii) In wounds *without* gross loss of soft tissue, suturing should be attempted up to twenty-four hours, provided there is *no* tension. If there be any tension or wound older than twenty-four hours, dress with saline pack.

(iv) In wounds *with* gross loss of soft tissue, mucous membrane should be stitched to the skin-edge without tension. Where there is loss of bone eventually necessitating grafting, this suturing should attempt to cover the raw-bone ends. Soft tissue loss should be dressed with saline packs to encourage the formation of a granulating wound suitable for grafting.

(v) Reduction of fracture of maxilla and facial bones should be carried out at this stage.

(B) DENTAL.

(i) Avoid damage in introducing Mason's gag or prop.

(ii) Obtain impressions of both jaws if possible with composition and shallow trays.

(iii) Reduction and immobilisation of fragments.

(a) Interdental wiring if sufficient natural teeth are in occlusion.

(b) Splinting.

(iv) Removal of throat packing before fixing the wire.

(v) Pass suture through mid-line of tip of tongue for control during recovery.

(vi) *Drainage.*—Adequate dependent drainage must be provided for every obviously compounded fracture.

(E) POST-OPERATIVE.

(i) Sitting posture with macintosh bib.

(ii) Hourly irrigation of mouth fracture-lines, and drainage tubes with warm 4 per cent. soda bicarb.

(iii) Frequent feeds by intra-oral or intra-nasal tube (fluid intake kept up to six pints in twenty-four hours).

(iv) Lubrication of soft tissues, lips, etc., with liquid paraffin or acriflavine in glycerine, 1/1000.

(v) Preparedness for secondary hæmorrhage from second to twelfth day.

(vi) Co-operation with physician to avoid chest complications.

(F) REMOTE TREATMENT.

(I) *Bone Grafting.*—At least *six months* should have elapsed from disappearance of sepsis. As a general rule, gaps of over 1-5 cm. will require grafting. The ideal graft obtained from iliac crest. Graft after bending and shaping should be fixed with stainless steel ligature. Fixation should be firm enough in four weeks for movement to be allowed.

(II) *Plastic Repair.*—To be undertaken by specialist plastic surgeons. Except for :

(i) Buccal epithelial inlay, which enables a sulcus to be made between the cheek and the bone-graft, and so enable a denture to be applied over this area.

(ii) Excision of scars where the final cosmetic result is not in doubt.

(iii) Thiersch grafting for the less extensive granulating wound.

TREATMENT OF COMPOUND FRACTURES WITH SPECIAL REFERENCES TO COMPOUND FRACTURE OF LOWER LIMB

I. GENERAL FIRST-AID MEASURES.

(1) Treatment of hæmorrhage a primary consideration. Avoid unnecessary tourniquet.

(2) General shock measures.

(3) Alleviation of shock by protection from further risk of exposure, loss of heat, hunger, fatigue, and fear.

(4) Relief of pain : (a) morphia; (b) immobilisation.

(5) A.T.S.

(6) A gas gangrene serum.

No manipulation or attempted reduction. Pain is controlled by adequate immobilisation. The Thomas splint should be used for fracture of femur, knee-joint, tibia, and fibula, and equally important for extensive wounds of the soft parts.

Clothing should not be removed, and it is not often necessary to take off the boot. The whole limb must be elevated either on suspension bar or by other means.

The wound.—Sprinkle with iodine. Cover with pad and bandage or shell dressing. During *transport,* bandages, slings, and traction device should be frequently

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inspected, also watch kept for hæmorrhage. *Record* of morphia and/or other medication given.

II. Surgical Treatment.

No operation should be undertaken except where full surgical facilities are at hand. General measures are only discussed, as each case presents its own problems, and the surgical approach must be a matter of individual experience and judgment. The main points at issue :—

1. When to operate.
2. When to excise.
3. When to do a rapid debridement.
4. When to amputate.

(1) Under civil conditions, about eight hours may be regarded as the limit allowed, when after surgical toilet, an open fracture may be converted into a closed one. This may be regarded as the period of contamination, during which excision may safely be practised. Under war conditions, excision may be practised up to twelve hours, but it is well known that after six hours the hæmolytic streptococcus may have gained entry into the tissues.

(2) *The operation.* Preparation of skin. No tourniquet is employed. The wound is covered with gauze soaked in H^2O^2. Surrounding skin washed, shaved, and washed again, always in a direction away from the wound. The skin is then painted with iodine. Remove dressing. Excise skin and subcutaneous tissue, if possible in a single piece. Then invert sterile towels over skin-edges, and secure. Dealing with compound fractures of femur and tibia, it is an advantage to provide some form of skeletal traction during the operation.

Deep tissues are searched for foreign bodies. Damaged non-contractile muscle is removed. Fascial planes are incised to lay open all the recesses of the wound, and to prevent future pocketing. It should be remembered that the velocity of the H.E. particle is such, that resistance by bone converts it for practical purposes into an explosive agent, and the injury to soft parts may be out of all proportion to the size of the particle or size of the entry-wound.

Completely detached bone-fragments are removed, and those retaining attachment to soft parts are preserved. Contaminated bone-edges are trimmed with a thin osteotome. Every effort is made to render the wound dry, and only larger vessels require ligature. It is our practice to flood the area with H^2O^2, which is reasonable in the face of possible infection by gas-forming organisms. Hydrogen peroxide also will often bring to light small foreign particles and separated tissue.

No attempt is made to suture muscle nor aponeurotic layers.

As a rule, only the skin is closed by silkworm gut, and there must be no tension. Many extensive wounds are unsuitable for closure. Closure of the wound makes the fracture a closed one, and the surgeon proceeds to further manipulation if required, and to immobilisation of the limb. The local application of sulphonanilamide is desirable, and may be distributed throughout the wound with an insufflator, or in handy fashion with a spoon.

Immobilisation may be secured with skeletal traction, and the Thomas or

Brauns splint. If plaster of paris is employed, the case is more readily portable.

PLASTER OF PARIS.

(1) Should be applied directly to skin, but bony prominences, such as the crest of the ilium, tibial crest, and malleoli, may be protected with adhesive felt.

(2) The wound, whether closed or open, together with a generous area of surrounding skin, should be covered with vaseline gauze. This will prevent the troublesome dermatitis which often follows when the skin is exposed for long periods to discharge.

(3) Plastering by slabs is preferable.

(4) On no account should anyone except the responsible surgeon be allowed to nibble the plaster, for swelling always follows the plaster-edge. Should the plaster require alteration, it should be split throughout its whole axis. In this respect it is an advantage to score the plaster with a knife before it is dry, to provide an easy line of cleavage.

(5) Windows over wounds are not desirable, and merely cause bulging of the tissues and congestion.

(6) An evenly-applied plaster will provide an even distribution of pressure.

(7) The limb should be elevated.

OBSERVATION OF PLASTER CASE.

1. *Circulation.*—Circulation in the toes frequently observed. Test each digit by pressure, when the blood-return should be rapid. Do not confuse bruising with ischæmia. If the circulation is embarrassed, split plaster throughout the whole length.

2. *Pressure sores.*—Due to pressure over bony prominences. Inequality of pressure. A frequent cause is due to allowing an edge of the plaster bandage to cut across the case and produce a ridge on the inner surface.

Roll bandage on, and do not pull.

3. Finger and thumb depressions of assistant.

4. *Cracking* at joint level.

5. *Depression* by allowing cast to dry on hard surface.

6. Some *œdema* is inevitable in the digits, but elevation and active exercises of toes is usually sufficient to control. Cast should always extend to the web of toes (dorsum) and beyond toes (sole).

7. *Temperature.*—Nearly always an initial rise.

8. *Dermatitis.*—Characteristic burning pain and irritation.

9. *Blistering* of skin hardly ever occurs in supported limb.

10. Progress of *inflammation*—characterised by the usual signs and symptoms.

11. General complications :—Hypostatic pneumonia.
Mental impairment and degeneration.

12. *Odour.*—The unpleasant odour from a compound fracture may be mitigated by the use of filter cloth.

13. *Sharp edges* should be trimmed and bound with adhesive tape.

14. *Bed exercise* should be graduated and begun at once. Cases confined to bed should be nursed outside if possible.

15. Bed cases to become *ambulatory* at the earliest opportunity.

16. In our experience, *segregation* of bone cases is desirable in all cases.

17. Allow no one to NIBBLE the plaster.

The Treatment of Late Cases.

No excision should be attempted after eighteen hours, although there can be no inflexible rules. These cases fall into two classes :—

(a) Those with evidence of toxic absorption.

(b) Those exhibiting no such signs.

The treatment of both is *debridement*. This implies the removal of dead and foreign matter with a wide opening up of fascial and aponeurotic planes, to provide easy and unhampered drainage. Use of sulphanilamide is desirable. The wound is lightly packed with vaseline gauze, and the surrounding skin is similarly protected. The whole limb is encased in plaster, after the lines of the Winnett Orr treatment. This procedure may be accompanied by an initial rise of temperature. Successful treatment results in :—

(i) Relief of pain.

(ii) Rapid improvement of general condition.

Indications for Removal.

1. Laxity of cast.
2. Secondary hæmorrhage.
3. When condition of patient fails to improve, or deteriorates.
4. When plaster becomes softened by discharge.
5. When smell becomes intolerable.

Indications for Amputation.

Lower Limb.	Upper Limb.
Injury to femoral or popliteal is very serious.	Arterial injury relatively less serious.
Loss of skin and disruption of main neurovascular supply.	Do.
As a life-saving measure to control hæmorrhage.	Rarely necessary.
Other Considerations.	
Artificial limbs excellent.	Almost useless.

Contamination.—Lower half of body is always more soiled than the upper.

Gas gangrene.—Fairly common in leg. Rare in arm.

Shortening of importance in legs; not so much in the arm.

An additional military factor.—A long evacuation phase, and absence of skilled surgeons may be in favour of primary amputation.

Methods of Applying Traction to the Lower Limb.

In most cases it is an advantage to provide means of skeletal traction before attacking the fracture-site.

Femur.

1. Thomas splint. Kirschner wire extension via tibial crest.
2. Brauns splint and skeletal traction.
3. Sliding-bed traction.

Tibia and fibula.

1. Watson Jones apparatus.
2. Thomas splint and flexion-piece, with Kirschner wire extension via os calcis.
3. Brauns splint and skeletal traction.
4. Distraction apparatus.

Two Final Words.

1. The treatment of all fractures is only just begun when reduction is secured and infection is controlled, and only ends when rehabilitation is complete.

2. The commonest single cause of non-union of any fracture is incomplete immobilisation and failure to immobilise for sufficiently long.

THE TREATMENT OF BURNS AND SCALDS (EXCLUDING CHEMICAL BURNS)

In view of the varieties of treatment, simple and elaborate, which have from time to time been advocated for the treatment of burns, and also the considerable controversy which is, at the present time, centering on this practical subject, members of R.A.M.C. hospitals and consultant members of civil hospitals in the Belfast area have met and considered what measures might be taken to unify and standardise the treatment of burns.

As a result of these meetings, the following lines of treatment are recommended :

First-Aid for Cases of Burns and Scalds Requiring Hospital Treatment.

Give Warmth.
Fluids (hot sweet tea).
Morphia in adequate doses, e.g., one-eighth to one-half grain for adults, noting times and dosage on label.

Avoid Undue exposure.
Clothing should only be removed by or under orders from the medical officer, and only when delay in transport to hospital is likely (this does not apply to clean burns or scalds).

Apply Clean dry lint to exposed areas of the burn. Greasy dressings should not be applied, as they make subsequent cleansing and treatment difficult.

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General Measures.

1. *Primary shock.*—Onset immediate, recovery usual, may pass into secondary shock without recovery.
   Bed, minimum interference.
   Warmth (shock-cage or bottles).
   Morphia, one-eighth to one-half grain for adults. Children, heroin (under two, 1/96-1/24 grain).
   Fluid by mouth.
2. *Local treatment.*—(See below) when condition of patient permits.
3. *Routine.*—Hourly pulse, blood-pressure, and, in severe cases, hæmoglobin estimation.
   Four-hourly temperature.
4. *Secondary shock.—Time.*—Usually two to four hours; may be delayed; may follow primary shock without recovery.
   Signs.—The typical signs: note fall in blood-pressure and rise in hæmoglobin, e.g., 135 to 140 per cent.
   Treatment.—(a) *Plasma transfusion* (restore Hb. to normal).
   (b) *Fluid* with glucose as tolerated orally.
   (c) *If unable to swallow*: intravenous drip, saline alternating with glucose saline, forty drops per minute (adults).
5. *Toxæmia.—Time.*—Thirty-six to forty-eight hours. May even be later.
   Signs.—Pyrexia, fall in blood-pressure, vomiting, rising pulse, restlessness, delirium, albuminuria.
   Treatment.—Replace the chloride lost by intravenous drip, give D.O.C.A. (desoxycorticosterone acetate) ten mgm. four-hourly, or other adreno-cortical extract, control by fall in pulse-rate and return of chlorides in urine. Examine coagulum carefully, and treat as below.
6. *Sepsis.*—Sulphanilamide in addition to local treatment, e.g., two tablets four-hourly day and night for two days, then reduce dose.

Local Treatment.

First, second, and minor third-degree burns (excluding those of *face, hands, feet, and perineal region*).

Anæsthesia.—Gas and oxygen (no cyanosis) is ideal.
Avoid ether, as it causes exudation.

Exposure.—Expose small areas at a time, and so minimise heat-loss.

Cleansing.—*Normal saline* at body temperature on gauze, or *soap and water.*
No rough handling or scrubbing, but remove all raised epithelium, *especially at the edges.*
If possible, dry by hot air, e.g., electric hair-drier or cage.
Avoid ether, as it causes exudation and great heat-loss.

Coagulation.—(1) *Bettmann's technique.*— 1 per cent. gentian violet.
5 per cent. tannic acid.
10 per cent. silver nitrate.
1 per cent. gentian violet.

Apply at body temperature in the above order by dribbling from a piece of gauze, drying all the time.

N.B.—In multiple cases much time is saved by one team cleaning and the next coagulating, and thus remaining uncontaminated.

No dressings or clothes in contact with the coagulum.

Nurse on least burnt surface under large shock-cage.

After treatment.—Four-hourly application by nurse with fledget of wool on forceps of methylated ether, followed by gentian violet or sulphanilamide powder to edges and any moist or poorly coagulated areas. *Watch the edges* constantly.

2. *Compress method.*—Suitable for single limbs and smaller areas and for the least burnt surface in extensive burns. Cleanse as above. Apply gentian violet. Apply four layers of gauze impregnated with 5 per cent. tannic acid in water, or in extreme emergency as a powder.

FACE, HANDS, AND FEET.

Triple dye.—1 in 400 gentian violet
1 in 400 brilliant green } equal parts.
1 in 1,000 acriflavine

Cleanse as above, apply the dye. Leave uncovered. Reapply four-hourly.

SEVERE THIRD- AND FOURTH-DEGREE BURNS AND BURNS OF PERINEAL REGION.

Saline compresses.—Keep moist. Sulphanilamide as prophylaxis and for established sepsis. Treatment is directed towards the development of healthy granulations for early skin-grafting. Sulphanilamide powder or albucid paste are alternative methods.

Limbs require splinting (e.g., plaster shell) *and elevation to avoid contractures and œdema.*

Burns of any severity require rapid evacuation (on stretcher, irrespective of whether the legs are burnt or not).

REVIEW

HANDBOOK OF ANÆSTHETICS (formerly ROSS and FAIRLIE). Revised by R. J. Minnitt, M.D., D.A. Fifth Edition. 1940. Edinburgh: E. & S. Livingstone. Pp. 364. 12s. 6d. net.

DR. MINNITT, who has revised this well-known Handbook, states in his preface that he has included descriptions of "new methods and new apparatus which have been established as having a definite claim to recognition," and he suggests that more radical alterations will be made in the next edition.

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The subject matter is well arranged, accurately and clearly presented, and the large number of useful illustrations should prove most helpful to the student.

Apply at body temperature in the above order by dribbling from a piece of gauze, drying all the time.

N.B.—In multiple cases much time is saved by one team cleaning and the next coagulating, and thus remaining uncontaminated.

No dressings or clothes in contact with the coagulum.

Nurse on least burnt surface under large shock-cage.

After treatment.—Four-hourly application by nurse with fledget of wool on forceps of methylated ether, followed by gentian violet or sulphanilamide powder to edges and any moist or poorly coagulated areas. *Watch the edges* constantly.

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Weil's Disease

By N. C. GRAHAM, M.C., M.B. AND M. G. NELSON, M.D., M.R.C.P.
Institute of Pathology, and Royal Victoria Hospital, Belfast

WEIL'S DISEASE, since its original clinical description by Adolf Weil of Heidelberg in 1886, has been diagnosed clinically in many parts of the world. Inade and Ido in 1914 discovered the pathogenic agent by inoculating the blood from a patient during the first week of the disease into a guinea-pig and found that the organism responsible was a spirochæte. Noguchi, who also studied the organism, described its typical morphology and called it the Leptospira Icterohæmorrhagica. This opened the way to more accurate diagnosis by bacteriological methods and to experimental investigation of this infection.

