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THE ULSTER MEDICAL JOURNAL



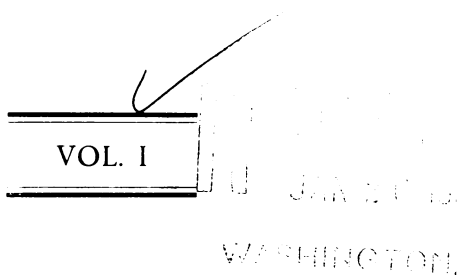
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THE ULSTER MEDICAL SOCIETY

The Ulster Medical Journal



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1932

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FORTHCOMING MEDICAL MEETINGS

January	7	Ulster Medical Society	-	-	-	-	8.30 p.m.
„	14	British Medical Association, Ulster Branch	-	-	-	-	1.15 p.m.
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affections died, their nearest and dearest ceased to be more to them than the man in the street.

To the casual observer it might appear that there are few conditions, if any, that could be confused with this malady. Unfortunately, however, this is not the case, and such widely differing diagnoses as nephritis, hyperpiesis, brain tumour, myocarditis, disseminated sclerosis, anæmia, are amongst the labels that some of these unfortunates carry about for years, much to their detriment. These diagnoses have no doubt been made owing to too much stress being laid on certain symptoms of the disease. One is apt to forget that these people are liable to dizziness, staggering gait, headaches, serious eye changes, epileptiform attacks, slowness of speech; that they may have a coincident secondary or even primary anæmia and a myocardium suffering as a result of such. Their pulse may be above the normal rate, they may have varying amounts of albumin in their urine, and the blood pressure may be raised. The writer can recall two cases of myxœdema who passed through typical attacks of acute nephritis, and another who has the blood-picture typical in every respect of pernicious anæmia.

One of the saddest features of these cases is that eye changes have often supervened before the disease is diagnosed, and that they do not yield to treatment like the rest of the condition. To see the large, pale, sodden tongue, that can barely be protruded, transform itself within a few weeks into the normal red active muscular organ, is to witness one of the miracles of medicine. But to see the eyes that can no longer read, and to which faces are but at best a blur, is to weep over the limitations of medical science.

One last point that should be stressed as strongly as early diagnosis is the question of adequate dosage in treatment. It is a common thing to find that the case has been correctly diagnosed, and thyroid prescribed, but that the patient after taking the prescription for months, and feeling no benefit, has eventually discarded it. It is somewhat difficult subsequently to persuade these people that the line of treatment was perfectly correct, and must be recommenced in an intensified form. A case that the writer has seen had faithfully continued an inadequate dose for some years, and has unfortunately irreparable eye damage now.

Like every other serious condition that doctors encounter, myxœdema demands both early diagnosis and adequate treatment in order to obtain results which rank amongst the most dramatic in medical science.

FORTHCOMING PAPERS

It is hoped to publish in the April number of THE ULSTER MEDICAL JOURNAL amongst other papers, the following:—

“Recent Advances in Calcium Metabolism in Relation to Clinical Medicine,” by Professor W. W. D. Thomson.

“Diagnosis and Treatment of Ante-Partum Hæmorrhage,” by Mr. C. H. G. Macafee.

“Heart Block following Coronary Thrombosis,” by Dr. S. B. Boyd Campbell.

“The Functional Divisions of the Large Intestine,” by Dr. R. H. Hunter.

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THE ULSTER MEDICAL JOURNAL

PUBLISHED QUARTERLY ON BEHALF OF THE ULSTER MEDICAL SOCIETY

Vol. I

1st JANUARY, 1932

No. 1

APOLOGIA

THE Ulster Medical Society has published for many years, at irregular intervals, the papers and discussions which took place at its meetings. This publication was known as "The Transactions," and it filled a useful part in the activities of the Society. The Fellows and Members, however, consider that the time has come when a journal should be issued at stated intervals to contain these papers and discussions. At the last general meeting of the Society, it was decided to inaugurate the publication of a new quarterly medical journal to replace the irregularly-issued Transactions. It was thought that such a journal would increase the usefulness of the Society, and help in some small way medical advance in Northern Ireland. The matter was left in the hands of the Council, and THE ULSTER MEDICAL JOURNAL is the result.

On first thoughts it would appear that there is a sufficiently large number of medical journals already before the profession; but the Ulster Medical Society thinks that there is room for yet another: one that will represent the outlook of modern medicine particularly suited to the needs of the busy practitioner of Northern Ireland: one which will place before him in a succinct manner the latest methods of diagnosis and treatment in medicine, surgery, obstetrics, and gynaecology.

The Editorial Board is frankly proud of its charge. But although the journal is financed and controlled by the Ulster Medical Society, it is only by the support of the general practitioners of Northern Ireland as a whole that the Board can hope to carry on the work of publication. If this support is forthcoming, then THE ULSTER MEDICAL JOURNAL will extend its sphere of service, but if support is denied, its existence must end.

There are two ways in which the practitioner can assist this Journal. He can subscribe to the Journal directly, or indirectly by joining the Ulster Medical Society, each member of which will receive a free copy of the Journal on publication, for THE ULSTER MEDICAL JOURNAL is really *his* Journal. The practitioner can help in another way: he can contribute to its pages. From time to time he sees cases of

unusual interest in the practice of his profession, and short accounts of such cases will be welcomed by the editor for publication. Space is to be devoted to the problems of the Panel Medical Service, and here the point of view of the practitioner will receive its place, and the help of the general body of practitioners will be welcomed. The editor will also be glad to receive reports of the various medical societies in different parts of Ulster. Many points of interest are raised at their meetings, but at present there is no outlet for their publication. THE ULSTER MEDICAL JOURNAL hopes to supply a place for their publication. But here again it depends on the secretaries of the societies concerned to supply these reports. The editor is already in communication with a number of societies, but if there is any secretary who has not yet received an invitation from the editor, he is asked to send up a report of the meetings in time for the next number of this Journal.

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Observations on Enlargement of the Mesenteric Lymph-Glands

By S. T. IRWIN, M.CH. (BELF.), F.R.C.S.ED.

President, Ulster Medical Society, 1931-32

THE lymphatic glands of the abdomen may be called the silent members of a closed corporation, their silence accounting for the difficulty in describing their physical characteristics and accounting for their clinical behaviour. Surgical textbooks still fail to throw much light on their clinical and pathological obscurity. Many practitioners look upon the diagnosis of mesenteric lymphadenitis as a "refuge of the destitute," used in haphazard fashion by those who cannot make up their minds between appendicitis, cholecystitis, duodenal ulcer, and the half-dozen other diseases which frequent the right side of the abdomen. Abdominal problems, more than those of any other speciality, still depend for their solution on the old-fashioned clinical methods. True enough, we can invoke the aid of the radiologist and the biochemist, and from them gain valuable information, but this information lacks the impersonal precision of such instruments as the ophthalmoscope and the cystoscope. And in the end we must make our diagnosis and outline our treatment on what we have discovered by means of our own five senses. In regard to acute abdominal emergencies, owing to the supremacy of the time factor, this is most true, and in the category of acute emergencies, especially in childhood, lymphadenitis occupies an important place. No surgeon is long engaged in this type of work before he discovers his own personal limitations, and recognises the wide gap often existing between the premises afforded by the clinical examination and the inference which aims at being an accurate diagnosis. And yet the circumstances are often so critical that success or failure in diagnosis may entail the life or death of the patient.

The mesenteric lymph-glands, like those found elsewhere in the body, show a wide range of pathological change. Let me begin by defining the types of enlargements with which I propose to deal. The greater number of these enlargements must, for obvious reasons, be excluded altogether. For example, I shall exclude all the primary glandular enlargements such as those occurring in Hodgkin's disease, lymphosarcoma, and in the leukemias. I shall make passing reference only to the enlargements associated with gastric and duodenal ulcer, and obvious ulceration of the small and large intestine, as well as those which ordinarily follow malignant disease. In short, my paper will be limited to a discussion of those mesenteric enlargements which are not directly associated with obvious naked-eye diseases of the alimentary tract. Though I shall refer and refer frequently to tuberculous lymphadenitis, I shall exclude those enlargements from this cause which produce a palpable swelling, or which, by adhesion to gut or otherwise, produce the usual symptoms and signs of either acute or chronic intestinal obstruction. In the title of the paper I have purposely avoided the term lymphadenitis, because I am not sure whether in the present state of our knowledge we are justified in assuming that all the types are definitely of inflammatory origin.

My interest in this subject was first aroused in 1913, when I was asked to go forty miles into the country to operate on a girl of 14, said to be suffering from acute appendicitis. Her illness had already lasted four days. It began with a sudden attack of right-sided abdominal pain. There was no rigor, and though she felt sick she had not vomited. The pain, severe at first, had abated slightly as the result of poultices and sedatives, but was still present. On examination, the girl was plump and healthy-looking. There was no previous history of any illness. She had a clean tongue, and her breath was not foul. Her pulse was 80, and her temperature 100.4°F. The abdomen was slightly distended, but moved freely on respiration. There was no increase of the pain on flexion or extension of the thigh. There was definite tenderness and a suspicion of rigidity in the right iliac fossa, but no phlegmon was detected. Rectal examination was negative. The kidneys were not palpable, and the urine was normal.

The picture seemed to me a familiar one, and most surgeons would, I think, have been prepared to acquiesce in the diagnosis already made. Some might even have gone further and said that the appendix was lying towards the ileum, that it was perforated, with the perforation protected by a mop of omentum, and from the condition of the pulse that the peritonitis was localised and subsiding.

Operation, however, revealed a disappointingly normal appendix, but proved the attack to be due to the presence of a mass of caseating glands in the ileo-caecal region, one of which had ruptured and produced a small area of localised peritonitis.

The condition of glandular enlargement is a common one, probably the most frequent alternative, in this country at all events, to disease of the appendix, but rupture of a gland, on the other hand, is remarkably rare.

Since then I have been carefully observing the glandular enlargements in the abdomen, not only in connection with other lesions such as ulceration of the stomach, duodenum, and intestine generally, but more especially those cases in which no lesion can be found sufficient to account for the patients' complaints.

The recognition of enlarged mesenteric lymph-glands as a definite clinical entity apart from an associated enteritis or peritonitis, began in this country in 1905. In that year Edred Corner reported two cases in patients of the ages of 14 and 6 years respectively. In one he removed a large mass from the right iliac fossa, and in the other a similar mass from the left hypochondrium. Both masses proved on subsequent examination to be enlarged and caseating lymph-glands of tuberculous origin. In both these cases the swellings were easily palpable through the abdominal wall, there was no obvious lesion of the intestine, and no involvement of the peritoneum. In 1908 Corner reported several similar cases, in some of which no palpable mass could be felt before the abdomen was opened.

In 1912, Floderus in Germany reported a series of one hundred cases collected from the literature, all verified by operation. He commented upon the absence of a palpable lump beforehand, and on the fact that a correct diagnosis was made in only seven of the one hundred cases. He considered tubercle to be the cause of all the cases, but quotes Payr, who, while agreeing with this view in regard to the glands found in the ileo-caecal area, threw doubt on the smaller and more discrete glands found elsewhere in the mesentery.

The most complete contribution to the literature of the whole subject from a clinical point of view was made in 1918 by the late H. W. Carson, who reported in great detail fifty cases from his own practice. This article is still the classic on the subject. He considers the main cause of the disease to be tuberculous infection

of the bovine type conveyed by milk, predisposed to by disease of the appendix or other form of sepsis.

In 1921, Struthers agreed that these cases are tuberculous, that the glands rarely go on to suppuration, that the most frequent site is the ileo-cæcal angle. He thinks the disease is commoner in some districts than others, notably where tuberculous disease of the cervical lymph-glands is also common. The correct diagnosis is rarely made before operation, the appendix is generally normal, and the prognosis is always favourable. He found 22 cases of lymphadenitis to 187 of appendicitis.

In 1923, Freeman pointed out that the condition is not *tabes mesenterica*, though the latter may be the final stage of it. The glands are "small, soft nodules." He quotes Huesser as being opposed to the then theory that all the cases are tuberculous. Huesser submitted forty specimens obtained at operation to histological, bacteriological, and inoculation, as well as naked-eye, investigation. He found twenty-five of these negative to tubercle, and makes the important statement that where the result was positive for tubercle, the result would have been the same had judgment been based on naked-eye examination alone. That is, that where caseation or calcification was present, the condition was tubercle, but not otherwise.

McFadden discussed the subject in a paper of considerable interest and importance in 1927 before a meeting of the Ulster Medical Society. He reviewed thirty-seven cases of his own, considered the cause to be the bovine tubercle bacillus, and made the original suggestion that the acute symptoms in these cases were due to an acidosis, and advanced the theory that the acute abdomen of children, said to be the result of acidosis, is really due to an underlying mesenteric lymphadenitis.

In the same year (1927) Bell agreed with the prevailing opinion that nearly all the cases are of tuberculous origin, that on x-ray examination shadows of calcified glands are frequently seen, and that during an attack a leucocytosis of twelve to fifteen thousand may be expected.

In 1928, papers were published by Rendle Short and by Jennings Marshall. Both classify mesenteric adenitis into simple and tuberculous.

Freeman, in a second paper in 1929, and Walter Alvarez in 1930, both from America, lay stress on the views of Heusser. Freeman entitling his paper "Non-specific Enlargement of Mesenteric Lymph-Glands," and stressing the clinical evidence for regarding some of the cases as due to causes other than tubercle; Alvarez, directing attention to the resemblance of the condition to appendicitis, expresses his firm belief in Adami's work, and finds in it support for the view that these cases of lymphadenitis are due to absorption of low-grade infections, not only tubercle bacilli, but other micro-organisms as well.

ANATOMICAL CONSIDERATIONS.

The lymph vessels of the abdomen group themselves around the main arterial trunks, and the glands are found at intervals along these channels. It is, in my opinion, important to remember that normally in all cases the largest glands in any group are proximal in position.

The stomach, duodenum, and gall-bladder drain through a scanty series of lymph vessels and glands into a proximal group which lies around the cœliac axis.