It was found that in the early stage of the disease, usually during the first week, the leptospira was present in the blood and that inoculation of a guinea-pig with the blood from a patient resulted in the production of leptospirosis in the experimental animal in a high percentage of cases. Later in the disease, i.e., about the third week, the organism was shown to be present in the urine, where its presence might be demonstrated by dark ground illumination of a urinary deposit or by animal inoculation. The discovery of the infecting organism by either of these methods is a difficult procedure, requiring careful co-operation between the clinician and laboratory as well as careful attention to details of technique.

A method was discovered by Schuffner of demonstrating the presence of antibodies in serum during or after the second week by means of an agglutination technique, using a young culture of the organisms. This technique has been widely applied and its specificity proven. The only drawback to this procedure is its technical difficulty and the necessity of maintaining stock cultures of strains of leptospira at a suitable growth-stage for the agglutination reaction. This latter difficulty has been somewhat overcome by the use of the formalised cultures advocated by Major Brown, which display the same specificity and sensitivity as living cultures.

Weil's disease is largely an occupational disease with a special incidence among workers employed in rat-infested premises. Thus it has been described among sewer workers (Alston and Brown, 1935; Halstead, 1935; Maxwell, 1935); fish-workers of Aberdeen (Davidson and Smith, 1935, 1939); miners (Gulland and Buchanan, 1924; Swan and McKeown, 1938; Rees, 1939; Sladden, 1939); tripe workers (Stuart, 1938). Besides this occupational incidence, sporadic cases do occur often following immersion in rat-infested streams (Robertson, 1939). In all these cases the rat is considered to be the vector of infection. In America, however, the leptospira icterohæmorrhagica of Weil's disease and the leptospira canicola, an organism which can cause 'yellows' in dogs, are considered to be identical. This raises the possibility of the dog as a possible vector of infection, and the source of infection has been traced in one case to a dog with 'yellows.' Both the patient

and the dog gave positive sero-agglutination to leptospira icterohæmorrhagica in the same titre.

Although Weil's disease has been described in many parts of Great Britain, no cases of the disease have yet been recorded from Northern Ireland, despite the fact that the disease has been suspected in this region for some years.

Two cases of suspected infectious jaundice occurred within a few weeks of one another among sewer workers in the city of Belfast, and one of these was diagnosed clinically as Weil's disease by one of us. From this case the leptospira was recovered from the urine, and has since been maintained on culture. Both cases gave a positive sero-agglutination of leptospira icterohæmorrhagica (stock strains) by Schuffner's technique in high titre.

CASE I.

Patient—Male aged 31 years; *occupation*—labourer, city sewers; *admitted*—27th August, 1939; *discharged*—22nd September, 1939.

Three weeks before the patient was admitted to hospital he took ill with a severe frontal headache, rigors, and sweatings. He was seen and treated by his own doctor with some symptomatic relief. About four days before admission his condition became worse, and he then complained of vomiting and a general feeling of nausea. The headache was still present, but had become more severe, whilst generalised pains developed throughout his body so that the slightest movement caused extreme agony. Abdominal pain was severe in character and situated mainly in the epigastrium. His throat was sore and swallowing difficult. Micturition was normal in frequency and free from pain.

On admission.—Temperature 99°; pulse 96. The patient was a well-built male subject of good nutrition, who showed slight signs of general muscular wasting. He was obviously in extreme pain, which was accentuated by any movement entailed during his physical examination. There was a generalised mild icteric tinge of the skin and conjunctivæ, but no evidence of gross anæmia or œdema. The conjunctivæ were inflamed and chemotic. The skin was dry and warm, but showed no evidence of rash or purpuric eruption. The joints could be moved passively, but the muscles were extremely tender to pressure.

The teeth were carious; the throat injected and the tongue covered with a thick white, dry fur. The abdomen was somewhat retracted, but moved slightly on deep inspiration. Abdominal tenderness was generalised with some localization in the epigastrium, right subcostal area, and both renal angles. The liver was not palpable nor the spleen enlarged. Examination of the other systems revealed no abnormality, the heart-sounds were clear and regular, murmurs absent, and the blood-pressure 100/70. The chest was clear except for an occasional moist rale.

Urinalysis.—Dark amber in colour: reaction acid: specific gravity 1018: albumen + + : bile pigment + : casts + .

Van den Bergh.—Biphasia + : indirect reaction + + .

Blood urea.—169 mgm. per cent.

Widal.—Negative.

Wassermann.—Negative.

Blood examination.—R.B.C. 3,900,000; W.B.C. 7,488; Hb. 83 per cent.
Film—nil abnormal in white or red cells.

The patient was put on alkalis, intramuscular injections of collosol calcium, and glucose drinks.

The urinary excretion was low, and the patient was still very ill on the third hospital day. The tongue was dry and covered with a brown fur. The liver was now enlarged to three finger-breadths below the right costal margin. The spleen was not palpable.

A continuous intravenous drip of glucose saline was set up on the fourth hospital day and a careful water balance kept. Despite this the patient's condition deteriorated, the blood urea rose to 278 mgm. per cent., and jaundice became deeper and vomiting recommenced.

On the seventh hospital day, when the patient's condition seemed hopeless, 100 c.c. of convalescent serum was administered intramuscularly and the continuous intravenous administration of fluids stopped owing to venous thrombosis. Two days later his clinical condition was much better and his urinary excretion improved.

The blood urea gradually returned to normal levels, and on discharge was 32 mgm. per cent. The temperature, which was intermittent, became normal, and the jaundice disappeared, although the liver enlargement persisted for some weeks.

The urinary deposit was inoculated into a guinea-pig on the twenty-fourth day of illness with negative results. A subsequent inoculation of a guinea-pig on the forty-sixth day of his illness produced typical leptospirosis. The leptospira could be seen on dark ground illumination of a mush of liver and kidney and in the heart's blood. From the blood and organs of this guinea-pig, cultures were made into Fletcher's medium and the disease transmitted to another guinea-pig by inoculation.

CASE II.

Patient—Male aged 21 years; *occupation*—labourer, Corporation sewers; *admitted*—5th September, 1939; *discharged*—16th September, 1939.

The patient stated that two days before he took ill he was working in a sewer, when a cart emptied its contents down the manhole, causing him to swallow some sewage. Two days later patient developed a severe headache and felt weak and ill. Owing to severe generalised myalgia he was unable to lie comfortably in bed, and tossed repeatedly without relief. Vomiting developed, and the patient was soon unable to retain anything in his stomach. Besides the generalised pains felt in all the muscles of the body, severe pain appeared under the left costal margin. The bowels were obstinately constipated and sleep was poor. Micturition was normal in frequency and devoid of pain.

His previous history revealed nothing of note.

On examination.—Temperature 100.2°; pulse 72. The patient was a well-nourished male of good build and nutrition. The face was markedly flushed. There was generalised mild icterus, which involved the conjunctivæ. The superficial lymph-glands were not palpable. Rash and purpuric changes were absent. The

tongue was dry and furred and the fauces inflamed. The abdomen was of normal contour and moved freely with respiration. There was no evidence of rigidity. Considerable tenderness was felt over the right and left costal margins. The liver was slightly enlarged, but the spleen was not palpable. Tenderness was marked in the R.I.F., and the cæcum could be palpated. There were no signs in any of the other systems; the lungs were clear, the pulse regular, blood-pressure 122/80, and the heart-sounds normal.

INVESTIGATIONS.

Urine.—Amber colour : neutral : specific gravity 1022 : trace of albumen : trace of blood : bile + .

Blood urea.—50 mgm. per cent.

Van den Bergh.—Biphasic reaction.

W.B.C.—10,500.

Wassermann.—Negative.

Fæces.—Occult blood present.

Urinary deposit repeatedly examined by dark ground illumination was negative for leptospira. Urine inoculated repeatedly into guinea-pigs without result.

Serum.—Schuffner's sero reaction positive in a titre of 1 in 1,000.

The temperature dropped to normal, and the patient rapidly recovered within a few days.

COMMENTARY.

Occurrence.—These two cases of Weil's disease are, we believe, the first to be recorded in Northern Ireland. Although on some occasions in the past the disease has been suspected on clinical grounds, these previous cases were without bacteriological or serological proof.

Occupation incidence.—Both of these cases occurred in sewer workers, and thus tends to confirm the high occupational incidence of the disease among these workers, which has been stressed by many authors. Both the men were employed on a rat-infested stretch of sewer, and both fell ill within a few weeks of one another. A fellow-worker was found to have been ill some weeks previously with an obscure illness and jaundice, and his serum gave a Schuffner's reaction in a titre of 1 in 10,000. He was previously also a case of Weil's disease, and it was the serum from this man which was used in the treatment of Case I.

Clinical picture.—The clinical picture was classical in both cases, in that it comprised the triad of an acute fibrile illness, jaundice, and a toxic nephritis with nitrogen retention. The clinical severity varied greatly, and while one case had what was considered a hopeless prognosis, the other was little disturbed by the disease. The more severe case had marked diminution of urinary output with evidence of extreme renal damage, and nitrogen retention which rose to 278 mgm. per cent. However, at no time during his clinical course did he show any evidence of generalised disease of the capillaries and hæmorrhages were entirely absent. Case II, on the other hand, had a little melæna and yet a much milder clinical course. Neither of the cases showed the three clinical stages which have been described for the course of the disease, and Case I was already three weeks ill

when he was first admitted to hospital, and he remained in hospital for a further four weeks before he was fit for discharge. The generalised myalgia was one of the more distressing complaints, and caused the symptoms to be greatly out of proportion to the physical signs observed. This point has been stressed in diagnosis and is of considerable importance.

While no attempt at generalisation can be made from observations on a single case, it is not without significance that a very severe case of Weil's disease showed a dramatic response to convalescent serum. The patient was in his fourth week, and showed jaundice, pyrexia, myalgia, headaches, and marked toxic nephritis. His urinary output was very low, the urine loaded with albumen and showed casts, while his blood urea had risen to 278 mgm. per cent. Clinically, his prognosis appeared to be very poor, yet within a few days of the administration of 40 c.c. intravenously and 20 c.c. intramuscularly of convalescent serum with a titre of 1 in 10,000, he had improved dramatically. He appeared better clinically, his urinary output was restored, and the nitrogen retention fell. Serum was not tried out on the other case, as his condition was so mild that it appeared unnecessary.

Commentary on the Procedure Necessary for the Diagnosis of the Disease.

The general procedure is to demonstrate the organism in the blood during the first week of the disease, in the urine during the subsequent stages, and then to confirm the diagnosis by serological means. The exact procedure will depend upon the duration of the symptoms. In the first week every effort should be made to demonstrate the leptospira in the blood, either by direct microscopic examination using a dark ground illuminator, or better, by inoculating 5 c.c. of freshly-drawn blood into the peritoneal cavity of a young guinea-pig and reproducing the disease. The cases here described were considered too far advanced for either of these procedures to be successful. The leptospira is fairly easily recognised. Its morphology can only be studied satisfactorily by dark ground illumination. It is a delicate organism about from 6 to 12 m. in length and about 0.2 m. in thickness. The coils, which are regular and closely wound, are too fine to be resolved in a stained preparation. The ends are tapering and characteristically "hooked." The growth of the organism in a fluid medium gives rise to "nests," which appear as highly refractile balls composed of many interlaced organisms.

It will probably be during the second state of the disease that a confirmation of the diagnosis will be sought in most cases. Observations indicate that the leptospira begin to appear in the urine towards the end of the second week and persist for a few weeks only, but their existence has apparently been demonstrated in some cases for several months. Direct microscopic examination of the urine is unsatisfactory, as other varieties of spirochætes may be found in normal urine. The centrifuged deposit of 60 to 80 c.c. urine in 5 c.c. of normal saline is inoculated peritoneally into a young guinea-pig. This method will only be found satisfactory if the inoculation is carried out within one hour of the urine being voided. It is necessary to administer some reagent to the patient to make his urine approxi-

mately neutral. This precaution is important, as the organism is quickly destroyed by strongly acid or alkaline urine. The successful isolation of the organism from Case I on the forty-sixth day of the disease was probably due to the adoption of this technique, as a previous inoculation was unsuccessful. Most animals will become ill, jaundiced, and die within a week. At post-mortem, the animals show generalised jaundice and there are hæmorrhages into various parts of the body, particularly the lungs, which show irregular areas of varying sizes sharply demarcated from the surrounding tissues, resembling the mottled wings of a butterfly. The spirochætes are most easily demonstrated in a suspension of the liver. It is now recognised that some animals may survive an actual infection.

A considerable amount of attention has been paid to the serological diagnosis. Specific antibodies begin to appear in the blood about the tenth day, but are not of diagnostic importance until about the fourteenth day to the eighteenth day of the disease. A definite negative reaction after the thirtieth day rules out infection.

We are indebted to Dr. R. D. Stuart of Glasgow for carrying out some preliminary tests on the agglutinating titre of the patient's serum in both of these patients. In both cases he reported a positive result. Subsequent serological examination carried out by us, using Schuffner's technique, and the strain isolated from Case I as well as strains sent to us by Dr. Stuart, confirmed this finding. In Case I a positive result was obtained in a dilution of 1/3000 on the thirty-second day of the disease. It cannot be too strongly emphasized that diagnosis of the disease is a matter of co-operation between the clinician and the bacteriologist.

References.

Alston, J. M., and Brown, H. C. : *B.M.J.*, 1935, ii, 339.
Davidson, L. P. S.; Campbell, R. M.; Rea, H. J.; and Smith, J. : *B.M.J.*, 1935, ii, 1137.
Davidson, L. P. S., and Smith, J. : *B.M.J.*, 1939, ii, 753.
Gulland, G. L., and Buchanan, G. : *B.M.J.*, 1924, i, 313.
Halsted, E. A. M. : *B.M.J.*, 1935, i, 1067.
Inada, R., *et al.* : *Jour. Exper. Med. and Hyg,*, 1916, xxiii, 377.
Maxwell, J. : *Lancet*, 1935, ii, 998.
Noguchi, H. : *Jour. Exper. Med.*, 1918, xxvii, 575.
Rees, W. E. : *B.M.J.*, 1939, i, 603.
Robertson, K. M. : *B.M.J.*, 1939, ii, 1300.
Schuffner, W. : *Tr. Soc. Trop. Med. and Hyg.*, 1934, xxviii, 7.
Sladden, A. E. : *B.M.J.*, 1939, i, 605.
Stuart, R. D. : *Lancet*, 1938, i, 603.
Swan, W. G. A., and McKeown, J. A. : *Lancet*, 1938, i, 201.
Weil, H. A. : *Deutsches Arch. f. Klin. Med.*, 1886, xxxix, 209.

Aspects of Medical History

The Annual Address to the Students of the Royal Victoria Hospital Session 1941-42

By R. S. ALLISON, M.D., F.R.C.P. (LOND.)

IT is my privilege to-day to be the one chosen to welcome you to another year of work at the hospital. Believe me, I do so with real pleasure and with the sincere wish that, within these walls, you students may acquire that understanding of human nature and knowledge of disease which must be the rewards of love of medicine and diligent study.

Some of you will be returning to familiar surroundings; others will be preparing for the final year's work. But there must be many of you who have just completed your preliminary studies in the dissecting-room and laboratory. You, especially, will gaze eagerly upon the promised land of clinical medicine, and I think you will not be disappointed. It is a fair prospect, with wide open spaces that delight the eye; but I must warn you, the distances are deceptive! You must allow plenty of time if you would journey inland to the great city, and you must carry sufficient store of the guiding principles of the basic sciences: anatomy, physiology, and pathology, otherwise you may lose your way. There are signposts at the cross-roads, of course, and most of them reliable, but only the incurious will follow them blindly. Let me remind you, that in this country to-day, we rely less and less upon signposts! Most of them, indeed, have been removed. Medicine, like the nation, may be approaching a decisive phase in its history, and it is you who will be setting forth on the road of practice in the immediate post-war era.

The year 1940-41 has been an unfortunate one for the hospital, for it has seen the deaths of three distinguished members of our honorary consulting and visiting staffs—the Emeritus Professor of Surgery: Colonel Thomas Sinclair; a former senior surgeon of the hospital: Mr. T. S. Kirk; and the senior radiologist: Dr. Robert Maitland Beath. Professor Sinclair became a student at Queen's in 1877, that is, sixty-four years ago; at about the same period as the Bell telephone and the incandescent electric lamp were invented. He was still a student when Pasteur discovered the streptococcus, and Eberth isolated the typhoid bacillus. I mention these facts not because they are new, but because they illustrate the great changes that have taken place in this most crowded epoch of our medical history, the past fifty years. As the poet says:

> "Time is the feather'd thing: . . .
> His minutes, whilst they're told,
> Do make us old." . . .[1]

During the thirty-seven years that Professor Sinclair held the Chair of Surgery at the University, he was responsible for the surgical training of more than two thousand students. As a colleague has written:—"He outlived all his contemporaries in the school and some of his earlier students, . . . but it was never his fate to be forgotten or neglected."[2] Truly a great man, the passing of Thomas

Sinclair severs an important link in the chain that unites us to the surgery of the previous generation.