The small intestine has a plentiful supply both of vessels and glands. The latter number 150-200. They lie between the layers of the mesentery. They form three tiers—the smallest (para-intestinal) close to the intestinal wall are the most numerous. The intermediate tier in position is also intermediate in size and numbers. The proximal tier lies at the origin of the superior mesenteric artery, and is composed of relatively few but large glands. These superior mesenteric glands converge as they pass upwards. They drain the whole of the small intestine with the exception of the last six inches of the ileum.

The last six inches of the ileum, the ileo-cæcal valve, the appendix, and the cæcum, are drained by the ileo-cæcal group of glands. They are relatively plentiful, numbering from ten to twenty or more. They form a chain lying behind the parietal peritoneum, are bounded laterally by the cæcum and ascending colon, and on the medial side and below by the root of the mesentery. Normally the lymph flows upwards by the side of the ileo-colic artery, but though this artery is a branch of the superior mesenteric, the ileo-cæcal glands do not discharge into the superior mesenteric glands. There is a lymph shed between these two adjacent sets of lymph vessels. The ileo-cæcal lymph vessels pass upwards in front of the duodenum, discharge into the lumbar glands, and thence into the *receptaculum chyli* (Braithwaite).

The large intestine, with the exception of the cæcum and rectum, has a relatively poor lymph system. The ascending colon drains into the ileo-colic glands; the transverse colon into the mesenteric glands; the descending colon and sigmoid direct into the lumbar glands, and the rectum into the glands which lie upon the inferior mesenteric artery.

For our present purpose I would direct attention specially to three groups of vessels and glands in their order of pathological importance.

1. The group which accompanies the ileo-colic artery and drains the lower six inches of the ileum, the ileo-cæcal valve, the cæcum, and the appendix.

2. The group of vessels and glands which surrounds the superior mesenteric artery. These compose the final gland station for the lymphatic drainage of the jejunum and the ileum, with the exception of its lower six inches.

3. The group of vessels and glands which converge upon the celiac axis, and receives lymph from the stomach, duodenum, and gall-bladder.

It will be useful for us at this point to remember that the lymph-glands in the neck present a very close analogy to those in the abdomen, and often by a consideration of glands which are seen and palpable, we shall be able to infer the condition of those which are unseen and impalpable. For example, the main aggregation of lymphoid tissue in the pharynx is the tonsil; it is in close connection with and is drained by the upper deep cervical group of lymph-glands in the neck. The main aggregation of lymphoid tissue of the intestine is found at the lower end of the ileum in the Peyer's patches and solitary follicles. These are closely connected with and drained by the ileo-cæcal group of lymph-glands extending upwards from the ileo-cæcal angle. Moreover, as everybody knows, both these groups of glands are common seats of tuberculous infection, and both

in the case of the tonsil and in the case of the lower ileum it is rare to find any sign of tuberculous disease, and we are forced to the conclusion that the tubercle bacilli can pass through intact mucous membrane without leaving any trace behind it. This has been proved experimentally by Calmette, McWeeney, and many others. We must remember, however, that when tuberculous ulceration of the intestine does occur, as, for example, in pulmonary disease, the ulceration will be found in this region in eighty-five per cent. of cases: (1) in the lower six inches of the ileum, (2) in that part of the cæcum where the stream of contents from the ileo-cæcal valve strikes it, and (3) in the ileo-cæcal valve itself, in this order. On the other hand, where tuberculous infection of a lymph-gland takes place, and is of such severity as to pass through it and cause disease elsewhere, it cannot do so without leaving permanent, unmistakable evidence of its passage (Cohnheim's Law, quoted by Cobbett).

PATHOLOGICAL CHANGES IN GLANDS.

1. *Alteration in size.*—Normally glands vary in size from being hardly visible to the size of a hazel-nut (Quain) or an almond (Cunningham). In deciding the question of size, the position of the gland must be taken into consideration, e.g., a gland the size of a hazel-nut occurring close to the gut must be held to be enlarged.

It has been pointed out by many observers that obvious inflammatory diseases such as appendicitis, gastric and duodenal ulcer, and even tuberculous ulceration of the intestine, are often unaccompanied by marked glandular enlargement. In tuberculous ulceration, for example, marked enlargement of glands only occurs in 22.8 per cent. (Godbery, Sweaney, and Brown). Winkler in three hundred cases of intestinal tuberculosis found gross enlargement of glands in only two cases.

On the other hand, in the absence of any gross intestinal lesion or gross involvement of peritoneum, great glandular enlargement may be found. In acute lymphadenitis of the ileo-cæcal group, the mass may resemble a bunch of purple grapes, the ileo-colic artery representing the stem.

2. *Alteration in size relative to position.*—Normally the largest glands are the most proximal. If this relationship is reversed, so that the largest glands are distal in position, this indicates the presence of a local pathological cause.

In the ileo-cæcal area, this reversal is found in the enlargements with which we are here dealing, whilst, on the contrary, in the superior mesenteric group it is the rule to find the largest glands near the mesenteric root.

If it may be assumed that the most affected gland is nearest the seat of infection, then it can be argued that the route of infection in the superior mesenteric area is different from that in the ileo-cæcal area.

3. *Alterations in naked-eye appearance and consistence of the glands.*—(1) The glands may be red in colour, soft, fleshy on section, tend to be flattened—the appearance found in acute inflammation. These are generally found in the ileo-cæcal area. (2) The glands may be firm or even hard and fibrous, suggestive of a chronic inflammation. These tend to be discrete, and are most characteristic of the enlargement found affecting the superior mesenteric or celiac group of glands. (3) *a.* The glands may be hyaline or caseating, indicative of a tuberculous infection.

b. The glands may show definite calcification. This may occur in one or two isolated glands, or a group may be affected. In the latter case they tend to become matted together, and in latter stages become adherent to the overlying peritoneum.

c. Chronic abscess formation. This is relatively rare in the abdomen, considering the number of cases in which the foregoing types of enlargement are found. Rupture of a chronic glandular abscess gives rise to acute symptoms, with signs of localised or generalised peritonitis.

4. *Changes in the other abdominal contents in the presence of gross glandular enlargement:—*

(a) Abnormal irritability of the intestine so that even gentle handling of the gut produces spasmodic contractions, especially of the circular muscle-coat. These contractions tend to occur in an irregular fashion, and quite unlike the orderly passage of a normal peristaltic wave.

Carson has found definite evidence of intussusception, and in two cases observed this develop under his eyes during the progress of an operation. He has suggested that the pain which is so common a feature of the condition in its acute forms, may be caused by temporary intussusceptions which spontaneously resolve. In favour of this view, it might be pointed out that in the ordinary ileo-cæcal type of intussusception, gross enlargement of the ileo-cæcal group of glands is very common.

Irregular peristalsis due to the cutting off of central control to the autonomic nerves by the swollen glands, would seem to afford a ready explanation for the pain, but Corniolay records a case upon which he operated where the symptoms had previously suggested a perforated ulcer of the stomach. He found about 30 cms. below the duodeno-jejunal junction a segment of gut 20 cms. long, port wine in colour, and very oedematous. In the corresponding parts of the mesentery about twenty large glands, the mesentery thickened, but the peritoneum and the bowel normal. Three weeks later the gut was normal to barium-meal examination. In view of this and another similar case, he suggests a vascular cause for the pain so typical of these cases.