Mr. T. S. Kirk had also ceased active hospital work at the time of his death, but he had laboured there for a period of thirty-five years. He retired in 1935, so that few, if any, of you students to-day will recall his teaching. It was unorthodox, but it aimed at conveying the practical essentials, and it was the sincere expression of one who had a high conception of the purpose of life. Belief in certain principles was like a religion to Surgeon Kirk, and he had many followers, especially among his former pupils and house-surgeons who were most closely associated with him in his work.

Death knows no law, follows no set course, for while it summoned these two senior members of our staff after they had reached their allotted span, it claimed Dr. Maitland Beath, our senior radiologist, while he was still comparatively young and at the most active period of his career. His death came as a great blow to all, especially to his colleagues on the staff of this hospital, by whom he was whole-heartedly beloved. These are banal phrases with which to salute the memory of a colleague who was especially dear, but they are sincerely spoken, and in truth, mere words can no more express than they can retrieve the loss we have suffered in his untimely death. A brilliant student, Dr. Beath graduated with first-class honours and took first place in his year. As a soldier in the previous Great War, he served with the R.A.M.C. in France and in the Middle East, and with keen enthusiasm for his chosen subject, in the ensuing years he gained an international reputation among radiologists. Both at home and abroad he was respected for his good judgment, keen discernment, and absolute freedom from narrow bias or prejudice. These are achievements of which any man might have been proud, but Maitland Beath was not proud. The qualities that will ever linger in our memory were his unassuming modesty, his cheerful friendliness, and his absolute honesty of purpose. He had no enemies—not even death, for he met his end bravely; his quiet imperturbability undismayed by foreknowledge of its certain approach.

We remember the loss of these our teachers and former colleagues, but the year has seen other trials. The bitter turmoil of war has spread to Belfast, and left its mark on many homes and enterprises. It is good to know, however, that this hospital, like its sister institutions, was not found wanting, but that it proved itself fully capable of dealing with the emergencies that arose. We are pleased to remember, too, that we were accorded official recognition on this occasion, for Her Royal Highness the Duchess of Gloucester visited the hospital on the 23rd of April, and personally interviewed the many victims of the air-raid who were under treatment.

In the ever-changing scenes of to-day, when everyone is making the greatest effort for the common cause, it is difficult to relax or to desist even momentarily from the tasks upon which we are set. Yet it is necessary that we should do so, occasionally, if only to refresh ourselves, or to see more clearly the path before us. In this brief hour that we are together, I propose to try the experiment, and talk to you of some aspects of medical history in which I have been interested. So, for the moment, let us forget the case to be written up in the ward, the lecture to be

attended at Queen's, the examination to be passed next June. Let us step back into bygone ages, and consider history.

Now, no one who is interested in this subject, and especially in the history of medicine, can afford to make the mistake of thinking only in terms of the past. You will remember the American writer, Clare Boothe, in her "European Spring," refers to a conversation she had with an American businessman in Rome. The latter, in answer to a question as to what he thought of Mussolini, replied that the Italian dictator was the Killyoo bird of Europe—he flew backwards because he didn't care where he was going; he just wanted to see where he had been! . . . If we are to fly backwards, ladies and gentlemen, we must also glance forwards, and I think you will naturally be impatient with me if I do not select a subject that has a bearing on the stirring times in which we live. Last year Mr. Purce told you the history of the ligature and of some famous military surgeons such as Ambroise Paré. To-day, I propose to introduce to you another character, the sea surgeon; to tell you something of the men who conquered scurvy and the other sea diseases; to pause for a moment whilst we glance at the romantic figure of Thomas Dover; ship's surgeon, privateer, and inventor of the famous powder that still bears his name; and finally to end the story, by paying tribute to the Ulsterman, William Beatty, surgeon to H.M.S. "Victory," who attended Lord Nelson when the latter was fatally wounded at the battle of Trafalgar.

It is not known when the practice of carrying doctors in ships was first introduced. Probably it dates from earliest times. Certainly in the days of Augustus Cæsar, surgeons were carried in the Roman war-galleys to attend to the wounded. Some authorities suggest that the physician St. Luke may, at one period in his life, have been a ship's doctor, and his description of seafaring episodes, such as the shipwreck of St. Paul, supports the contention. But this is pure conjecture. The first actual records of surgeons in the Royal Navy occur in the reign of King Henry VIII, when we learn that the "chif sirurgyon" was paid the sum of 13/4 a month in wages, and other surgeons "beyng most expert to be namyd by the Admyall by the monethe" 10/-. In the year 1588, when the threat of the Spanish Armada drew near, Elizabeth and her councillors took steps to provide medical assistance for the Fleet, and an order appeared in the Acts of the Privy Council which reads as follows:—

> "Whereas a dysease and sickness began to encrease in her Majestie's Navye, for remedie of the dysease and for a staie of further contagion, their Lordships thought meet that some learned and skilful physicians should presently be sent thither; and for that their Lordships hard that good reporte of the sufficiency learning and care of Dr. Gilbert, Dr. Marbeck, Dr. Browne, and Dr. Wilkinson, as they were thought very fytt persons to be employed in the said Navye." . . .4

The Dr. Gilbert referred to is the first important figure that occurs in naval medical history. He was one of the foremost thinkers of his age, and by his researches into the properties of the magnet he may be regarded as founder of the modern science of electricity. Gilbert was physician to Queen Elizabeth and President of the College of Physicians. He took a large part in helping to prepare the first edition ever published of the London Pharmacopœia.

The Royal Navy was comparatively small in Elizabethan times. In the seventeenth and eighteenth centuries it expanded progressively, until at the close of that era it was almost as large as it is at the present day. As the number of ships increased, so did the need for men grow, but the living conditions within the old wooden walls were so bad and the pay so wretched that it was difficult to get seamen to volunteer for the service. The press-gang, as you know, made up the deficiency, but as surgeons were also required, the Company of Barber Surgeons in London was ordered to supply surgeons to the Fleet in times of national emergency. In this way, it is of interest to note, the old Company of Barber Surgeons fulfilled the same rôle as is taken to-day in war-time by the British Medical Association. John Woodall, Master of the Company early in the seventeenth century, records how in 1626 : "there were rumours of war, and our Company of Barber surgeons were then commanded by his Majesties authority to provide sixteen of the best surgeons as the then time afforded, . . . for his Majesties whole service by Land and Sea."[3]

I believe John Woodall has the distinction of being the first doctor in this country to recommend lemon juice as a cure for the scurvy. He made this pronouncement in the year 1617, yet one hundred and seventy-nine years elapsed before the use of lemon juice became general in the Navy and scurvy was abolished. Another of Woodall's achievements was a famous book called "The Surgeon's Mate," which was intended as a guide to young doctors going to sea for the first time.[5] Woodall had been a surgeon himself, and as Master of the Company of Barber Surgeons it was his duty to examine all candidates for warrants as surgeons' mates. It is no wonder that his book had a great vogue, and was regarded as a standard text book by all aspirants to such posts. In the words of its author, it described "the cures of the most frequent diseases at sea, namely, Wounds, Aposthumes, Ulcers, Fistulas, Dislocations, with the true manner of Amputation, the cure of the Scurvie, the fluxes of the belly, the Colics and Illiac Passio, tenesmus and exitus ani, Callentures." All the usual instruments to be found in the sea-surgeon's chest were described, and the author mentions a special spoon-shaped device, which he himself had invented for the treatment of impacted fæces as he remarks delicately —"to serve upon occasion of extreme costiveness." He advised the surgeons to take great care of their instruments, especially their scissors; one pair to be used only for operating, and another set aside and kept specially sharp for the purpose of cutting hair. Among the many curious drugs referred to in this book is "Dens Elephantum," or powdered elephant's tooth, which was said to be of especial benefit in cases of the flux of diarrhœa, and in worms or intestinal obstructions.

Woodall must have been a very lovable character, for in the preface to the last edition of his book, which he completed when he was nearly seventy, he gave the young surgeons some advice that would be as appropriate to-day as it was then. Their first duty, he said, was to God, "who seeth not as man seeth, and who is the searcher of the heart, and knoweth men's thoughts long before, whom if he and I have the Grace to honour with our whole hearts and fear he will direct our ways aright."

It is to be regretted that the young medical men of the seventeenth and eighteenth century who went to sea did not follow these precepts more closely. Drunkenness and debauchery were only too frequent among them, and a contemporary account describes one of those who served in H.M.S. Salisbury in 1784-85 as follows[6]: "Our second assistant surgeon was another wet soul, and coming from the play half drunk went to sleep in an empty cask that was lying on Quigley's wharf, when a squall of wind rolled the cask overboard and poor Andrew Reardon would have been drowned had it not been for the boat-keeper of the cutter. Old Andrew is now dead. He dearly loved grog, and when told new rum was a bad article, he said he didn't care; if it fractured his brain it was all he wanted."

One does not wish to glorify such episodes in the lives of the sea-surgeons, but we may excuse them when we remember that the surgeons, and especially the surgeons' mates, held very inferior positions in the ship's company. They were not commissioned officers and their names were rated in the ship's books beside those of the gunner, the boatswain, and the carpenter.[3, 7]. They drew less pay than these worthies and received infinitely less thanks for the work they did. Not all of them were drunken and dissolute. Many were highly conscientious men, and, though handicapped by lack of knowledge, they fought desperately against the sea-diseases, tending the sick with their own hands. In their practice they might well claim, as indeed one of their number did—"We lose not so many in proportion as candid physicians in London town, that is, one in every five sick, and think that they come off well to boot."[8]

In 1626 the monthly pay of the sea-surgeons was 19/4, not much more than the pay in Henry VIII's time. In 1675 it had risen to thirty shillings. There was in addition a grant of twopence per head for every man in the crew, and fifteen shillings was awarded for every case of venereal diseases treated and cured.[3] But, as the sailors were expected to pay the surgeon this sum out of their own pockets, it can readily be understood that little venereal disease was reported. There was, in truth, little financial benefit to be expected from serving in the Navy as a surgeon in the seventeenth century, and as the Fleet continued to expand and the demand for doctors to increase, some added inducement had to be offered. In 1730, the pay of the surgeons was raised to five pounds a month. There were the other benefits already referred to, and further, if a surgeon was unfortunate enough as to be killed in battle, his relatives were awarded the sum of fifty pounds in compensation. These improved rates of pay had an immediate stimulating effect on recruitment, and the Navy office was soon thronged by an eager crowd of young doctors trying to get warrants. Among these was Tobias Smollett, who has immortalised his experiences as a surgeon's mate in his famous novel "Roderick Random." If some of you have not already read this book, I recommend it to you as a masterly picture of conditions at sea in the early part of the eighteenth century. I know it is the fashion with some writers to decry the episodes related by Smollett in "Roderick Random" as gross exaggerations or caricatures of the truth, but this, I think, is incorrect. Smollett was a medical man himself, he actually served as a surgeon's

mate, and his object, I believe, was deliberate: to draw attention to abuses and irregularities that undoubtedly existed.

Roderick Random, the hero of the novel, found it was not so easy to get into the Navy as he had thought. He learned to his dismay that he had to pass an examination at Surgeon's Hall before he could be accepted, and no doubt you will sympathise with him in his experiences on that occasion. He says:—

> "At length the beadle called my name. . . . I was conducted into a large hall, where I saw about a dozen of grim faces sitting at a long table; one of whom bade me come forward, in such an imperious tone that I was actually for a minute or two bereft of my senses.
>
> "The first question he put to me was: 'Where was you born?' to which I answered 'in Scotland.'
>
> " 'In Scotland,' he said; 'I know that very well; we have scarce any other countrymen to examine here; you Scotsmen have overspread us of late as the locusts did Egypt.' . . . He then proceeded to interrogate me about my age, the town where I served my time, with the term of my apprenticeship; and when I informed him that I served three years only, he fell into a violent passion; swore it was a shame and a scandal to send forth raw boys into the world as surgeons. . . .
>
> "I was scarce able to stand, which being perceived by a plump gentleman who sat opposite to me, with a skull before him, he said, . . . I need not be afraid . . .; then bidding me take time to recollect myself, he examined me touching the operation of the trepan. . . ."

Roderick Random was successful in passing the examination before the Master of the Barber Surgeons' Company, and soon afterwards found himself at sea, enduring the same hardships as were the general lot of surgeons' mates in the eighteenth century, on board a man-of-war. The living-quarters of the junior surgeons, of which there might be three or even four in a large vessel, were situated low down in the ship, beneath the water line on the orlop deck, which lay just above the hold. Here they had a small place amidships about six feet square, screened off by canvas cloths nailed to the bulkheads. The medicine-chests and a rough deal board served them as table and chairs, and at night they slept in hammocks slung from the deck-beams overhead. There were no port-holes, of course, and the only light between-decks was from candle or oil-lanterns, and what little ventilation there was, brought with it the unpleasant odour of the bilge water in the hold. Leaky ships were considered to be much more healthy than dry ships in those days, probably because the salt water, seeping through the hull, tended to purify the bilge water, and because the constant need for pumping prevented it from ever becoming seriously contaminated.

The crew lived on the gun-decks, and at night, when their hammocks were swung, the whole deck was taken up, not more than sixteen inches breathing space being available per man. Usually the congestion was so great that it was customary to berth the men of the starboard and larboard watches in alternate hammocks, so that with one watch on deck, the men of the watch below might have at least two feet space each in which to turn round.[9] Seamen were not the only living inmates of the old wooden battleships. Cockroaches and rats abounded, the latter being a powerful force in spreading disease. Professional rat-catchers were employed to destroy them, and one of these was Thomas Swaine of Greenwich, who proudly

styled himself "Ratcatcher to his Majesties Royal Navy"—surely one of the most curious ranks in the service! His highly diverting pamphlet published in 1773, entitled "The Universal Directory for Taking Alive or Destroying Rats and Mice," showed that he was no mean exponent of his craft. In one ship, the "Duke," a second rate of ninety guns, he says, he accounted for no less than 2,475 rats, but this was an exception. Few ships yielded over 1,000 rats![10]

It requires little stretch of the imagination to understand how prevalent the sea-diseases were under these conditions. Ships, newly commissioned with pressed crews drawn from the jails and from the slums of cities, were swept by the lice-borne scourge of typhus-fever. One physician to the Fleet, William Cockburn, described such an epidemic that attacked it in 1697. H.M.S. "Dutchess" was especially affected. "This ship," he wrote, "was the most sickly in all the Fleet, and had above an hundred sick persons whom I visited every day. The first period of the Fever was in the space of twelve hours commonly, and then they were furiously delirious, they had a great pulse, and died in three days in that number we commonly buried four or five in a morning for the first four days."[11] Ships voyaging to the Americas or to the East Indies suffered heavily from scurvy. The voyage of Commodore Anson, in the year 1740, resulted in the loss of 626 lives from the disease while his ships were on passage from England to the Pacific. In the flagship "Centurion," half of the crew of 506 men were already dead before they reached land, and of the remainder there were scarcely sufficient left to handle the ropes and sails. The description given by Walter of the suffering of the victims of scurvy is one of the best accounts of the disease ever written. He tells how the scurvy caused old wounds to break down again and suppurate—"nay, what is still more astonishing," he says, "the callus of a broken bone, which had been completely formed for a long time, was found to be hereby dissolved, and the fracture seemed as if it had never been consolidated."[9] These observations made two hundred years ago have been considered again by the modern surgeons of 1941, who now insist on the importance of supplying a sufficiency of Vitamin C in all cases of delayed healing of war-wounds. Indeed, history has insufficiently recorded the important part played by the sea-diseases in determining the fate of nations, for as James Lind, "the father of nautical medicine," said: "The number of seamen in time of war, who die of shipwreck, famine, fire or sword, are inconsiderate in respect of such as are destroyed by the sea diseases. . . ."[12] Anson lost 626 men from preventable diseases, but in his attack on the Spanish city of Paita in South America, as a result of which he gained £30,000 in gold, beside other treasure, he lost only one man killed and two wounded.

But, to return to the sea-surgeons. It can be seen that, however poor their position, however unsatisfactory their living conditions and remuneration, they certainly saw plenty of practice. They could not complain of having nothing to do. In frigates and ships of the line the sick-bay, or sick-berth as it was then called, was situated on the upper gun-deck beneath the forecastle, or in the fore part of the hold. The day's work commenced with mustering the sick, a boy making the rounds of the mess-decks, ringing a hand-bell, and "in rhymes composed for the

occasion, inviting all those who had sores to repair before the mast," where one of the surgeon's mates attended to dress them.[13] The surgeon's mates had to perform all the duties now undertaken by sick-berth attendants or nurses, such as preparing food for the sick, making fomentations, spreading plasters, washing towels, administering clysters or enemata, and attending to other sanitary arrangements.