(b) Spasm of the pylorus will often be noted in these cases at operation. It persists for long periods whilst the organ is under observation, but as a rule will have disappeared if the stomach be examined again before the abdomen is closed. It may be noted that these cases both in acute and chronic stages frequently show a moderate gastric residue at six hours after ingestion of a barium meal.

(c) Freeman claims that a similar irritable state of the abdominal wall exists in those patients, making closure of the wound difficult unless under deep anæsthesia.

NATURE AND ROUTE OF INFECTION.

Since the time of Corner, the view has been held that the infecting micro-organism in these cases is the tubercle bacillus, and recent researches have shown the prevalence of the bovine type of the bacillus. This was held by Floderus, Carson, and Struthers, and more recently by McFadden, Bell, and others.

In 1923, Heusser threw doubt on this assumption by reason of the results of his

extensive and detailed examination of actual clinical material. These results have been stated already—briefly, he holds that some cases are certainly tuberculous, but others are as certainly not. On clinical grounds Freeman, Rendle Short, Jennings Marshall, Wilensky, and Halm have accepted Heusser's views. Payr goes farther, and asserts that the glandular enlargement in the ileo-cæcal area is tuberculous, but the smaller, harder, more discrete, and more diffuse enlargements in the mesentery of the upper part of the small intestine are of some other origin.

That most of the enlargements occurring in the ileo-cæcal area are tuberculous, few will deny. Arguments both direct and indirect may be adduced in favour of it:—

(1) Tubercle is a common infection—as evidenced by the intracutaneous tuberculin test. Opie (Philadelphia) tested four thousand school-children. Of these thirty-seven per cent. were infected under 5 years; seventy-one per cent. under 10; ninety per cent. under 18. Abt gives the following figures for Vienna:—Fifty to sixty per cent. under 6 years; eighty per cent. under 10; ninety per cent. under 14.

(2) Mesenteric glands affected by tubercle are common at post-mortem examinations. Leonard in 1931 found, out of *fifty post-mortems* in which there was evidence of tubercle, *forty-five showing mesenteric adenitis*. Infection with tubercle is so common in this region that some, including Calmette, look upon it as the primary intestinal focus corresponding to Ghon's so-called primary focus in the lung.

(3) When intestinal ulceration occurs in tubercle, Brown and Sampson have shown that in eighty-five per cent. of cases it occurs in the ileo-cæcal area.

(4) When enlargement of glands occurs in this area, it shows the distribution which might be expected if infection took place from the gut.

(5) The enlarged glands seen in an early case are of the type of an acute lymphadenitis. Later they show caseation and calcification or other definite evidence of tuberculous infection.

(6) Similar appearances occur in the deep glands of the neck. Many of these show a sudden onset with acute symptoms and high temperature, but without an obvious lesion in the throat. These on section show tubercle bacilli.

A boy, A. N., aged 2 years, reared on tubercle-free milk up to 1st June, 1925, when he was taken to the seaside and given ordinary milk. On 19th July he took suddenly ill with rapid and marked swelling of the tonsillar glands on the right side of the neck. Temperature varied from 100-103°, and the child was very ill. Subsequently the glands were removed, and were found to be swarming with tubercle bacilli as the only type of infection.

Many other cases of this kind might be quoted, but it will probably be agreed that with very few exceptions these acute cases of lymphadenitis in the ileo-cæcal region are of tuberculous origin, that the infection usually occurs in infancy or in early life, and that the vehicle of infection is milk.

A similar type of infection directly from gut probably occurs at times in the glands of the upper part of the mesentery of the small intestine, e.g., occasionally a gland as big as a large marble or even larger may be found in the distal part of the mesentery. This may lie opposite an obvious ulcer in the small intestine, or the intestine may be free of obvious disease. In either case, we may assume with Cobbett that infection has come from the area of gut drained by the gland.

This is, however, a rare type of case in my experience. It is much commoner

to find a moderate but generalised enlargement of these glands. They maintain the normal relationship of size to position, but the most proximal glands may form quite a large mass. How are these to be accounted for? There are three possibilities :—

(1) They may be tuberculous, and sometimes they undoubtedly are. Such cases show caseation or calcification, and may throw shadows on the x-ray plate. They may coalesce into a large palpable mass, such as Corner removed in one of his original cases from the left hypochondrium. If we can accept the general rule that the largest glands are nearest the seat of infection, then these glands must be infected from the blood and not from the gut. In short, these glands are infected in the same way as the axillary, inguinal, femoral, and other subsidiary glands, which cannot obviously be infected from their own drainage area, but which are very frequently enlarged in the cases we are describing. Infection in these cases is blood-borne, arising either by the primary invasion passing rapidly through the lower intestinal glands and *receptaculum chyli* into the blood-stream, or from a focus which has remained quiescent for longer or shorter periods. These foci, temporarily innocuous, are potentially virulent, in the former case bacilli from them killing the patient by a miliary tuberculosis or meningitis, in the latter producing disease of a less extensive character or in a less important organ. The lymph-glands are especially prone to infections of this kind. These phenomena have been reproduced and proved experimentally in animals by many observers.

(2) They may be due to infection by other micro-organisms. Adami has shown that what is true of tubercle bacilli is true also of other microbes. He has recovered *B. coli* streptococci as well as tubercle bacilli from the lymph of the thoracic duct of animals one to two hours after feeding with an infective meal. Wilensky believes these glands are so infected, and react by increasing in size and later becoming hard and fibrous.

This type of infection may occur in influenza, according to Freeman, and accounts for the frequency with which enlargement of glands follows on this disease.

If the infection be regarded as micro-organismal but non-tubercular, the primary focus may lie within an inflamed appendix. There is some support for this view from the fact that many cases are improved by operation in which the appendix alone is removed—though it must be admitted that it is rare to find it the seat of obvious disease.

It is well known that abscesses at the roots of teeth and infections of the tonsil give rise to septicaemias of various forms, notably certain forms of rheumatism, and it is possible that mesenteric lymph-glands may be infected in a similar manner from these sources.

(3) The third view on these glands is that they are not really pathological, but that they arise from a simple hypertrophy of glandular tissue owing to excessive functional activity. Corroboration for this view comes from the histologist, who, in the absence of tubercle, usually reports a simple hyperplasia; from the comparative anatomist, who finds in the human subject a much higher development of the lymph-gland system than in the lower animals; and from the theorist, who

regards this development as a consequence on the requirements of digestion, owing to the multifarious diets which civilisation has demanded.

Having thus outlined the anatomy of the mesenteric lymph-glands, their pathological variations, and the nature and routes of infection, let us consider them from their clinical aspects. Such a survey will prove that we are in fact dealing with a definite clinical entity. It will not be denied that often the symptomatology is diffuse and the findings at operation manifold. In spite of these facts, however, we can, after excluding those in which a palpable lump can be detected and those in which intestinal obstruction is caused by adhesions or ulceration into gut, classify our cases under three heads :—

1. Those due to acute lymphadenitis of the ileo-cæcal group of glands, seen during the acute stage.

2. Those due to a chronic lymphadenitis, sequel to a previous acute attack seen at some time, days, weeks, months, or years after the acute symptoms have passed off.

3. Those due to chronic glandular enlargements (I do not assert that they are all inflammatory), where there is no history of an acute attack.