It was in such a hard school as this that James Lind and Thomas Trotter gained the experience which later they turned to such good advantage in insisting on reforms to improve the health of the seamen. James Lind went to sea at the age of twenty-three, and spent nine years there as a surgeon before he returned to Edinburgh to read for his M.D. and Fellowship. His famous works—"A Treatise on the Scurvy," and "An Essay on the Most Effectual Means of Preserving the Health of Seamen in the Royal Navy," appeared some years later, in 1754 and 1757 respectively. These books were a challenge to all who could think logically and who had the welfare of their country at heart. They made it clear that drastic medical reforms must be instituted in the Navy if wars were to be won and communications maintained with far-distant lands. They shed light in ugly corners where men had hitherto seen only darkness, and they prepared the way for the great reforms that were introduced towards the close of the century by Sir Gilbert Blane. To Lind, sailors should ever feel grateful that it was he who first suggested that they should be clothed in clean and warm attire at the public expense; in fact, that they should be granted a uniform; that pure drinking-water should be provided for them; and that lightning conductors should be fitted to the masts of ships. He was the first also to point out that the policy of sending ships abroad with more than their full complement of men was a harmful one. The idea behind this practice, of course, was that deaths being unavoidable, twice the required number of men should be despatched, so that there should be at least half that number left alive and fit for duty when the ship reached its destination! Not only was this policy wrong, Lind said, but it actually led to an increased mortality, to double or treble the figures, than might have been expected had reasonable hygienic precautions been taken.[12]

Thomas Trotter was another who was probably insufficiently rewarded for the great zeal and interest he showed in trying to improve the health of the seamen. He became a surgeon's mate at the early age of 18; served in the West Indies, fought in the action with the Dutch at the Dogger Bank in 1781, went a voyage in a West African slaver, and returned to study under the celebrated William Cullen at Edinburgh in 1788. He found that he knew a great deal more about the treatment of scurvy than did his Edinburgh teachers, and Cullen appears to have acknowledged his especial experience in this subject. He presented a thesis for the M.D. on an unusual subject—one that hitherto had not been attempted at the University: "On Drunkenness," or "de Ebrietate," and this so tickled Cullen's sense of humour that in conferring the degree, he praised the thesis with the words: "Certe non ebrius erat, qui hand dissertationem scribebat!"[14] = the man who wrote this thesis certainly was not drunk!

Trotter was a fiery, quick-tempered character, and did not hesitate to express himself in strong terms when he thought the occasion demanded. The amount of drunkenness in the service appalled him, and he succeeded in getting an order passed in the West Country, which had the effect of reducing the number of grog-shops in Plymouth from three hundred to one hundred.[15] He declares that he was nearly murdered because of this reform. When he was serving at the Royal Hospital, Haslar, he found things far from his liking—"Among other deficiences," he said, "I think a suit of baths the greatest."[3] As Physician to the Channel Fleet under the command of Admiral Earl Howe, he lived on board the hospital ship "Charon," and this was one of the most efficient vessels of its kind in the service. They were afflicted greatly by scurvy in the spring of 1795, and he approached the Admiralty on the subject, after some difficulty succeeding in gaining the necessary permission to purchase supplies of green vegetables and fruit. Within a very short time he had rid the Fleet of the disease. He writes :—"The reader may smile at the idea of a Physician of the Fleet, attending the stalls of a vegetable market or perambulating the country, to calculate the produce; but it never appeared to me below the dignity of the profession, nor did I consider it a mean task to serve the salad with my own hands from the 'Charon's' quarter-deck."[3]

Trotter was a great fellow, a man of strong convictions, and utterly wrapt up in the love of his profession. His appreciation of the good qualities of sailors is shown by his remark : "Was I ever to be reduced to the utmost poverty, I would shun the cold threshold of fashionable charity to beg off a seaman; where my afflictions would never be insulted, by being asked through what follies or misfortunes I had been reduced to penury." He was all for increasing their amusements and for making life more pleasant for them. Music and dancing he recommended particularly. He asks : "Why a regiment is allowed a band more than a ship of the line? Give your tars, O my Countrymen ! their amusements; and while you enjoy yours on shore, remember who they are that give you security !"

One might recount other instances in the heroic vein from the lives of the old sea-surgeons, but I should like to pass to the romantic side of the picture, and tell you something of the exploits of Drs. Thomas Dover and William Beatty.

If we could roll back the years as one may turn over the pages of an old manuscript, we might be able to catch a glimpse of young Thomas Dover as he lay sick abed in the house of his master and teacher—the great Dr. Sydenham. We would notice then an unusual sight for a sick-room of the seventeenth century—the window wide open, no fire in the grate, the patient propped up in bed without shawls or other draperies, the bedclothes laid no higher than his waist, and on the side table by his bed twelve bottles of small beer ![16] The twelve bottles of beer was the diet prescribed by Dr. Sydenham for his patient daily, but, lest you should entertain any false ideas as to its purpose, let me explain that the beer was of exceptionally light quality and that it was acidulated with spirits of vitriol. Dover had the smallpox. He was a student-pupil under Sydenham, as the celebrated Hans Sloane had been before him. If we pass on for twenty-five years, you will see the same Thomas Dover, now a man of forty-six, and a physician with a good practice

of twelve years' standing in the city of Bristol.[17, 18] Nothing very remarkable about him, you would say; he looks as if he had settled down for life. But nothing is further from the truth. At 46, Thomas Dover threw up his practice, invested his life's savings in ships of war, guns and munitions, left his wife and family, and embarked on a privateering voyage around the world. A foolish act, possibly, yet Dover returned a rich man three years afterwards; he had more than doubled his original capital of £3,000,[19] and with this fortune he proceeded to London, where he began practice again, proclaiming not without some reason :—"If travelling be necessary to make an accomplished physician, I am sure that I have travelled more than all the physicians of Great Britain put together."

William Osler, who was fascinated by the personality of this extraordinary man, remarked what a pity it is that we should remember him chiefly because of the powder which bears his name when he had other and greater claims to our remembrance which have been forgotten.[20] The famous prescription for "Dover's powder" is found under the treatment of gout in his book : "The Ancient Physician's Legacy to His Country, Being what he has collected Himself in forty - nine years of Practice." It reads :—

> "Take opium one ounce, Saltpetre and Tartar vitriolated each four ounces, ipecacuanha one ounce. Put the Saltpetre and Tartar into a red-hot mortar, stirring them with a spoon until they have done flaming. Then powder them very fine; after that slice in your opium, grind them to a powder, and then mix the other powders with these."

There you have the original prescription for "Dover's powder"; but the really romantic part of Dover's life was the three years he spent at sea, where, like Podalirius of old, the son of Æsculapius, he combined the duties of physician with those of warrior-leader of the privateering expedition which set out from Bristol on 2nd August, 1708. Captain Woodes Rogers was the commander-in-chief of this force, and there were two ships, the "Duke" and the "Dutchess." They sailed across the South Atlantic to the coast of Brazil, then round Cape Horn up the west coast of South America and returned to England by way of the Pacific, the East Indies, and the Cape of Good Hope. Sword in hand, Dover was in the van of the successful attack they made on the Spanish city of Quaiaquil. The privateers lost only four men in the storming of this rich prize, two killed and two wounded, but the city took its revenge of them later, in traditional fashion, for they were not back at sea long when the plague broke out among them.

Dover, then, had to revert to his original rôle as physician. But he was ready again to fight when they intercepted and captured the Spanish treasure galleon from Manila. Gallant achievements these, but they are dwarfed beside the romance of his rescue of Alexander Selkirk from the desert island of Juan Fernandez.[21] Juan Fernandez was the first place at which the privateers rested after leaving Brazil, and shortly after their arrival, while the ships lay some leagues from the shore, their yards backed in the light sunset airs, Dover took the first boat inshore to explore the island. The act was characteristic of his impetuosity, but it led to the discovery of the castaway, Alexander Selkirk, who on his return to England gave Daniel Defoe the inspiration to write his celebrated "Robinson Crusoe." There has been much speculation as to where Defoe obtained the material for his book,

but a single glance at the description of Selkirk's rescue will supply the answer. When rescued, an eye-witness states, the castaway was wearing goat skins, which gave him an appearance even wilder than the original owners of that apparel. He claimed that he had been left on the island with "only a musket, some powder and balls, a knife, kettle, a bible, and tobacco." He had built himself a hut and had "employed himself in reading, praying and singing psalms, so that, he said, he was a better Christian during his solitude than he had ever been before."[21] The similarity between these statements of Selkirk and the words attributed to Robinson Crusoe leave no doubt that they were one and the same person, and it is this circumstance which Osler thought should ever endear Thomas Dover to us, for he made possible the creation of Robinson Crusoe.

Dover and his merry men, like Commodore Anson, were fortunate in securing their booty without great loss of life, but the naval actions of the past were as a rule bloody affairs in which the surgeon's strength and endurance was taxed to the utmost. The ships engaged each other at point-blank range, and their wooden hulls were capable of withstanding a prodigious amount of battering without sinking. Casualties were heavy, and for every man killed outright there were usually about three wounded. The principal causes of wounding were the jagged splinters of wood, which were sent flying about in all directions by the impact of round-shot or cannon-balls upon the decks and hulls.[22] Other causes of wounds were musket-fire, burns from gunpowder blazing up suddenly, and cutlass slashes sustained in hand-to-hand combat.

The surgeon and his mates saw little of the actual fighting because their place, as already mentioned, was in the cockpit on the orlop deck, far beneath the waterline. Here, a platform or table was made ready for operating, and the chaplain or purser told off to assist. The wounded were carried down to the cockpit by their comrades, and dealt with strictly in order of their arrival. One of the chief difficulties was to distinguish between minor and major casualties, and the dim illumination and overcrowding, the cries of the wounded, and the noise of the cannonading, made this task well-nigh impossible. Robert Younge, surgeon to H.M.S. "Ardent" at the battle of Camperdown in the year 1797, described his experiences. The ship lost 41 men killed and 107 wounded out of a crew of 485, so that his account is not exaggerated.[23] He says :—

"I was employed in operating and dressing till near four in the morning, the action beginning about one in the afternoon. So great was my fatigue, that I began several amputations under dread of sinking before I should have secured the blood-vessels. Ninety wounded were brought down during the action. The whole cockpit, deck cabins, wing berths, and part of the cabin tier, together with my platform, and my preparations for dressing, were covered with them, so that for a time they were laid on each other at the foot of the ladder where they were brought down, and I was obliged to go on deck to the commanding officer to state the situation and to apply for men to go down the main hatchway and move the foremost of the wounded further forward into the tiers and wings, and thus make room in the cockpit. As to the results of his work, Younge remarks, not without

some pride: "I have the satisfaction to say that of those who survived to undergo amputations or to be dressed, all were found next morning in the gunroom, where they were placed in as comfortable a state as possible, and on the third day were conveyed on shore in good spirits, cheering the ship at going away, smoking their pipes, and jesting as they sailed along, and answering the cheers of thousands of the populace who received them in Yarmouth Bay."

Many sea-surgeons recorded their experiences in battle, but none so ably as Sir William Beatty, whose "authentic record" of the battle of Trafalgar is generally regarded as being the most reliable account of that memorable day ever written.[24] Beatty has an especial interest for us in Ulster, for he was reared on the banks of the Foyle, being the eldest son of a Mr. James Beatty, an official of H.M. Customs, in the city of Londonderry.[25] Though the details of his early career are somewhat obscure, it is fairly certain that, like many young men from the County Derry, he was above the average in intelligence and in his capacity for hard work. He received no medical education other than the customary apprenticeship, and entered the Navy first in the humble capacity of a surgeon's mate, gradually rising to the position of surgeon. He was appointed to Admiral Lord Nelson's flagship the "Victory" in 1804, following a Dr. George Macgrath, possibly another Ulsterman, for whom, like William Beatty, Nelson had a real affection. When the Admiral was fatally wounded, he was carried to the cockpit, and it was Beatty, his friend and surgeon, who ministered to him, and who, after his death, took charge of his mortal remains. In his "authentic record," Beatty gives an accurate account of the closing scenes in Nelson's life, of the nature of his wounds, and of the steps he took to preserve his body during the long period that had to elapse before the ship reached England.

You know the story of Nelson's death—It was about a quarter to one in the afternoon when he was wounded. He was walking with Captain Hardy on the quarter-deck, the latter a little in front of him, and was in the act of turning to the right, near the main hatchway,when he was struck on the epaulette over the left shoulder and fell forward on his face. Nelson realised at once that he had been shot through the spine and was fatally wounded, for he lost all sensation and power below a line drawn round the breast. Even so, whilst being carried down to the cockpit, he still had sufficient control of his faculties to notice that the tiller ropes controlling the rudder had been shot away. You may picture the tragedy of the scene. The melancholy procession halting while the stricken Admiral requested a midshipman to inform Captain Hardy of the fact, and to ask him to have fresh ropes rigged immediately.[27] Three hours and a quarter elapsed before Nelson died. He became speechless towards the end. Many diverse accounts are given of his dying declarations, but as Beatty shrewdly remarks in his narrative: "It must occur to the reader that from the nature of the scene passing in the cockpit, and the noise of the guns, the whole of his lordship's expressions could not be borne in mind, nor even distinctly heard, by the different persons attending him." As you all know, however, he retained consciousness sufficiently long to learn from Captain Hardy that a great victory had been won.

Some years before, after the battle of the Nile, Nelson had been presented with a coffin made out of the mainmast of the Orient, flagship of the defeated French fleet. The donor of this curious gift, one Captain Hallowell of the "Swiftsure," wrote :—"Sir, I have taken the liberty of presenting you a coffin made from the mainmast of l'Orient, that when you have finished your military career in this world you may be buried in one of your trophies. But that that period may be far distant is the earnest wish of your sincere friend Benjamin Hallowell."[26] Nelson accepted the gift, but after his death it was decided not to bury him at sea, but to convey his body back to England. Some means of preserving it had to be found, and as embalming it was impossible, Beatty hit upon the ingenious expedient of placing the body in a large cask filled with brandy. This was changed at intervals and fresh spirit substituted, so that even after two months, by which time they had reached England, the corpse was still in a good state of preservation. Before transferring it finally to a leaden shell and placing this inside Captain Hallowell's oak coffin for state burial, Beatty took the opportunity of performing a post-mortem examination and of satisfying himself fully as to the cause of death. His description of the findings proves that he had a sound knowledge of anatomy, and the whole report, I think you will agree, equals any modern record as a model of precision. The exact text of it is as follows :—[23]

"The ball struck the forepart of his lordship's epaulette, and entered the left shoulder, immediately above the acromion process of the scapula, which it slightly fractured. It then descended obliquely into the thorax, fracturing the second and third ribs; and, after penetrating the left lobe of the lungs, and dividing in its passage a large branch of the pulmonary artery, it entered the left side of the spine, between the sixth and seventh dorsal vertebræ, fractured the left transverse process of the sixth dorsal vertebra, wounded the medulla spinalis, and, fracturing the right transverse process of the seventh dorsal vertebra, made its way from the right side of the spine, directing its course through the muscles of the back, and lodged therein, about two inches below the inferior angle of the right scapula. On removing the ball, a portion of the gold-lace and part of the epaulette, together with a small piece of his lordship's coat, was found firmly attached to it."

And now to bring this story to a close. If you have been glancing forwards even as you have been journeying backwards, you will not be at a loss to recognise what bearing the circumstances I have related have on future events. The answer is surely this : that good work can be done under any conditions however adverse; that clinical enthusiasm is not a hot-house plant that thrives only in the luxurious soil of a well-equipped hospital; that original thought requires not peace and calm for its inspiration. Undoubtedly these are advantages, but the sea-surgeons knew them not. They had none of the paraphernalia of modern scientific methods; no special facilities or encouragement. Our fellow-countryman, William Beatty, working in the narrow, dimly-lit spaces between decks in the "Victory"; men like John Woodall, James Lind, and Thomas Trotter—to mention the most outstanding of that gallant company, achieved distinction through their own unaided efforts, their love of the art of medicine, and their diligent pursuit of truth. It is these

qualities which we must strive especially to acquire if we are to be good doctors; as students, practitioners, consultants, specialists, and teachers, we must never lose sight of them, for they are qualities to be cultivated continuously throughout the whole course of our years of practice. I shall end with my favourite, Trotter's words, and you could not do better than to take them away with you to-day :—

"As study and diligence lead to preferment in every department of life, so in our line they have their rewards. We fulfil a most important station in the service of our country; nay of much more importance than we can expect credit for, because many of our best actions must sleep with ourselves, as medical abilities are not to be appreciated by common observers. Yet, this very circumstance is a stimulus to exertion; for it keeps alive the spirit of perseverance, from the hope that merit will at last be discerned and meet with success."