1. Those due to acute lymphadenitis, the result of infection by tubercle. This is the type which very closely simulates acute appendicitis. I am prepared to admit that the likeness between the two diseases is often very striking, and that in some it is not possible to exclude the possibility of an appendicular cause for the symptoms. On the other hand, I cannot agree with those who hold that it is not worth while trying to make the distinction, and that operation can do no harm. In the various series of recorded cases there is a definite and often quite a large operative mortality, and, in addition, there is a grave risk, especially in the acute cases, of spreading the infection by handling the infected glands. In the literature there are many cases which show that this has actually taken place, and where a second operation has been rendered necessary, apparently by such extension.

Hence the importance of arriving at an accurate diagnosis without operation. This demands both a detailed history as well as a thorough clinical examination, for there is no single symptom or sign of appendicitis which may not be simulated in lymphadenitis.

Let me give a brief outline of two cases—one of my own and one kindly supplied by Mr. McConnell.

1. On 7th January, 1928, I was asked to see a small girl aged 6½, by Dr. Martin of Banbridge. On the previous day she had taken ill with a sudden severe abdominal pain. After the onset of the pain she had vomited. Dr. Martin found her in bed with a normal temperature and normal pulse; her abdomen was soft and pliable, but there was marked tenderness without rigidity in the right iliac fossa. The following day the temperature was 99.4° and the pulse 104. At this stage I was asked to see her.

She was a small, rather puny, pale child; there were many lymph-glands palpable on both sides of the neck and in the axillæ, the abdomen was not distended and moved freely on respiration. There was definite tenderness in the right iliac fossa, but no rigidity, and there was no tenderness or abnormal mass to be felt in the pelvis. As the pain had begun in the epigastrium, had been followed by vomiting, and later by tenderness in the right side, and as the pulse and temperature at the beginning were normal and had gone up afterwards, it was thought safer to operate, though a glandular enlargement seemed the more likely. At operation a normal appendix was removed, and many enlarged and caseating glands were found in the ileo-cæcal region.

2. A child, a girl, aged 2 years 2 months, was referred to Mr. McConnell by Dr. McDonald of Portaferry.

Previous History.—Mother quite sure she had never had a previous attack of abdominal pain.

Present attack.—On the day of the attack she felt quite well at breakfast at 8 a.m., but the mother thought she was not looking well. At 12.30 she was unable to eat any dinner, complained of abdominal pain, and vomited. Seen by Dr. McDonald at 6 p.m. He found her looking ill. Temperature was 102° , and pulse rapid. Tender and possibly rigid in the right side of the abdomen. He thought of the possibility of an appendicitis or intussusception from the crampy nature of the pain, but there was no blood and no mucus passed per rectum.

Mr. McConnell saw the child four days after the onset of symptoms. A sturdy, well-developed child, looking ill. Temperature 101° . Right side of abdomen tender, but not rigid. Pulse rapid. Tender swelling high up on the right side. No blood. No mucus. She had vomited many times since the onset of illness. Seen in consultation by Professor Lowry, who agreed that as it was not possible to exclude appendicitis, it was safer to operate.

The appendix was found to be normal. Numerous acutely inflamed, enlarged glands were found in the ileo-caecal angle and mesentery.

It may be useful at this point to compare the outstanding characteristics found in acute lymphadenitis with those found in acute appendicitis :—

1. *Previous History.*—Acute appendicitis, especially in early life, comes as a bolt from the blue. Glandular cases will frequently give a history of previous attacks of pain, rarely severe, relieved by lying down, occurring during or just after meals, not seldom associated with some deterioration in general health or loss of flesh or colour.

2. *Age of Patient.*—Twenty-nine of Carson's fifty cases occurred between the ages of 5 and 15. It may, however, occur as early as 1 year.

3. *Onset* is sudden, but not quite so sudden as in appendicitis, especially of the obstructive type. For some hours before the onset of pain the child may look poorly.

4. *Progress of the Case.*—In appendicitis, as pointed out by Zachary Cope, there is a definite march of events with differing symptoms and signs corresponding to a varying pathology, and dependent on the structures involved—first, the appendix only, then the peritoneum, then, following rupture of the appendix, involvement of rectum, bladder, psoas muscle, etc., according to position. These changes do not occur in the glandular cases.

5. *Pain.*—The site of the initial pain in my experience is most often right-sided, though it may begin in the epigastrium or elsewhere. Carson, however, considers the pain an important diagnostic point, which he describes as "a sudden centralised abdominal pain which makes the child cry, lasts fifteen minutes or less, and is relieved by pressure or heat, and stops as suddenly as it began."

6. *Vomiting* occurs in about half the cases, but nausea is almost universal.

7. *Temperature and Pulse.*—Temperature varies greatly—it may be as high as 103° on the day of onset. This is strong evidence against appendicitis. In appendicitis you would guess the temperature at 102° , when in reality it is only 99° . In lymphadenitis the figures might be reversed.

Pulse varies so much that it is a poor guide.

8. *The tongue* is moist and less furred than in appendicitis, and the breath less foul. It lacks the characteristic fetor which is found in *B. coli* infections in general.

9. *The superficial glands* are usually enlarged, but not markedly so. Neck,

axillae, groins, and rectum should be searched carefully for palpable lymph nodes.

10. Abdomen:—

(a) *Inspection.*—Normal or slightly distended. Moves freely and equally throughout.

(b) *Palpation.*—Pain on pressure in right iliac fossa is not referred to middle line as it often is in early appendix cases.

Payr has described two tender spots, one on the right above and medial to McBurney's point, the other above and to the left of the umbilicus.

Real rigidity is absent, and a lump was found in only three of Carson's fifty cases.

(c) *Percussion.*—Some fluid may be present, but is too scanty to be recognised by percussion.

11. There is no limitation of thigh movements.

12. Two other points may be mentioned :—

(a) Mr. McConnell finds in most cases that the mother has noticed some swelling of the child's abdomen.

(b) Dr. Tate has found free fluid in the pleural cavities in cases of abdominal lymphadenitis.

PROGNOSIS.

This is as a rule favourable, and the disease a self-limiting one. As a general rule the mere fact of glandular enlargement is a sign of successful resistance to the infection by the tubercle bacillus.

OPERATION.

This is not necessary, and therefore undesirable. If the abdomen be opened for purposes of diagnosis, I am convinced that in the acute cases at any rate, no attempt should be made at removal of the glands, for the reasons already stated.

2. The second group of cases is due to a chronic lymphadenitis, sequel to an acute attack, in which the patient is seen at some time (weeks, months, or years) after the acute attack. These cases are subject to frequent exacerbations with pain, rise of temperature, pulse, etc., and, just as in the acute type, they also are of tuberculous origin. Obviously the glandular condition will depend on the interval of time since the original acute attack. The glands may show a general enlargement of uniform type, but usually caseation or calcification will be found in some glands, whilst others are soft and fleshy. The enlargement may affect the ileo-cæcal group alone, or may be generalised, affecting the other groups as well.

Two examples of this group may be cited :—

Case 1.—The first was a schoolboy aged 17, whom I saw in May, 1929. He was a boarder at a large public school. His previous history was beyond reproach. The finest wing-three-quarter playing school football in the winter 1926-7, he carried all before him in the school sports in April, 1927. Just after the sports his illness began with a sudden severe attack of right-sided abdominal pain, with a rapid pulse and a temperature going up on the first day to over 103°. The school doctor had diagnosed appendicitis, put him to bed on a scanty fluid diet, and advised removal of the appendix when the attack had subsided.

At this stage, about a fortnight after the onset of the illness, I examined him. I found him a tall, thin, rather pale, but apparently wiry type of schoolboy. His pulse, temperature, and general condition were then normal, but there was still deep tenderness in the right iliac fossa. Some small, hard, shotty glands were palpable in both sides of the neck, in both axillae, and both groins:—

from the latter region they extended above Poupart's ligament. Rectal examination was negative.

At this operation again no gross disease of the appendix was found, but there were many enlarged lymph-glands at the ileo-cæcal angle. One of these was definitely caseating, but to confirm the diagnosis of tubercle it was removed for histological examination.

Case 2.—The second case of this group was in a doctor, a woman, who came under my care in March, 1923. Her first attack occurred during the war, when she was working as a student house-surgeon. She was confined to bed at this time with a moderate rise of temperature and pain and tenderness in the appendix region. The attack was thought to be due to appendicitis, but owing to the stress of wartime work, operation was deferred. Similar attacks occurred from time to time, especially when she worked too hard. The attack in which I saw her for the first time followed close reading for her M.D. degree. She complained of pain between the appendix and gall-bladder regions, and at this point she was tender, but not rigid. Her temperature was 100°, and her pulse 100. She herself felt sure she had appendicitis—this is a common history even in lay patients.

At operation her appendix was kinked, but not inflamed, and unlikely to account for any rise of temperature. Her gall-bladder and duodenum were normal, as were also her pelvic organs. There were a large number of fleshy glands in the ileo-cæcal region, and also in the mesentery of the small intestine. These varied in size from a small pea to a hazel-nut, and one definitely calcareous gland was found in the peripheral part of the mesentery of the jejunum.

Subsequently she made a slow convalescence, but still got attacks of right-sided pain, especially, just as before her operation, when she became fatigued with excessive work. Following one of these attacks, she went for a time to Switzerland, and whilst there some enlarged glands were demonstrated by X-rays in the right side of the mediastinum, and were thought to account for her pain. She has never shown any sign, either physically, clinically, or radiologically, of pulmonary disease, and I believe her condition has arisen by infection of her ileo-cæcal glands with a bovine tubercle bacillus.

Diagnosis in these cases is particularly difficult. They show gastric symptoms, often including pain one to three hours after food, hyperacidity, nausea, loss of appetite, and constipation. They are easily exhausted by overwork and confinement. They are almost invariably tender in the right iliac fossa, though the tenderness is above and internal to McBurney's point. Test-meal and radiological examination may show hyperacidity and delay in emptying of the stomach. They are often regarded as examples of so-called appendix dyspepsia.

Operation for removal of this unoffending organ will not seldom be performed, and laparotomy will at once clear up the diagnosis and indicate the lines of treatment, which are those of tuberculosis elsewhere. With such treatment the prognosis will be good, though the patients will be well advised to avoid over-fatigue and confinement.

In both the foregoing groups the surgical pathology is clear and definite. Though symptoms may be obscure and diagnosis difficult, the pathology leaves no doubt as to the nature of the infection and the correct lines of treatment.

3. The third group is less clearly defined.

The symptoms are vague in the extreme, suggesting at one time a diseased gall-bladder, at another a duodenal ulcer, at still another a chronic appendicitis, and yet the picture of any one of these is not quite complete. There is no rise of pulse or temperature to indicate an inflammatory origin for the symptoms. These are the cases that pass from one hospital bench to another. They have their appendices removed by one surgeon, and their gall-bladders removed by another. In the early days of gastro-enterostomy they had this operation done because their

pyloric valves did not admit two fingers, and this was later undone because to their previous symptoms were added profuse vomiting of bile. They are usually edentulous as the result of traumatism, and their tonsils have been successfully enucleated.

A complete epitome of their symptoms would be impossible on the present occasion, but I shall give you an outline of the notes of a case recently operated upon in my ward (kindly supplied by my house-surgeon, Dr. George Kane) :—

Mrs. J., aged 34, married. Two children alive and well. Pleurisy eight years ago. Bilious attacks as a child and since. For the last one-and-a-half years dragging pain in the right side close to the umbilicus. Attacks begin with vomiting. Pain has no relation to food, and is not relieved by it. Alkalies do not relieve. Appetite good. Bowels constipated. Micturition and menstruation normal. Loss of weight recently.

On examination, patient is pale and anæmic. Tongue moist. Teeth—uppers artificial, lower incisors good. Fauces infected. Abdomen—outline normal, movements good. No superficial tenderness, but on deep palpation two tender spots are found corresponding to Payr's points as already described. Liver, spleen, and kidneys show nothing abnormal. Heart, lungs, and central nervous system normal.

X-ray examination after an opaque meal showed nothing abnormal, and the test-meal figures lie within normal limits.

Operation through a right paramedial incision shows the gall-bladder, stomach, and duodenum normal. Appendix seemed fibrotic and distended at the tip, but no sign of inflammation—it was removed. The glands of the superior mesenteric group were markedly enlarged, both those of the intermediate zone as well as those within the root of the mesentery. The glands in the lesser omentum were also enlarged.

That these glands are enlarged to a pathological degree I have no doubt whatsoever; that the enlargement is frequently tuberculous in origin is also beyond doubt; but there are other enlargements which have not yet been adequately accounted for. Such cases form an appreciable proportion of the chronic abdominal problems occurring in practice. Out of 275 cases of this type, including appendicitis, duodenal ulcer, and mesenteric glandular enlargement, I find the following, all proved by operation :—

Appendicitis	-	-	-	-	-	82
Duodenal Ulcer	-	-	-	-	-	240
Enlarged Lymph-glands	-	-	-	-	-	53

With the exception of Heusser's work, I can find in the literature no complete reports on the histology, the bacteriology, or inoculation examination of these cases. The clinical side of the problem has been studied, and many papers have been published, but the subject awaits fuller investigation by the laboratory worker.

REVIEW

BEDSIDE INTERPRETATION OF LABORATORY FINDINGS. By Michael

G. Wohe, M.D. London: H. Kimpton, 1931. pp. 321; 133 figs. 25s.

DR. WOHE has written a most readable book. In it he describes laboratory investigations which the practitioner who wants to cultivate a modest measure of personal laboratory work might reasonably perform in his own consulting-room. He makes no attempt to make every practitioner an expert in the more complicated chemical and biological tests, but confines himself to the simpler tests. The book can be warmly recommended to the general practitioner as being eminently practical.

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Forty-four Consecutive Cases of Gastric Ulcer Treated by Gastrectomy

By P. T. CRYMBLE, M.B., F.R.C.S.ENG.,
from the Royal Victoria Hospital, Belfast

AN examination of the records of the Royal Victoria Hospital, Belfast, for the period 1919-1927 inclusive, a period of nine years, shows that during this time only twenty-eight gastrectomies and pylorotomies, apart from the series under discussion, were carried out by the seven surgeons of the staff. If we may conclude that the surgery of this hospital is a fair sample of the surgery of Ulster, then we are justified in stating that in the past our Province has had its gastric ulcers treated medically, or by gastro-enterostomy, with or without local excision of the ulcer.