Bibliography.

1. Mayne, Jasper : "Time," *Oxford Book of English Verse,* Oxford University Press, 1925, p. 301.
2. *B.M.J.*, 1940, ii, p. 811.
3. Turnbull, Alexander : *History of the Naval Medical Service from the Earliest Date to the Present Time,* 1904. (Typescript in the Library of the Royal Hospital at Haslar.)
4. Singer, Charles :"Dr. William Gilbert" (1544-1603), *Jour. Royal Naval Medical Service,* 1916, ii, 495.
5. Woodall, John : *Surgeon's Mate; or, Military and Domestic Surgery,* London, 1617.
6. Tunstill, Brian : "Recollections of James Anthony Gardner" in *Anatomy of Neptune,* London, 1936, 513.
7. Hakluyt, Richard : *Principal Navigations, Voyages, Traffics, and Discoveries of the English Nation,* London, Dent, Everyman's ed., vol. 1.
8. Shaw, Sutherland : "(The) Hospital Ship" (1608-1940), *Mariner's Mirror,* 1936, xxii, 422.
9. Walter, Richard : *Anson's Voyage Around the World,* ed. by Laird G. S. Clowes, London, Hopkinson, 1928.
10. *Mariner's Mirror,* 1929, xv, April.
11. Cockburn, William : *Sea Diseases; or, A Treatise of their Nature, Causes, and Cure,* 3rd edition, London, 1736.
12. Lind, James : *Essay on the Most Effectual Means of Preserving the Health of Seamen in the Royal Navy,* 3rd edition, London, 1774.
13. Smollett, Tobias : *Roderick Random,* London, 1746.
14. Cockburn, Alexander : "Biographical Sketch of the late Dr. Thomas Trotter, Physician to the British Fleet," *Edin. Med. and Surg. Jour.*, 1854, 64, 430.
15. Rolleston, Sir Humphry Davy : "Thomas Trotter," *Jour. Royal Naval Med. Service,* 1918, 5, 412.
16. Dover, Thomas : *Ancient Physician's Legacy to His Country, Being What He Has Collected Himself in Forty-five Years of Practice,* 3rd ed., London, 1732.
17. Nixon, J. A. : "Thomas Dover," *Bristol Med.-Chir. Jour.*, 1909, 27, 31.
18. "Thomas Dover, Physician and Circumnavigator," *B.M.J.*, 1913, i, 619.
19. Rogers, B. M. H. :"Woods-Rogers' Privateering Voyage of 1708-'11, *Mariner's Mirror,* 1934.
20. Osler, Sir William : "Thomas Dover, Physician and Buccaneer," in (*The*) *Alabama Student.*

21. Woods-Rogers : *Cruising Voyage Around the World, 1726,* edited by G. E. Mainwaring, Seafarer's Library, 1938.
22. Blane, Sir Gilbert : *Select Dissertations on Several Subjects of Medical Science,* new edition, 1832.
23. Hill, Sir Robert : "Retrospective View of Naval Medical Conditions," *Royal Soc. Med. Proc.,* 1918-'20, 13, War Section, 1-14.
24. Beatty, William : *Authentic Narrative of the Death of Nelson* (copy in the British Museum).
25. Chippingdale, S. D. : "Sir William Beatty," *Jour. Royal Naval Med. Service,* 1915, i.
26. Southey, Robert : *Life of Nelson,* London, 1813.
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OBITUARY

ROBERT HALL, L.R.C.P. & S. EDIN.

Robert Hall is dead. The announcement cast a shadow on hundreds of his former patients, and on many generations of his former students; for he was at once the true friend and adviser of the former, and not only teacher, but friend and counsellor to the latter.

Educated at Queen's College, Belfast, Dr. Hall qualified in Edinburgh (with first place) in 1886, and in that spring was appointed Physician to the Belfast Infirmary, a post which he held for the succeeding fifty years. During this period he instituted many progressive changes, which resulted in bringing the Infirmary to its present up-to-date efficiency. He was the first physician in Ireland to segregate consumptive patients in separate wards apart from other patients, and he was a recognized authority on diseases of the chest. In his presidential address to the Ulster Medical Society (November, 1921), he described for the first time ballooning of the lung and a number of other rare and obscure pulmonary and pleural conditions.

An able physician, with outstanding ability to impart his knowledge, students were always eager to obtain places in his cliniques, and by his skill and sympathetic understanding he earned the affection of his pupils to such an extent that the post of "houseman" under him was regarded as one of the "plums" of newly-qualified medical men.

The remains of the beloved physician were laid to rest in the heart of the Mourne Mountains, which he knew and loved so well, and so passed over that stream from which no traveller returns, the remains of one whose name and memory will remain for ever fresh on all those who came under his just and kindly influence.

21. Woods-Rogers : *Cruising Voyage Around the World, 1726,* edited by G. E. Mainwaring, Seafarer's Library, 1938.
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The Incidence of Bovine Tuberculosis in Humans in Northern Ireland

By James McMurray, M.D.
Institute of Pathology, Queen's University of Belfast

In 1901-1910 a Royal Commission established beyond doubt, by isolating the bovine bacillus from human lesions, that the bovine type was virulent for man. From then onwards the incidence of the bovine bacillus in human lesions has always been an indication of the efficacy of the supervision of the milk supply. The Ministry of Agriculture has always striven to reduce the bovine tuberculosis and thereby the dissemination of tubercle bacilli amongst the population. In Ayrshire, Jordan (1933) estimated that over a period of six years compensation for slaughtering tuberculous animals and the rejection of meat meant a loss to the country of about £750,000 per year. In spite of this, the bovine strain causes two thousand human deaths every year and is responsible for at least four thousand fresh cases annually. These cases have to be treated. And for this, expenses of sanatoria amount to about £500,000 per year. Thus the killing of cattle, the rejection of tuberculous meat, and the treatment of cases of human tuberculosis, in all cost the country one-and a-quarter-million pounds a year. The check on the incidence of bovine tuberculosis amongst humans is thereby essential in the interests of everybody. Such investigations have been carried out in Scotland and England, but never in Belfast. The findings below are the result of such investigation carried out in Belfast, Northern Ireland.

This work consisted in the isolation of strains of tubercle bacilli from specimens of tuberculous material chiefly derived from unselected cases of non-pulmonary tuberculosis. After the primary isolation, each strain was typed. The technique used was similar to that used by Griffith, Blacklock, and others.

If the specimen was uncontaminated and free of other organisms, as frequently in the case of cerebro-spinal fluid, and some urines, it was centrifugalised for twenty minutes at 3,000 in a sterile tube. The deposit was then spread over the surface of the media. However, when material contained extraneous cellular or amorphous material or was contaminated, as with pus from glands, sinus washings, stomach washings, urines, it was mixed and incubated with normal solution of sodium hydroxide. This destroyed contaminating organisms. After centrifugalisation the deposit was neutralised to litmus with eight per cent. hydrochloric acid and spread over the media. At least two tubes of media containing glycerine and two without glycerine were cultured. Early in this work Dorsets egg-media was used in isolation along with Lowenstein media, but finding the Lowenstein more satisfactory for isolation, the use of Dorsets was reserved for subculture and typing work.

The standards used in the recognition of the two types of the tubercle bacillus were based on the rate of growth, character of growth, the reaction of growth to

presence of glycerine, and the virulence of the bacillus for rabbits. The bovine type grew slowly, taking sometimes four to six weeks to appear, whereas the human type only required two to three weeks to produce the same amount of growth. The bovine type gave a finer and smoother growth, which in the early stages was smooth, moist, and clear, whilst the human strain produced a coarser granular dry growth which was sometimes pigmented. When glycerine was added to the media, the growth of the bovine strain was usually inhibited or unaffected, but a human type usually produced a more profuse growth in its presence. When injected into rabbits, a bovine strain produced generalised miliary lesions, whilst ten times the dose of a human culture only gave rise to localised lesions in lungs and in a few of the viscera (see figures 1 and 2). The typical bovine type, because of its poorer non-pigmented growth on glycerine, was labelled 'dysgonic,' and the human with its more profuse and pigmented growth was called 'eugonic.' Atypical strains have further been divided into eugonic bovine, dygonic human, and attenuated bovine and virulent human types.

A thorough investigation therefore means subcultural work and animal inoculation. In the following cases not all were inoculated into rabbits.

Tuberculous Meningitis.

Twenty-five strains: Cerebro-spinal fluid taken from thirty-two patients was examined for the presence of tubercle bacilli by direct examination of a smear of the deposit and by culture of the deposit. Of these, twenty-nine died from tuberculous meningitis, the other three recovered and were not proven to be of tuberculous origin. Twenty-five of the fatal cases gave positive cultural results, and in some of these, those on whom autopsy was done, strains were isolated from tuberculous lung-tissue, hilar or mesenteric glands, or other caseous material. In all instances these strains were identical with those obtained from the ante-mortem spinal fluid. No strains were obtained from cases which recovered from the disease. Some of the cases are worthy of a short description of their interesting features.

C.1.—This was a man aged 35 years who had a primary pulmonary focus in the right upper lobe. This had healed. There was a tuberculous osteitis of the bodies of the thoraco-lumbar vertebræ, psoas abscesses, and epidural abscesses along the spinal canal. This had involved the dura mater and spread through it and produced a tuberculous meningitis. The maximum exudate in this case was on the basal surface of the brain. The tubercle bacilli isolated from the spinal fluid ante-mortem, and post-mortem from psoas pus, were of the eugonic human type. This was an example of how a caseous involvement of the dura mater in the spinal canal may also spread to the arachnoid and set up a tuberculous meningitis.

C.8 was the first dysgonic bovine strain isolated in this series. It was derived from the fluid of a boy aged one year and three months who died after four weeks' illness. This strain appeared as small clear colonies about pinhead size on Lowenstein medium after forty days. Thus after almost six weeks a growth became easily visible to the naked eye. The media in this case contained glycerine. The length of time necessary to produce visible growth was the striking feature about this dysgonic strain.

C.11.—This was the case of a fatal tuberculous meningitis due to the eugonic human type occurring in a breast-fed female child aged two months. The family history gave a clue to the possible source—an uncle who was often in close contact with the child was admitted to another hospital suffering from acute pulmonary tuberculosis and had a positive sputum. This is the youngest case in the series, and it emphasises the need for earlier detection of the cases with positive sputa, say by miniature radiography. In many of these cases spread occurred from advanced cases usually undiagnosed till later or from inadequately treated patients.

C.13 was a bovine strain which was isolated from the fluid from a girl aged 14 years taken on the sixteenth day of illness. This strain was a dysgonic bovine one and was similar in behaviour to a strain isolated at post-mortem. Autopsy showed a primary tuberculous complex in the mesenteric glands. There was no family history of tuberculosis.

C.16 turned out to be a dysgonic bovine strain, and came from the spinal fluid which was taken from a ten-year-old girl on the sixth day of her fatal illness. This girl, one month before the outset of the meningitis, had been admitted to hospital with the history of having had attacks of abdominal pain, occasional vomiting, and tenderness in the right iliac fossa for the previous two months. She was found to have palpable mesenteric glands, a positive mantoux test, and nothing definite in a barium meal X-ray films. The appendix was surgically removed, and found to be short and straight. The mesenteric glands were enlarged. Post-operatively she had a temperature of 103° for a few days, but was discharged symptomless after ten days in hospital. About three weeks later she was re-admitted on the sixth day of a similar type of illness. However, drowsiness and irritability became marked, and the diagnosis of tuberculous meningitis was made. She was removed from hospital contrary to medical advice, and autopsy was not carried out. Findings at operation suggested a primary abdominal tuberculous complex, but whether the surgical interference precipitated a blood-spread to the meninges or not could not be proven.

C.22 was a strain which came from the fluid of a boy aged seven years. This was a eugonic human strain. Autopsy displayed the probable sequence of events as a primary pulmonary tuberculous complex, tuberculous pneumonia, enteritis, mesenteric and hilar adenitis, and tuberculous meningitis. The pulmonary lesion was extensive, but so also was the ulceration of the small intestine and the enlargement and caseation of the mesenteric lymph-glands. The involvement of the latter was so much that it resembled a primary complex. This suggested the possibility of perhaps a mixed infection. The isolation, however, of a strain from the abdominal glands and one from hilar glands, both of which resembled the eugonic human strain recovered from the cerebro-spinal fluid before death, did not confirm this. Only the meningeal strain was inoculated into a rabbit.

The Types of the Bacilli Isolated.

By culture on solid media, twenty-five strains of tubercle bacilli were isolated from the cerebro - spinal fluids of twenty - nine cases of tuberculous meningitis.

Eighteen of these strains turned out to be eugonic on subculturing, and were found to be of low virulence on intravenous injection into rabbits. Thus eighteen were eugonic-human-type strains. The other seven isolated were highly virulent to rabbits, possessed dysgonic characteristics, and were therefore labelled dysgonic bovine strains: so seven out of twenty-five, or twenty-eight per cent., of strains from meningitis were bovine type. Other workers in England and Scotland have found varied results.

	Per Cent. Bovine	Region
Griffith ... ... (1919)	16.0	
Griffith ... ... (1929)	21.8	
Griffith and Munro ... ... (1932)	13.3	Scotland
Griffith ... ... (1934)	21.8	England (151)
	40.5	Scotland (37)
MacGregor, Kirkpatrick, and Craig (1935)	28.0	Edinburgh
Saenz ... ... (1938)	5.4	France
(1940)	28.0	Belfast

Thus it is seen that the incidence of bovine tubercle bacilli in Belfast is the same as in Edinburgh. This is reasonable, because of the similarity in size and milk supply in both places. The figure is lower than that for Scotland, possibly because of the greater incidence of rural cases in the Scottish investigation, and higher than the figure for the industrial centres of England.

TUBERCULOUS MENINGITIS.

Locality	Cases	Human	Bovine	% Bovine
Aberdeen	15	8	7	47
Newcastle-upon-Tyne	13	13	0	0
Sheffield	9	6	3	33.3
Liverpool	10	8	2	20
Bristol	5	4	1	20
Edinburgh (Macgregor) ...	80	61	19	24
Belfast	25	18	7	28

The age of the patients varied from two months to thirty-five years; twenty were under fifteen years old. From these, twenty-seven cerebro-spinal fluids gave the bovine type of tubercle bacillus. Thus of those under fourteen years, thirty-seven per cent. were due to bovine bacilli. Griffith (1929) found a corresponding incidence of thirty-four per cent. bovine.

Of the twenty-five cases, fourteen were males, and these gave three bovine type strains. Just more than half (fifty-five per cent.) of those under fifteen were boys—almost the same figure as Blacklock and Griffin (1935) found. From these eleven boys, three bovine strains were grown. The nine girls under fifteen had bovine bacilli in four of them.

The primary complex of infection was determined clinically or after some of the cases had been examined post-mortem. The site of entry of the tubercle bacillus in five—the dysgonic-bovine cases (8, 13, 15, 16, 17)—was in mesenteric complex. The other two cases were not investigated sufficiently to localise the primary tuberculous complex. Of the remaining eighteen eugonic-human strains, in fourteen there was definitely a primary thoracic lesion, and in the other four a probable

pulmonary complex. Thus out of nineteen cases satisfactorily examined, fourteen were originated in primary thoracic lesions—seventy-three per cent. This is higher than Macgregor and Alexander (1936) found in 333 cases, their figure being sixty per cent. It approximates that of Blacklock and Griffin (1935), however, who calculated the incidence to be 73.9 per cent. in 241 cases of tuberculous meningitis autopsies. They cite higher figures—96.4 per cent. of fifty-five cases by Wangenheim (1928), one hundred per cent. of fifty-four cases by De Villa Genoese (1924).

Primary Cervical Tuberculous Adenitis.

Twelve strains: This has been investigated in sixteen instances with the isolation of twelve strains of tubercle bacilli. Of these twelve, two cultures were bovine in type—a bovine incidence of about 16½ per cent. One of the bovine strains was obtained from a swab: cultures grew both from the medium, which was rubbed with the swab, and from that which received debris from the swab after it had been treated with alkali and neutralised. This child drank milk received from a local country farmer.

Admittedly the number of cases investigated is small, but it is a definite indication that the bovine bacillus is a potential infecting source in and around Belfast. The figures are much lower than others, possibly because milk is more strictly supervised and satisfactorily produced than it was in 1932 or 1929. Probably if figures were made now of those districts, the bovine bacillus might not be so commonly found.

Genito-Urinary Tuberculosis.

Cases of tuberculosis of kidneys, bladder, seminal vesicles, and epididymis were investigated in twenty-two strains. From these, three were dysgonic-bovine strains —an incidence of 16.6 per cent.

Tubercle bacilli were seen on direct smear in fifteen of those eighteen cultured, and in two cases which did not give a positive culture. Only two cases were under fourteen years old, and of these one gave a bovine strain.

CERVICAL ADENITIS.

Strain	Sex	Age	Specimen	Examination Direct	Culture	Cultural Type
A13	F	2 1/12	Pus	Pos.	Pos.	Eugonic human
A20	F	6	Pus	Pos.	Neg.	—
A32	M	12	Pus & gland	Pos.	Neg.	—
A33	M	39	Gland	Neg.	Neg.	—
A37	F	36	Pus	Pos.	Neg.	—
A41	M	1½	Pus	Neg.	Pos.	Eugonic human
A43	F	56	Pus	Pos.	Pos.	Eugonic human
A48	F	16	Pus	Pos.	Pos.	Eugonic human
A54	M	1 3/12	Pus	Pos.	Pos.	Eugonic human
A61	F	6½	Pus	Pos.	Pos.	Eugonic human
A69	M	10	Pus	Neg.	Pos.	Eugonic human
A72	F	31	Glands	Neg.	Pos.	Eugonic human
A74	F	6	Swab	Pos.	Pos.	Dysgonic bovine
A99	F	9	Gland	Pos.	Pos.	Dysgonic bovine
A104	F	19	Pus	Neg.	Pos.	Eugonic human
A105	F	20	Pus	Neg.	Pos.	Eugonic human

Thus in twelve strains from all ages 16.6 per cent. were bovine: in eleven from patients under sixteen years, two, or eighteen per cent., were bovine. The incidence varies very much in districts. Corresponding figures are:—

Munro in Scotland	6 per cent.	
Griffith (1929)	70 „ „	Scottish
Griffith	60 „ „	English
Blacklock (1932)	64 „ „	English
Saenz	1.4 „ „	(France)
Belfast (1940)	16.6 „ „	

The bovine incidence in Belfast corresponds closely to Giffith's figure (1929) of 17.4, when he found four of that type amongst twenty-three strains. Alston and Griffith found in forty-two strains, thirteen of the bovine type—30.9 per cent.

Bone and Joint Tuberculosis.

Seventeen cases were investigated, varying in character from a mastoiditis at the age of two months to a wrist-joint disease at seventy-four years. This resulted in the isolation of strains of bacilli in twelve cases; of these, two were regarded as bovine in type (A52 and A56)—16½ per cent. One case (A21) did not clinically resemble tuberculosis, and gave negative results direct and on culture. So out of sixteen cases of tuberculosis, twelve gave positive cultures, and in these twelve, six showed bacilli on direct examination. Two cases positive direct did not grow on culture.

BONE AND JOINT TUBERCULOSIS.

Strain	Sex	Age	Localisation	Specimen	Direct	Type
A1	M	16	Hip-joint	Pus	Pos.	Eugonic human
A7	F	2/12	Mastoiditis	Sequestrum	Pos.	—
A21	F	9	Elbow-joint ...	Pus	Neg.	—
A22	M	21	Knee-joint	Inguinal gland	Neg.	Eugonic human
A36	M	20	Knee-joint	Pus	Neg.	Eugonic human
A52	M	2	Mastoid, skull, phalanges ...	Pus	Pos.	Dysgonic bovine
A56	M	56	Hip-joint	Pus	Pos.	Dysgonic bovine
A62	F	18	Ankle-joint ...	Pus	Neg.	Eugonic human
A63	F	74	Wrist-joint ...	Pus	Pos.	Eugonic human
A65	F	40	Vertebræ	Urine	Pos.	Eugonic human
A68	M	50	Elbow-joint ...	Pus	Pos.	Eugonic human
A81	M	56	Ankle-joint ...	Pus	Neg.	—
A90	M	65	Vertebræ	Pus	Pos.	—
A97	F	22	Saciccliac joint ...	Sinus washing	Neg.	—
A103	M	23	Hip-joint	Pus	Neg.	Eugonic human
A106	M	27	Knee-joint	Pus	Neg.	Eugonic human
A107	F	34	Ankle	Pus	Neg.	Eugonic human

In Belfast, therefore, the incidence of the bovine strain is about 16½ per cent. in

TABLE 1.

POSITIVE RESULTS.

No.	Sex	Age	Lesions	Source of Infection	Specimen	Direct	Type	
							Culture	Virulence
C1	M	35	Healed pulmonary; vertebral osteitis; epidural abscess; T.B. meningitis (T.M.) ...	?	C.S.F.	Pos.	Eugonic	Human
C3	F	10	Pulmonary, miliary, T.M. ...	?	Pus	Pos.	Eugonic	—
C4	M	14	Pulmonary T.M.	Family history denied	C.S.F.	Pos.	Eugonic	Human
C5	M	10	Pulmonary T.M.	Parental	C.S.F.	Neg.	Eugonic	Human
C6	M	7	?Pulmonary hip-joint ...	Parental	C.S.F.	Neg.	Eugonic	Human
C8	M	1¼	?Abdominal T.M.	Parental ?	C.S.F.	Neg.	Eugonic	Human
C9	M	19	Pulmonary T.M.	Unknown	C.S.F.	Pos.	Dysgonic	Bovine
C10	M	10½	?Pulmonary T.M.	? Family	C.S.F.	Pos.	Eugonic	Human
C11	F	2/12	Pulmonary T.M.	? Family	C.S.F.	Pos.	Eugonic	Human
C12	F	5½	?Pulmonary T.M.	Family	C.S.F.	Pos.	Eugonic	Human
C13	F	14	Abdominal, miliary T.M. ...	?	C.S.F.	Pos.	Dysgonic	Bovine
				Milk	P.M. C.S.F.	Pos.	Dysgonic	—
					Gland	Pos.	Dysgonic	—
C14	M	19	Pulmonary T.M.	?	C.S.F.	Pos.	Eugonic	Human
C15	M	1¾	Mesenteric T.M.	Milk	C.S.F.	Pos.	Dysgonic	Bovine
C16	F	10	(Primary) abdominal T.M....	?	C.S.F.	Pos.	Dysgonic	Bovine
C17	F	10	Mesenteric T.M.	Milk	C.S.F.	Pos.	Dysgonic	Bovine
C18	F	19	Mesenteric T.M.	?	C.S.F.	Pos.	Eugonic	Human
C19	M	13	?Primary T.M.	?	C.S.F.	Pos.	Dysgonic	Bovine
C20	M	7	Pulmonary T.M.	Family negative	C.S.F.	Pos.	Eugonic	Human
C21	F	9	T.M.	?	C.S.F.	Pos.	Dysgonic	Bovine
C22	M	7	Pulmonary, secondary mesenteric T.M.	Family history	C.S.F.	Pos.	Eugonic	Human
				±	(P.M. gland),	Pos.	Eugonic	—
					hilar, abd.	Pos.	Eugonic	—
C23	F	6	?Pulmonary T.M.	Family	C.S.F.	Pos.	Eugonic	? Human
C24	M	3	Pulmonary T.M.	Family	C.S.F.	Neg.	Eugonic	Human
C27	M	12	Pulmonary T.M.	Family	C.S.F.	Pos.	Eugonic	Human
C29	F	2	Pulmonary T.M.	?	C.S.F.	Pos.	Eugonic	Human
C30	F	19	Pulmonary T.M.	Family	C.S.F.	Pos.	Eugonic	Human

TABLE 2.

NEGATIVE RESULTS.

CASE	SEX	AGE	LESIONS	SPECIMEN	DIRECT	CULTURE	REMARKS
C2	F	3	Mastoiditis, meningeal irritation ...	Sequestrum C.S.F.	Pos. Neg.	Neg. Neg.	—— Not T.M.
C7	M	8	Tuberculous meningitis ...	C.S.F.	Neg.	Neg.	Removed from hospital C.T.A.
C25	M	24	Pulmonary T.M.	C.S.F.	Pos.	Neg.	——
C26	F	57	Pulmonary T.M.	C.S.F.	Pos.	Neg.	——
C28	F	4	P.U.O., meningeal irritation	C.S.F.	Neg.	Neg.	No tuberculosis in a guinea-pig
C31	F		T.M. & primary unknown	C.S.F.	Pos.	Neg.	——
C32	F	12	Lymphatic meningitis ...	C.S.F.	Neg.	Neg.	No lesions in a guinea-pig

TABLE 3.

GENITO-URINARY TUBERCULOSIS.

STRAIN	SEX	AGE	LOCALISATION OF DISEASE	SPECIMEN	MICROSCOPICAL EXAMINATION	CULTURE	CULTURAL TYPE
A3	F	43	Cystitis	C.S.U.	Pos.	Pos.	Eugonic human
A5	F	30	Renal	C.S.U.	Pos.	Pos.	Eugonic human
A14	M	12½	Renal	C.S.U.	Pos.	Pos.	Eugonic human
A17	M	36	Renal	C.S.U.	Pos.	Pos.	Eugonic human
A23	M	23	Renal	C.S.U.	Pos.	Pos.	Eugonic human
A25	F	29	Bilateral renal	C.S.U.	Pos.	Pos.	Eugonic human
A26	M	36	Seminal vesicle	Urine	Neg.	Pos.	Eugonic human
A27	F	32	Renal	Urine	Pos.	Pos.	Eugonic human
A29	M	21	Renal	Urine	Pos.	Pos.	Eugonic human
A30	M	30	Epididymitis	Pus	Pos.	Pos.	Eugonic human
A34	M	9	Renal	Urine	Pos.	Pos.	Dysgonic bovine
A40	M	29	Renal	Urine	Neg.	Pos.	Eugonic human
A44	M	18	Renal	Urine	Pos.	Pos.	Eugonic human
A56	M	30	Epididymitis	Pus	Pos.	Pos.	Eugonic human
A83	M	19	Renal	Kidney	Pos.	Pos.	Eugonic human
A88	F	21	Renal	Pus	Pos.	Pos.	Dysgonic bovine
A93	F	25	Cystitis	C.S.U.	Pos.	Nil	—
A94	F	66	Renal	C.S.U.	Pos.	Nil	—
A95	M	33	Renal	C.S.U.	Pos.	Pos.	Eugonic
A100	M	32	Renal	C.S.U.	Neg.	Neg.	—
A102	M	13	Renal	C.S.U.	Neg.	Neg.	—
A108	M	19	Renal	C.S.U.	Neg.	Pos.	Eugonic human

bone and joint tuberculosis. Corresponding figures elsewhere show much variation :

Griffith ... ... (1929)	26	per cent.	England } all ages
	18	„ „	Scotland }
Blacklock ... ... (1932)	34	„ „	
Griffith and Munro (1932)	66.7	„ „	East Scotland (under 15)
Fraser ... ... (1912)	61.2	„ „	Edinburgh
Miller ... ... (1937)	10	„ „	
Belfast ... ... (1940)	16½	„ „	

Miscellaneous.

Only five sputa were examined : in all of them bacilli were seen direct, and were grown on culture. All of these were eugonic-human strains.

Commentary.

From eighty-eight specimens strains of tubercle bacilli were grown from seventy-two, and of these seventy-two, the bovine type was present in fourteen of them.

	No. Cases	Direct Positive	Cultures	Bovine	% Bovine
Meningitis	29	25	25	7	28
Cervical	16	10	12	2	16½
Genito-urinary	22	17	18	3	16½
Bone and joint	16	8	12	2	16½
Pulmonary	5	5	5	...	0
	88	65	72	14	19.4

Thus about eighty per cent. of the tuberculous specimens yielded bacilli on culture. This is somewhat higher than Blacklock's figure (1932), using guinea-pigs. Seventy-two strains produced fourteen of bovine type, or a proportion of 19.4 per cent. Relative figures for other districts are best appreciated in tabular form.

PERCENTAGE BOVINE.

Source	Tuberculous Meningitis	Cervical Adenitis	Genito-Urinary	Bone and Joint	Pulmonary
Griffith					
English	27.3	45.7	17.4	18.2	—
Scottish	40.5	70.6	30.9	26.6	—
Blacklock					
Scottish	49.1	64.3	—	34.6	2.7
Saenz					
French	5.4	14.0	1.1	0	½
Belfast					
Northern Irish	28.0	16.6	16.6	16.6	—

The finding of 19.4 per cent. of bovine tubercle bacilli in Belfast suggests that milk dissemination of the bacilli is still high. Thus the typing of the tubercle bacillus is a final check on the efficacy of the control of tuberculosis in milking cows.

BIBLIOGRAPHY.

BLACKLOCK, J. W. S.: "Tuberculous Disease in Children, its Pathology and Bacteriology," 1932, *M.R.C. Special Report,* No. 172.
BLACKLOCK, J. W. S., and GRIFFIN, M. A.: *Jour. Path. and Bact.,* 1935, 40, 489.
GAGGENHEIM, A., and FICKELSTEIN, M.: *Amer. Rev. Tub.,* 1939, 39, 397.
GRIFFITH, A. S.: *B.M.J.,* 1914, i, 1171.
GRIFFITH, A. S.: *Jour. Path. and Bact.,* 1916-17, 21, 54 and 329.
GRIFFITH, A. S.: *Jour. Path. and Bact.,* 1919, 23, 129.
GRIFFITH, A. S., and MUNRO, W. T.: *Lancet,* 1928, i, 384.
GRIFFITH, A. S.: *Jour. Path. and Bact.,* 1929, 32, 813.
GRIFFITH, A. S., and MUNRO, W. T.: *Jour. Path. and Bact.,* 1930, 33.
GRIFFITH, A. S., and MUNRO, W. T.: *Jour. Path. and Bact.,* 1932, 35, 271.
GRIFFITH, A. S., and SUMNER, G. T.: *Lancet,* 1933, i, 875.
GRIFFITH, A. S., and MUNRO, W. T.: *Lancet,* 1933, i, 384.
GRIFFITH, A. S.: *Jour. Path. and Bact.,* 1933, 36, 164.
GRIFFITH, A. S.: *Lancet,* 1934, i, 1382.
GRIFFITH, A. S., and SMITH: *Lancet,* 1935, ii, 1339.
GRIFFITH, A. S., and SMITH: *Lancet,* 1938, i, 739.
GRIFFITH, A. S.: *Tubercle,* 1937, 18, 529.
FRASER, J.: *Jour. Exp. Med.,* 1912, 16, 432.
MACGREGOR, A. R., and GREEN, C. S.: *Jour. Path. and Bact.,* 1937, 45, 613.
MACGREGOR, A. R.; KIRKPATRICK, A. J. R.; and CRAIG, W. S.: *Ed. Med. Jour.,* 1935, 42, 138.
MACGREGOR, A. R., and ALEXANDER, W. A.: *Ed. Med. Jour.,* 1937, 44, 561.
MILLER, A. R.: *Ed. Med. Jour.,* 1937, 44, 260.
MUNRO, W. T.: *Lancet,* 1928, i, 384.
MUNRO, W. T.: *Ed. Med. Jour.,* 1930, 37, 141
SAENZ, A., and CHEVE, J.: *Compt. rend. de Soc. debiol.,* 1937, 125, 934.
SAENZ, A.: *Comp. rend. de Soc. de Biol.,* 1938, 127, 269.
SAENZ, A., and EISENDRATH, D.: *Annal. Inst. Pasteur,* 1932, 49, 608.

A NATIONAL HOSPITAL SERVICE

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The objects of the Trust and the progress made during the eighteen months of its existence are set forth in some detail. Interesting memoranda are included on (a) General Medical and Hospital Policy, (b) Accident Services, (c) Pathological Services.

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Fig. 1

Rabbit—received 0.1 mgm. human strain. Killed ninety days later. Note: localised lesions in lungs, few small foci in kidney and liver.

Fig. 2

Rabbit — received 0.01 mgm. bovine strain. Died forty days later.
Note: Generalised miliary lesions in all organs.

BIBLIOGRAPHY.

BLACKLOCK, J. W. S.: "Tuberculous Disease in Children, its Pathology and Bacteriology," 1932, *M.R.C. Special Report,* No. 172.
BLACKLOCK, J. W. S., and GRIFFIN, M. A.: *Jour. Path. and Bact.,* 1935, 40, 489.
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GRIFFITH, A. S.: *Jour. Path. and Bact.,* 1933, 36, 164.
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Incidence of Pathogenic Staphylococci in a Group of Northern Ireland Munition Workers

By EILEEN O. BARTLEY, M.D., D.P.H.
Department of Bacteriology, Institute of Pathology, Queen's University, Belfast

DURING the winter of 1939-40 a number of men working in a munition factory attended at the skin department of the Royal Victoria Hospital, Belfast, where they were seen by Dr. Ivan McCaw.

They were suffering from staphylococcal lesions of the skin of the arms and thighs, which varied in severity from mild folliculitis with acne to severe boils. During work, the skin of the parts affected was in continuous contact with mineral oil and the clothing of the men examined was soaked with oil.

Since it has been shown by Hart (1937), Hallman (1937), McFarlane (1938), and others that carriers of pathogenic staphylococci are relatively common in groups of normal persons, an investigation was begun somewhat on the lines of that carried out by Gillespie, Devenish, and Cowan (1939) to determine the carrier-rate among the men exposed to oil, and if possible to discover the reason for the high incidence of skin lesions among these workers.