Modern gastric surgery originated in Vienna, and therefore we are inclined to look to Vienna for a lead in the surgical problems of the stomach. A number of papers by the leading Viennese gastric surgeon, Finsterer, some ten years ago, produced a marked change in method which is now beginning to bear fruit in foreign countries. In this small series of cases, I have merely followed the practice of Finsterer, which is to remove the lower half, or lower two-thirds, of the stomach, which includes the ulcerated area, and to restore the continuity of the alimentary canal by a retrocolic end-to-side gastro-jejunostomy.

There are six different surgical methods available for dealing with gastric ulcer:—

EXCISION OF ULCER.—This would appear to be insufficient, as there is a tendency for the ulcer to recur, since nothing has been done to diminish the ulcer tendency.

GASTRO-ENTEROSTOMY.—This method gives good results in cases of prepyloric ulcer. In my series, the gastric ulcer developed in the presence of a gastro-enterostomy in three cases. I have in my possession a radiogram of a gastric ulcer treated by gastro-enterostomy twenty-five years ago. The patient has never been free from indigestion, and the stoma is seen functioning to the left of the ulcer site.

WEDGE RESECTION AND GASTRO-ENTEROSTOMY.—This is suitable for small mobile ulcers, but it may leave behind a large area of diseased mucous membrane.

CAUTERISATION OF THE ULCER, plus gastro-enterostomy.—This method gives good results in Balfour's clinic, but I have no personal experience of it.

SLEEVE RESECTION, plus gastro-enterostomy into the lower compartment.—This operation takes longer to perform than a gastrectomy, and has the disadvantage of leaving the pyloric end of the stomach the gastrin (HCl ferment) producing area.

GASTRECTOMY.—Of these methods, this appears to offer the best chances of success, and in support of this view may be cited the following facts:—

(a) There is a reduction of acidity in the gastric contents.

- (b) The ulcer-bearing area (pyloric end of the stomach and lower two-thirds of the lesser curve) is removed.
- (c) It offers the safest and easiest means of dealing direct with an adherent ulcer.
- (d) Not only is the palpable ulcer removed, but the excised area of stomach is often found to be the seat of gastritis and multiple ulcers.
- (e) The danger of leaving a cancerous ulcer untouched is eliminated.
- (f) The danger of jejunal ulcer is almost completely eliminated. (It was absent in Finsterer's last three hundred cases.)

Every case in the series under review which was subjected to X-ray examination, showed some form of abnormal opaque meal result. A niche was present in eighteen out of thirty-three cases. This abnormality appears as an opaque spur projecting to the right from the lesser curve. It indicates the site of the ulcer, and is produced by the ulcer. Seven-hour gastric retention was the only abnormality found in twelve out of thirty-three cases. The retention is in the lower pole, and gives no indication as to the site of the ulcer. An hour-glass stomach was noted in ten out of thirty-three cases. In seven of these a niche was present, and in three there was no niche.

SYMPTOMS.

The symptoms present in the series may be discussed under four headings :—Pain, vomiting, hæmorrhage, loss of weight.

Pain.—This symptom was present in every case of the series. It would appear to be the one and only symptom which is invariably present. It is usually related to food, but the time of onset varies according to the patient. The usual period is half an hour to one hour after food, but it may be extended up to three hours. A number of patients complain of pain, but give no definite relation to food.

Vomiting.—This symptom was present in eighteen out of forty-four cases. It tends to relieve the pain.

Hæmorrhage.—In eleven out of forty-four cases hæmorrhage was present, either in the form of hæmatemesis or melena. It should, however, be noted that this condition is often found in the absence of ulcer.

Loss of Weight.—In nine out of forty-four cases there was a loss of weight. It is most unusual to find a gastric ulcer in a fat person, but it does occasionally happen. Case No. 26 was fairly stout, and showed a large chronic perforation on the posterior wall of the stomach.

THE AFTER-TREATMENT of patients who have had the operation of gastrectomy performed may be summarised as follows :—

- (a) Hæmostatic serum, 2 c.c.
- (b) Glucose saline per rectum, or intravenously if shock is present.
- (c) Fluids only, by the mouth, for five days.

- (d) No aperient stronger than semproline with phenolphthaline.
- (e) Gastric lavage for persistent hæmorrhage or obstructive vomiting.
- (f) Alkalies by the mouth for several weeks.

In discussing the treatment of gastric ulcer, it should be borne in mind that the most successful operation yet devised is that of gastrectomy, and in my experience it is the easiest and safest type of operation in those cases where the ulcer is adherent to the posterior abdominal wall : in cases of lesser-curve ulcers of medium size, and in which there is a considerable area of inflamed stomach around them : in all cases of gastric or jejunal ulcer following gastro-enterostomy.

It should be remembered that syphilis may produce gastric ulcer, in which case the ulcer will heal rapidly with the usual anti-specific remedies. Also, that quite a number of gastric ulcers appear to be associated with tubercular disease of the lungs, and this of course introduces an additional risk into the operation.

An interesting point in this series of cases is that in all cases the ulcer was situated in the lesser curve, and in most on the posterior surface. The commonest site was that part of the lesser curve which overlies the pancreas, the pancreas often forming the floor of the ulcer.

Does this pathological feature throw any light on the causation of the condition? The lesser curve is the most fixed part of the stomach, it is the site of the "magen strasse," the permanent stomach canal, and is intimately related to the gastric vessels, lymphatics, and nerves. My belief is that these lesser-curve ulcers begin as acute or sub-acute perforations, and that leakage is prevented by adhesion to the pancreas or lesser omentum, consequently they do not come to the surgeon as perforations.

Once the margin of the perforation becomes fixed to the pancreas, healing cannot take place. The stomach cannot grow a new wall, nor can it contract so as to obliterate the defect.

Nothing but a direct attack on the ulcer can remove it. No medical treatment, no alteration of the physiology of the stomach by gastro-enterostomy will suffice. Without a direct attack, the patient may live, may at times be free from the symptoms, but the pathological anatomy will persist.

I know of no variation in stomach anatomy which might be an ætiological factor, and which might be dealt with by some simple operation, unless possibly a hypertrophic narrowing of the pyloric canal. Some surgeons are performing pyloroplasty when they fail to find an ulcer in patients with ulcer symptoms. I have done this frequently without causing harm, and I have yet to see an ulcer develop after the operation.

Against the obstructive ætiological factor, advanced by Bolton, is the fact that pyloric and duodenal obstructions do not lead to gastric ulcer, and I have no record of a gastric ulcer developing under such conditions.

RESULTS.—There were forty-four consecutive cases of gastrectomy for gastric ulcer from 1923 to 1931. Of these twenty-seven were in males, and seventeen in females. Four of these died as a result of the operation, and one died subsequently (G.S.W. head). The patients have been communicated with recently, and the results may be classified as follows :—

Very good	-	-	-	-	27
Good	-	-	-	-	4
Fair	-	-	-	-	2
Not recorded	-	-	-	-	6
Deaths	-	-	-	-	5

Much may be done to prevent the appearance of gastric ulcer by bearing in mind the following simple rules :—

- (1) Three square meals a day should be the rule.
- (2) No intermediate snacks or drinks are taken, and food should be eaten slowly.
- (3) Tobacco should be avoided.
- (4) Careful mouth hygiene is essential.