Cultures were made from the nasal cavities, normal skin, and any existing lesion, whether slight or severe. The swabs were planted on blood-agar, incubated at 37°C. for twenty-four hours, and then kept under observation from four to seven days on the bench. Likely colonies of creamy or golden colour were picked off for investigation, and if no creamy colonies were present, white colonies were taken instead. In all the colonies selected the presence or absence of hæmolysis on blood-agar was noted.

Three hundred and sixty-three strains were isolated and studied. They were tested for their ability to ferment mannite and to coagulate human plasma. Most strains were also tested for the presence of alpha hæmolysin. These tests were carried out according to the methods described by Gillespie, Devenish, and Cowan (1939) in their investigation on a group of medical students attending University College Hospital, London.

Since it has been established by Cruickshank (1937), Chapman et al. (1938), and Gillespie, Devenish, and Cowan (1939), that the presence of coagulase is a reliable criterion for the pathogenicity of staphylococci, the test for this substance was used to determine the pathogenicity of the strains investigated.

In biological properties, the results attained were similar to those published by Gillespie, Devenish, and Cowan (1939). One albus strain of nasal origin gave a positive coagulase test, and so may be regarded as a potential pathogen. Devenish and Miles (1939) classified coagulase positive albus strains in the aureus group.

Eighty-two men submitted themselves for examination. Their ages ranged from 15 to 62 years, but most were 19 to 20 years old.

TABLE I.

	No.	Percentage Nasal-carriers.	Percentage Skin-carriers.	Percentage of both Nasal- and Skin-carriers.	Total Percentage of Carriers.	Percentage of Persons with Staphylococcal Lesions at some time.
Students U.C.H. ...	159	43.4	19.5	13	50	41
Munition Workers N.I. ...	82	21.9	20.7	6	50	75

The results are seen in table I, and are there compared with those of Gillespie, Devenish, and Cowan (1939). The percentage of nasal-carriers (21.9) is much less in the Northern Ireland group, and the percentage of skin-carriers (20.7) is approximately the same in both groups. The total percentage of persons carrying pathogenic staphylococci is also the same in both groups (50), but there is a striking difference between the groups in that the percentage of persons who had had active staphylococcal lesions was much higher in the Northern Ireland group than in the U.C.H. group.

TABLE II.

Type of Lesion.	No.	Percentage Nasal-carriers	Percentage Skin-carriers	Total Percentage of Carriers
None	20	25	15	40
Mild folliculitis or pustules ...	46	19.5	19.5	50
Boils	16	25	18.7	75

The relationship of skin-carrier rate to nasal-carrier rate is given in table II. For comparison, the figures given by Gillespie, Devenish, and Cowan (1939) in the U.C.H. group are included in the table. This shows that the percentage of persons carrying pathogenic staphylococci in both nose and skin is almost identical in the two groups. In the Northern Ireland group, however, a higher percentage of those who are free from staphylococcal infection of the nose are skin-carriers of potentially pathogenic staphylococci.

TABLE III.

Nasal-carriers		Skin-carriers		Percentage
	No.	+	−	Positive
	69	20	49	29.0
+ ...	*18*	*5*	*13*	*28.7*
	90	11	79	12.2
— ...	*64*	*12*	*52*	*18.7*
	159	31	128	19.5
Total ...	*82*	*17*	*65*	*20.7*

The figures in italics refer to the group of Munition Workers N.I.

The relationship between the carrier - rates and the presence and severity of infection is shown in table III. The types of lesion were classified as "None," "Mild Folliculitis or Pustules," and "Boils." In the "Mild" group the infection of the skin was superficial, while in the group with "Boils" there was a definite and usually severe type of infection. The results obtained show that the percentage

of carriers in nose and skin combined was greatest in the group of men suffering from boils, and least in those who had no lesions.

Since seventy-five per cent. of the Northern Ireland group had had at some time a staphylococcal infection, as compared with forty-one per cent. in U.C.H. students, it is evident that some factor other than the carrier-rate must be concerned in the production of lesions. The most obvious were the lack of personal cleanliness among the men examined, and the oiliness of the skin and clothes. Almost all the men were dirty both in their persons and clothes, while lack of ordinary cleanliness existed apart from the oiliness. They seldom bathed, and it was evident that their clothes were not washed often enough. As a result, in practically all of the men who were exposed to oil during work, the skin was covered with a film of oil continuously. The mechanical effect of this was to cause blocking of the sweat-ducts and to prevent the removal of organisms by sweating or friction.

Samples of the machine-oil used were examined bacteriologically, and were found to be sterile. An experiment to determine whether staphylococci could live in the oil was carried out. Inocula of 500 million, 50 million, 5 million, and 500,000 organisms per c.c. of oil were made and kept at room temperature. Subcultures on ordinary agar were made from the oil immediately after inoculation and after twenty-four, forty-eight, and ninety-six hours and at intervals up to seventeen days. Growth was obtained from the sub-cultures made immediately after the oil was inoculated. In twenty-four hours the oil which had had an inoculation of 500,000 cocci was sterile, and in forty-eight hours all samples of oil except that containing 500 million cocci were sterile. After seventeen days, that sample of oil containing 500 million cocci per c.c. was still capable of producing a good growth. Sub-cultures from controls on ordinary broth all gave good growth after seventeen days observation, except that with the 500,000 inoculum, which was then sterile. It is therefore possible that infection could be spread by means of the oil, but since no micro-organisms of any kind were obtained from the specimens taken from the machinery, it is unlikely that the oil acted as a vehicle for the spread of the organisms.

Summary.

The results of the investigation show that the total carrier-rate was the same in the Northern Ireland munition workers as in the U.C.H. students (i.e., 50 per cent.), but that the proportion of skin-carriers to nasal-carriers was increased in the Northern Ireland group. This was due to the fact that, although the skin-carrier rate was approximately the same in both groups, the nasal-carrier rate in the N.I. group (21.9 per cent.) was about half that found in the U.C.H. students (43.4 per cent.).

The high incidence of skin infections in the group described is probably due to the high incidence of potentially pathogenic staphylococci on the workers' skin, aggravated by lack of personal cleanliness and by the mechanical effect of the presence of a constant film of machine oil.

Results obtained from the bacteriological examination of the oil show that it is unlikely that it acts as a vehicle of infection.

REFERENCES.

1. CHAPMAN, G. H.; BERENS, C.; NILSON, E. L.; and CURCIO, L. G. (1938): *Jour. Bact.*, 35, 311.
2. CRUICKSHANK, R. (1937): *Jour. Path. Bact.*, 45, 295.
3. DEVENISH, E.A., and MILES, A. A. (1939): *Lancet*, 1, 1088.
4. GILLESPIE, E. H.; DEVENISH, E. A.; and COWAN, S. T. (1939): *Lancet*, 11, 870
5. HALLMAN, F. (1937): *Proc. Soc. Exper. Biol.*, N.Y., 36, 789.
6. HART, D. (1937): *Arch. Surg.*, 34, 874.
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Studies from the Institute of Pathology

A CASE OF DIPHTHERIA

CASE XI—A3054.

CLINICAL HISTORY.

THE patient, a boy of 13 years, had had pain and swelling in the throat for about three days. This was thought to be mumps. He had also a "septic" finger. The advice of a chemist was sought, and treatment was advised. Later a medical practitioner who was summoned in haste could only pronounce life extinct.

POST-MORTEM.

The body is that of a male child of normal development for the age given. Subcutaneous fat is rather scanty, but the skin is clean and there is no evidence of ill-treatment or neglect. There is an insignificant paronychial infection of one finger of the left hand. Oedema and abnormal pigmentation are absent. Lymph-glands in the axillæ, groins, and neck are not palpable. The neck is greatly swollen, and on incising it, considerable quantities of œdema fluid pour from the subcutaneous tissues and from between the fascial planes. Rigor mortis is established, and post-mortem lividity is normally distributed.

Serous cavities.—The right pleural cavity is obliterated by dense fine adhesions. The left contains only a slight excess of clear yellow fluid. The pericardial sac is normal, and there is no excess of free fluid and no adhesions in the peritoneal cavity. Only a few petechial hæmorrhages are visible beneath any of the serosa.

Left lung.—This is uniformly congested throughout. In the middle of the upper lobe, situated well below the pleura, there is a circular area of caseation almost 1 cm. in diameter. It is closely related to a much smaller but better calcified area slightly nearer the pleura. No satellite tubercles are visible. Changes in the hilar lymph-glands are not evident.

Right lung.—A small and entirely calcified nodule is present in the upper lobe. No lesion, old or recent, can be demonstrated in relation to the pleura, which is

REFERENCES.

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Right lung.—A small and entirely calcified nodule is present in the upper lobe. No lesion, old or recent, can be demonstrated in relation to the pleura, which is

roughened owing to the presence of many adhesions. Scattered throughout the lung, especially in the lower lobe, there are small areas of consolidation. The largest is about the size of a pea. They are a darker red than the surrounding lung, and in them the alveolar pattern is obscured. The mucosa of the bronchi of both lungs is deeply congested.

Heart.—This is of normal size and the normal proportions are observed between the different chambers. The epicardium shows only a very few small petechial hæmorrhages. The ventricular muscle is soft, pale in colour, and its pattern slightly indistinct. An area beneath the endocardium of the left ventricle is thickly beset with multiple subendocardial hæmorrhages. These lie chiefly in the septal wall, and extend over a distance of almost 4 cm. and to within 2 cm. of the aortic valve. They tend to become confluent in some areas. The endocardium is smooth and the valves are normal and free from vegetations or thickenings.

Large and small intestine.—These present no abnormality, and the lymph-glands of the mesentery are not enlarged.

Stomach and duodenum.—The mucosa is congested and shows numerous petechial hæmorrhages, but no ulceration or membrane formation. The wall of the stomach is slightly thickened and œdematous.

Pancreas.—The acinar tissue is well defined.

Liver.—This is normal in size, and the lobular pattern is preserved in spite of some pallor. There is no congestion or fatty change.

Spleen.—The capsule is wrinkled and the organ is only very slightly enlarged. In the dark-red pulp, fibrous trabeculæ are more prominent than malpighian bodies. Some minute white areas are surrounded by narrow zones, which are a darker red than the neighbouring pulp.

Kidneys.—Both are normal in size, and the cortex and medulla preserve their normal relations and architecture. The capsular surfaces are smooth. Ureter, bladder, and prostate are normal.

Suprarenals.—The cortex and medulla are well preserved and the glands are not unduly congested.

Aorta.—There are a few small yellow subendothelial fatty streaks in the intima running in the long axis of the vessel.

Tongue, soft palate, pharynx, larynx, and trachea.—These have been removed together. There is very marked swelling and œdema, especially of the soft palate, uvula, and pillars of the fauces. A finger can scarcely be passed from the mouth into the pharynx. By cutting through the faucial pillars, the palatine tonsils, the epiglottis, and pharynx are displayed (fig. 1). Here also there is marked œdema, and some epithelial folds are obliterated. In the larynx there is narrowing, but not complete occlusion of the air-way. A rough greyish membrane, necrotic in parts, extends over the soft palate, the faucial pillars, the tonsils, the epiglottis, and the front of the pharynx downwards into the pyriform recess. In the larynx it is more patchy, but it covers the false vocal chords and extends almost to the true vocal cords and ventricles. Over the soft palate and almost necrotic uvula the membrane is firmly adherent. The tonsils show grey necrotic, but not hæmorrhagic,

tissue continuous with the membrane and filling the whole tonsillar fossæ. On the epiglottis, and especially in the larynx, the membrane is most easily detached, and on the dorsum of the epiglottis it exists only as irregular islands. The lower part of the larynx and trachea are extremely congested, but no membrane and no ulceration is present.

Deep structures of the neck.—Considerable quantities of œdema fluid escape as subcutaneous and fascial planes are incised, and it is extremely difficult to be sure of its original distribution. Most of the deep muscles of the neck appear somewhat swollen. Lymph-glands are very little enlarged, but are redder than normal. The thyroid is normal.

HISTOLOGICAL EXAMINATION.

Left lung.—The alveolar walls are only slightly congested and the alveoli contain no exudate. Surrounding the area of caseation in the upper lobe there is a very abundant and dense fibrous tissue reaction with some infiltration by small mononuclear cells. Very occasional follicles composed of large mononuclear leucocytes and sometimes giant cells are present. Extending outwards from this there are several similar, but smaller, areas of caseation surrounded by less fibrous tissue. The lesion is nowhere in an active phase.

Right lung.—In very small, but irregularly-shaped, areas the alveolar walls are indistinct, or even necrotic. They separate alveoli packed with red blood-cells and with white blood-cells often present only in their normal hæmic proportions. A hæmorrhagic exudate containing polymorphonuclear leucocytes is present in some of the bronchi, but there is no fibrinous exudate in relation to the epithelium.

Heart.—The heart-muscle fibres show no demonstrable degeneration, and no free fat is present. There are no abnormal cells in the interstitial tissue, and the mitral valve is normal.

Liver.—The columns of liver cells around the central vein of each lobule appear somewhat narrowed from œdema between them and the sinusoidal wall. Nuclear and cytoplasmic detail is preserved, and fatty change is negligible.

Spleen.—The pulp is moderately congested. The large clear cells of the germinal centres, or secondary nodules, are indistinguishable. Instead, there are pale, eosinophilic masses containing strands of fibrin and showing pyknotic nuclei and chromatin fragments. These are situated to one side of the arteriole, but sometimes surround it and often occupy the greater part of the malpighian body. Around the larger of them the splenic sinusoids are congested (fig. 2).

Kidney.—A few of the glomeruli may appear slightly cellular, but polymorphonuclear leucocytes and fibrin thrombi are absent. The convoluted tubules show an excessive degree of cloudy swelling. Droplets of free fat are absent. In the lumen of some tubules, especially the collecting tubules, there is homogeneous eosinophilic material, but no red blood-cells. Similar material can be seen in a few glomerular spaces. No mononuclear cells are present in the interstitial tissue.

Suprarenals.—These are normally developed and show no degeneration.

Thymus.—This does not show any distinctive lesion. Mononuclear-cells are not degenerating. Uninuclear eosinophil-cells are fairly common.

Lymph-glands.—The glands least altered are those in the mesentery of the bowel. In the centres of some of the ill-defined follicles there is some proliferation of large eosinophilic mononuclear-cells. Some of these appear to contain nuclear debris, others show early necrosis. In the glands of the lung-hilum, the large endothelial-cells of the sinusoids are prominent, but these show no necrosis. Glands draining the affected area show infiltration of their peripheral sinuses and pericapsular tissues by polymorphonuclear leucocytes. The greater part of these glands is occupied by large follicles, which are nearly all necrotic and infiltrated by polymorphonuclear leucocytes.

Aorta.—There is no necrosis or disruption of the elastic pattern of the media.

Soft palate, tonsil and related structures.—The epithelial-cells of the greater part of the area covered by membrane are almost entirely necrotic. They are replaced by a reticulate or fenestrate arrangement of fibrin (fig. 3). Nuclear debris can sometimes be recognised in the spaces of this, but retrograde changes such as multiplication of nuclei, vacuolation of cytoplasm, and fragmentation of nuclei can only very rarely be distinguished. A fine fibrin reticulum around the epithelial cells —the so-called fibrinous membrane—is very rarely seen. In many areas the necrotic-cells, epithelial and inflammatory, have fused intimately with the fibrin, which thus appears as a reticulum with thick broad beams enclosing small spaces—the so-called hyaline reticulum. In many other areas there is necrosis and liquefaction, and the membrane has been destroyed or its pattern disrupted. In the tonsils, extensive necrosis has occurred in relation to much of the lymphoid-tissue. These necrotic areas are infiltrated by polymorphonuclear leucocytes, but the escape of fibrin or large numbers of red blood-cells is not a prominent feature. Inflammatory cells, polymorphonuclear leucocytes and mononuclear leucocytes, and red blood - cells contribute to the formation of the membrane, but it is rarely possible to identify them. In the subcutaneous tissues, strands of varying thickness permeate between connective tissue-cells, and around the wall of many small blood-vessels hyaline fibrinoid change is frequent (fig. 4). Oedema fluid, especially marked in the uvula, separates connective tissue elements and the underlying muscle-fibres. Inflammatory-cell infiltration is remarkably slight even at only a little distance beneath the lesion, and muscle-cells may show preservation of architecture, even when still separated by much œdema fluid. Bacterial aggregates are present on the surface, but do not extend into the underlying tissues.

Bacteriology.

Corynebacterium diphtheriæ of the *mitis* type were isolated from the larynx and lung. The spleen was sterile.

Anatomical Diagnosis.

Diphtheritic infection with extensive membrane formation on palate, tonsils, faucial pillars, anterior wall of pharynx, epiglottis, and upper larynx.

Oedema of deep structures of the neck.

Hæmorrhagic pneumonia right lung.

Necrosis of proliferating secondary germinal follicles of lymph-nodes and spleen.

Petechial hæmorrhages submucosa of stomach and in epicardium : subendocardial hæmorrhages septal-wall of left ventricle.

Toxic changes in kidney with albuminous material in tubules.

COMMENTARY.

The lesions described have resulted from an infection by C. diphtheriæ. No treatment was given, but essentially similar lesions have been seen in other cases receiving antitoxic serum late in the disease.

In diphtheria, local and general lesions can be distinguished. The local lesion, or diphtheritic membrane may be small, but a toxin, or probably several toxins, carried from it by the blood may damage many different tissues of the body. The disease-process may be further complicated by other coincident infections, especially streptococcal.

The toxin production may vary somewhat with different strains of infecting organism. As the toxin is of importance locally, as well as generally, in that it damages the epithelium, and is thus instrumental in the production of the membrane, it is of very special significance. The development and severity of the disease depend on the resistance of the host. We are fortunate in that we are able to measure the extent to which the body can neutralise the toxin, and thus approach nearer to a measure of the patient's immunity than is possible in almost any other infection where immunity usually depends on complex mechanisms, resulting in the destruction of bacteria. When a somewhat arbitrary amount of toxin injected intradermally is neutralised and no local damage results (Schick test negative), we can conclude that the body is unlikely to be damaged by C. diphtheriæ.

When the infection has become established, the site as well as the extent of the membrane exerts an influence on the outcome. This is reflected in the observation of Mallory (1908) on 148 cases dying while the distribution of the membrane was evident. Clinically, by far the greater number of cases show membrane on the tonsils, soft palate, or uvula. Primary involvement or spread to the larynx is rare. In these 148 fatal cases, membrane was situated on the larynx in 86, on the tonsils in 74, in the trachea in 73, on the epiglottis in 67, in the bronchi in 44, on the mucous membrane of the nares in 43, on the soft palate, including the uvula, in 15, in the œsophagus in 12, on the tongue in 9, in the stomach in 5, in the duodenum in 1, on the vagina in 2, on the vulva in 1, on the skin of the ear in 1, and on the conjunctiva in 1. Such figures also serve to show how widespread the lesions of diphtheria may be. Infection of a granulating wound is very rare, but may be mentioned.

A diphtheritic membrane is in no way specific for infection by C. diphtheriæ. A similar false membrane can follow the action of many bacteria, such as those of the dysentery group and streptococci. Many poisons, including alkalies and acids and the salts of heavy metals, also lead to damage of overlying cells and excite the outpouring of fibrin between the epithelial cells, or where epithelium is absent around the leucocytes and necrotic connective tissue.

The formation of the membrane can be explained as due to the escape of fibrin precursors from the blood. Fibrin is deposited around and on the surface of

epithelial cells. Later it also entangles inflammatory cells in a dense feltwork. It is probable that organisms first grow on the epithelial surface and produce degeneration of the epithelium by their toxins. Necrotic tissue and fibrin later provide a favourable medium for growth and toxin production. Bacterial growth in the tissue deep to the membrane is slight, and spread in the adjacent tissues or throughout the body is very uncommon. McLeod *et al.* (1939) failed to recover the organisms from the spleen in any of their fifty-one cases. Deep infection of the lung is, however, common.

In the trachea and bronchi the lesions are largely the result of direct spread. A stratified cylindrical epithelium lines the lower part of the larynx, the trachea, and bronchi. If involved, these cells, surrounded by their fibrin feltwork, separate early from their basement membrane and before the membrane becomes continuous with the fibrin meshwork in the underlying connective tissue. They thus contrast with squamous epithelium. This process is perhaps aided by the mucous-gland secretions which are poured out between the membrane and the wall. Large pieces of membrane may thus separate. In a child of 2½ years (A2744), a large and perfect membranous cast of the trachea and of the large bronchi was removed by suction two days before death.

Pneumonic changes in the lung are frequent, and this is especially so when the larynx or trachea are involved. Broncho-pneumonia, terminal hypostatic and hæmorrhagic forms are recognised. A variety of organisms may be found. Mallory in bacterial examination of the lungs of 104 cases found the diphtheria bacillus in 69, streptococcus pyogenes in 67, staphylococcus aureus in 37, and the pneumococcus in 10. Mixed infections are thus common, and their significance is often doubtful. The hæmorrhagic form of pneumonia is of special interest. In patchy areas of dark-red colour, the alveoli are packed with red blood-cells in about the normal hæmic proportions. There may be a purulent bronchiolitis and an inflammatory exudate in other areas, but the part played by infection is very uncertain. McLeod *et al.* describe three examples : one with gravis and two with intermedius infections. The lesions were evident in the present case, and in another case (A2427), also due to a mitis infection, they were even more marked. Areas of terminal pneumonia resulting from aspiration of gastric contents may sometimes be difficult to distinguish microscopically from such hæmorrhagic lesions. They are usually more irregular in outline and softer, and the larger bronchi also contain aspirated gastric contents.

A great variety of lesions result from the circulating toxin. In the spleen and lymphoid tissue generally, it may be difficult to find reticulum cells in the secondary germinal centres which have not undergone a fibrinoid necrosis. This is usually preceded by some proliferation of these large mononuclear cells. A similar change has been seen not infrequently in scarlet and rheumatic fever and other severe infections. Significant changes in the liver were absent in the present case. Where present they are in no way specific. Necroses may be focal, but are often central. They occur fairly frequently. In the kidney, cloudy swelling of tubular epithelium and some prominence of the endothelial cells of the glomeruli may occur along with

albuminous material in the tubules. The heart may feel soft and look rather pale, but changes in the heart-muscle cells are difficult to appreciate. In few organs is it more difficult to infer function from structure. Hyaline degeneration is very rare and occurs late in the disease. Aggregates of mononuclear cells may be present in cases which also show acute interstitial nephritis. Limited to the heart, they may be of some significance. They have been seen in a patient (A2399) who developed triple rhythm and ascites and who died in the third week. However, the sudden cardiac failure of diphtheria appears to result from a peculiar instability of the vasomotor system.

The petechial submucosal hæmorrhages in the stomach and the occasional sub-epicardial hæmorrhages were very terminal events, and probably resulted from toxic damage to capillary endothelium. The large sub-endocardial hæmorrhages resemble those sometimes seen to follow acute and severe shock and hæmorrhage, such as post-partum hæmorrhage. The possible rôle of an acute vasomotor collapse, or of asphyxia, is uncertain and cannot be excluded in the present case. There is little to suggest a connection between the scattered and usually central lesions of hæmorrhagic pneumonia and a symptomatic purpuric condition, where subpleural hæmorrhages are usually prominent.

An interstitial infiltration by mononuclear cells, lymphocytes, and especially plasma cells has been described frequently in streptococcal infections and noted sometimes in diphtheria (Brody and Smith, 1936). It is often best seen in the kidney between the tubules, and especially around the arcuate veins in the boundary zone of the kidney. It is then described as an acute interstitial nephritis. Esentially similar lesions occur in the suprarenals, in the portal tracts of the liver, often along the trabeculæ of the spleen, and in the heart. Brody and Smith have produced evidence that when these lesions occur in diphtheria, they always result from the secondary streptococcal infections so frequent in these cases. In spite of the claim of Smith et al. (1940), to produce these lesions experimentally in animals by streptococcal fractions, this cannot yet be completely accepted. It is of importance, however, to emphasize the frequency of secondary streptococcal infections. Even in well-managed fever hospitals there is considerable and undesirable sharing of streptococcal populations. One carrier of Group A streptococci in the ward-population may be the cause of many complications. A case (A2684) dying from acute bacterial endocarditis, due to a Group A streptococcus seven weeks after the onset of diphtheria, is of interest.

Much work in recent years has been concerned with the differentiation of the infecting organism into gravis, intermedius, and mitis strains, and the correlation of this with the clinical severity of the disease. This differentiation should be made not on one criterion, but on several. The colony form on a special medium containing tellurite; the nature of the growth in broth and the fermentation of starch are the more important criteria. Undoubtedly since 1927, a more severe form of diphtheria has been prevalent in Europe and in some British cities. McLeod *et al.* have found in fifty-one post-mortem examinations on typed cases, that in general there is rather less local membrane with gravis or intermedius infections,

but that the inflammatory reaction often involves the tonsil in hæmorrhagic and fibrino - purulent necrosis, and penetrates deeper into the neighbouring tissues. Death is more frequently due to toxic effects than to respiratory obstruction.

At present no pathological picture can be regarded as in any way specific for this type of infection. O'Meara (1940, 1941) has suggested that in gravis infections a second toxin or "substance B" is produced in relatively larger amounts. This is not neutralised by the available anti-toxins, and facilitates the extension of the local lesion and the continued discharge of toxin into the blood. This substance B is probably concerned with alterations of the intercellular tissues, and facilitates the spread of infection. It is probably not identical with the spreading factor extracted by McClean and Duran-Reynals from other bacteria and the testes (see McClean, 1941).

It may be confidently predicted that the therapeutic problems presented by these hypertoxic cases will be overcome. But this case may serve as a reminder that no therapy can reduce the death-rate unless cases are presented early to those competent to make an early diagnosis. It is especially important that these persons should at all times take every necessary step to make such a diagnosis. Especially in children, the examination of the throat and tonsils can never safely be omitted.

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REFERENCES.

1. BRODY, H., and SMITH, L. W. (1936): *Amer. Jour. Path.*, 12, 373.
2. MALLORY, F. B. (1908), in *Nuttall and Graham - Smith's* "The Bacteriology of Diphtheria," Cambridge, p. 82.
3. McCLEAN, D. (1941): *Lancet*, i, 595.
4. McLEOD, J. W.; ORR, J. W.; and WOODCOCK, H. E. de C. (1939): *Jour. Path. Bact.*, 48, 99.
5. O'MEARA, R. A. Q. (1940): *Jour. Path. Bact.*, 51, 317; (1941): *Lancet*, i, 205.
6. SMITH, L. W.; MORGAN, I. M.; and MUDD, S. (1940): *Amer. Jour. Path.*, 16, 87.

REVIEW

FEARS MAY BE LIARS. By John M. Ryle, M.D. Pp. 95. George Allen & Unwin. Price 3/6.

THE physician turned philosopher is always interesting, and whether one agrees with his philosophy or not it has behind it much observation of man that renders it valuable. This little book contains a series of six essays—Death and Dying, Life and Living, Two Kinds of Immortality, Pain, Fear, and Philosophia Medici—obviously stimulated by the times in which we live. They represent a courageous investigation of our beliefs about ourselves—an investigation based as far as possible on the application of reason. Dr. Ryle's philosopy has something positive to contribute. "The art of living here and now, rather than the habit of contemplation of a wish-fulfilment heaven, may be the nobler thing to cultivate." "Science must learn that it has direct social and moral as well as cultural and academic functions." "A little biology, a little physiology, a little pathology could have helped them so much. Dogma and prayer and ritual have helped them so little."

One is stimulated to fresh thought and endeavour by the author's rationalism—not a deadly materialism, but an inspired and inspiring materialism.

The weakest of the essays is the last, where we feel that the subject "Philosophia Medici" has suffered artistically and practically from its polemic presentation. There is much to stimulate and inspire in the book, and we recommend its perusal to all who are prepared to think.

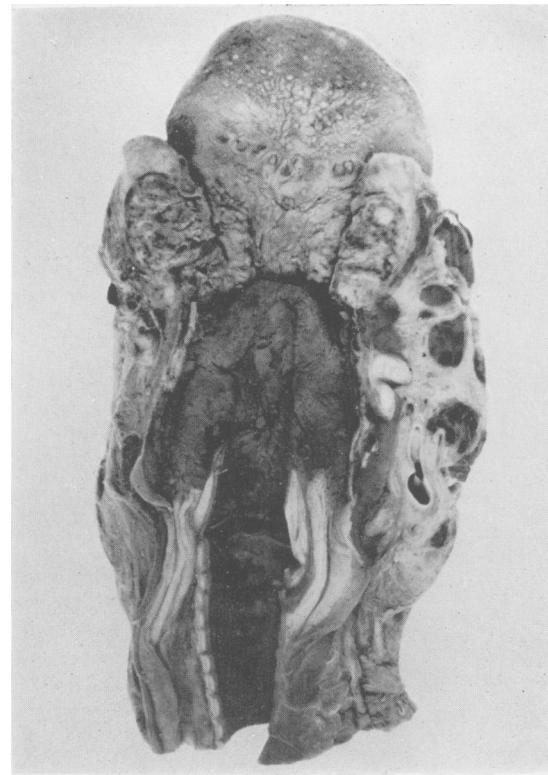

Fig. 1

The diphtheritic membrane is present on the tonsils, where there is underlying necrosis, and on the pharynx and larynx.

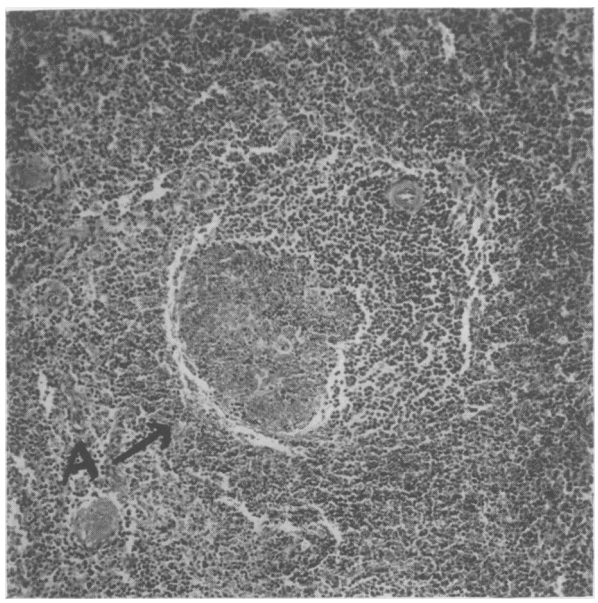

Fig. 2

Malpighian body in spleen, showing nuclear debris lying among the breaking-down large mononuclear cells of the secondary germinal centre. The immediately adjacent sinusoids (A) are packed with red blood cells.

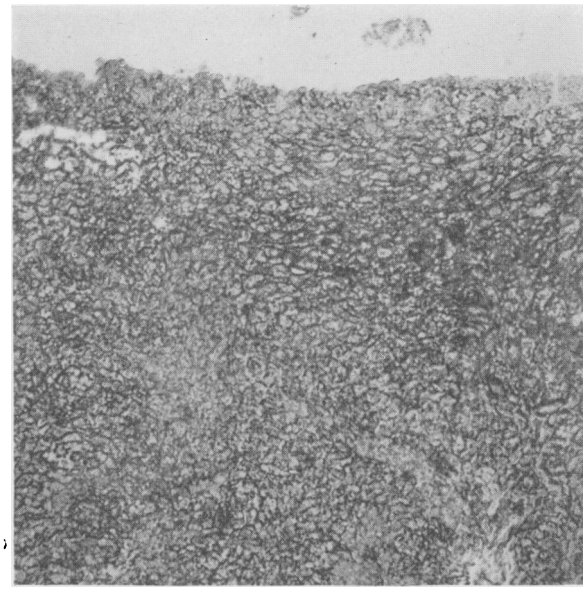

Fig. 3

Diphtheritic membrane showing a fairly thick and coarse fibrin reticulum. The spaces contain degenerated cells and are small and rounded. (Mallory's phosphotungstic acid hæmatoxylin.)

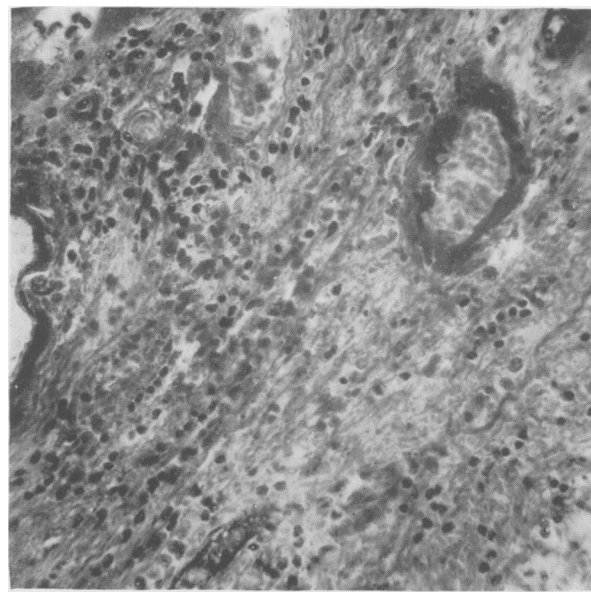

Fig. 4

Hyaline fibrinoid change in the wall of small vessels in the submucosal tissue. Some inflammatory cells are also present in the œdematous tissue. (Mallory's phosphotungstic acid hæmatoxylin.)

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