SPECIAL CASES.

Case No. 30.

1917—Gastro-enterostomy and appendicectomy for duodenal ulcer. Remained well for seven years, when pain and bleeding appeared.

1927—Duodenum normal. Margin of stoma red and inflamed. Gastro-enterostomy undone. Remained well for one year, when pain reappeared.

1930—Admitted to Royal Victoria Hospital for hæmatemesis and pain two hours after food. Operation showed a duodenal ulcer. Two-third gastrectomy performed.

Good result up to present.

This case shows the controlling effect of a gastro-enterostomy over duodenal ulcer.

Case No. 29.

This man started his abdominal career with a perforated duodenal ulcer, which was treated by another surgeon by closure and gastro-enterostomy.

Later he returned to the Royal Victoria Hospital with dyspepsia and hæmatemesis.

At operation one found—Scarring of duodenum; stoma normal; large posterior lesser-curve ulcer (3 inches broad by 1 inch high), floor formed by pancreas, at level of *incisura angularis*, and separated by a narrow bridge of stomach from the stoma.

Gastrectomy performed.

Good result up to present.

Case No. 39.

1921—Gastro-enterostomy for duodenal ulcer.

1923—Treated medically in Royal Victoria Hospital for indigestion.

1924—Operation.

Pathology.—Gastro-enterostomy, stoma normal. Immediately to the left of the stoma was an ulcer of the stomach, the size of a penny, which had perforated all coats and was supported by the pancreas.

Technique.—Gastro-enterostomy undone, jejunal opening closed. Lower two-thirds of stomach, including ulcer, removed, and an anticolie end-to-side gastro-enterostomy performed.

Follow-up, May, 1930—Has been in excellent health since operation. Carries out full work as a packer. Meals must be small. If a large meal be taken, he may vomit. No increase in weight.

Case 15 (Fig. 1).

Hour-glass contraction and Haudek's niche in the middle third of the stomach. Symptoms for twenty-six years, consisting of pain two hours after food.

The operation revealed a large aperture on the posterior wall of the lesser curve, floored by the pancreas. The perforation was the size of a crown piece.

Three-fourth gastrectomy performed.

The operation was performed four years ago, and there has been a complete abolition of symptoms. The patient carries out the full duties of a postmistress.

Case 8 (Fig. 2).

Hour-glass contraction at junction of the lower and middle thirds of the lesser curve. There is no Haudek's niche.

A man aged 63, with hunger pain for seven years.

The operation revealed a lesser-curve ulcer, which had perforated the stomach wall, and was floored by the pancreas.

Two-third gastrectomy performed.

The operation was performed five years ago, and the patient remains free from all symptoms.

Case 32 (Fig. 3).

A labourer aged 38. A medium-sized Haudek's niche about the mid-point of the lesser curve. No hour-glass contraction.

The symptoms, which included pain, vomiting, and hæmorrhage, had been present for one year.

At operation one found a free anterior lesser-curve ulcer, surrounded by an extensive area of hard, inflamed stomach.

Two-third gastrectomy performed.

Severe primary hæmorrhage into stomach cavity was controlled by gastric lavage and the instillation of twenty minims of adrenaline.

Result excellent.

Case 6 (Fig. 4).

Haudek's niche and hour-glass contraction in the middle third of the stomach.

A woman aged 53, who had complained of epigastric pain half an hour to one hour after food for ten years.

Operation revealed a lesser-curve ulcer. Two-third gastrectomy performed.

The operation was performed five years ago, and since then the patient has increased two stone, five pounds, and has been free from all dyspepsia. She takes ordinary food and does ordinary housework.

Case 23 (Fig. 5).

Woman aged 54. The specimen consists of the lower half of the stomach, the pylorus, and a small portion of duodenum. The pylorus is contracted, and is occupied by a glass rod. The stomach, viewed from behind, shows a perforation on the posterior wall the size of a florin, and surrounded by scar tissue. In the body this aperture was closed by the pancreas.

History.—Stomach trouble for twenty-five years. Periodic attacks of severe pain one hour after food. Operation for perforation of gastric ulcer nine years ago. Symptoms persisted after operation, which consisted of closure of the perforation.

Gastrectomy was performed in 1929, and the specimen obtained.

Case 34 (Fig. 6).

Man aged 38. The specimen consists of the lower two-thirds of the stomach and a small portion of duodenum, viewed from the front. There is a large aperture on the lesser curve which, in the body, communicated anteriorly with a cavity in the liver and posteriorly with an ulcer in the pancreas. The whole lesser curve was firmly bound to the posterior abdominal wall and the pancreas.

History.—Symptoms for eight months—severe pain one to one and a half hours after food, hæmatemesis, vomiting.

The operation was rendered difficult by the involvement of most of the lesser curve and by adhesions around the duodenum. Two-thirds of the stomach was removed, but the remaining posterior stomach wall was in poor condition for suturing. The patient died two days after operation with leakage from the posterior line of the anastomosis.

(For tabulated list of cases, see next page.)

LIST OF CASES

NO.	YEAR	NAME	SEX	AGE	DURATION	SYMPTOMS	X-RAY	OPERATION	RESULT	REMARKS
1.	1923	Collins	F	30	5 years	Pain	Retention	Antecolic	V.G.	Three children since
2.	1924	Turnbull	M	39	Years	Pain	Niche	Antecolic	V.G.	Previous gastro-entero-stomy. Ulcer developed to left of stoma
3.	1924	Forsythe	F	35	1 year	Pain 3 hours after food. Vomiting	Retention	Antecolic	V.G.	
4.	1925	Carson	F	40	6 months	Pain 1-2 hours after food	No record	Retrocolic	Not known	
5.	1926	Smith	F	35	8 years	Pain 2 hours after food	Retention	Retrocolic	No reply	Severe hæmorrhage at operation
6.	1926	Brown	F	53	5 years	Pain $\frac{1}{2}$ -1 hour after food	Niche	Retrocolic	Not known	
7.	1926	Gordon	M	60	30 years	Pain 1 $\frac{1}{2}$ hours after food	Retention	Retrocolic	G	Heavy feeling after food. Palpitation
8.	1926	Lamb	M	63	7 years	Pain	Notch on great curve	Retrocolic	V.G.	
9.	1927	Luke	M	32	9 years	Pain. Vomiting. Hæmorrhage	No record	Retrocolic	No reply	
10.	1927	Hillis	M	43	3 years	Pain 1 hour after food. Loss of weight	Niche	Retrocolic	In America	
11.	1927	Donnelly	M	48	3 years	Pain 2-3 hours after food. Vomiting	Niche. Retention	Retrocolic	No reply	
12.	1927	Kennedy	F	32	4 years	Pain. Vomiting	Niche	Retrocolic	Fair	Improved, but easily upset

NO.	YEAR	NAME	SEX	AGE	DURATION	SYMPTOMS	X-RAY	OPERATION	RESULT	REMARKS
13.	1927	Gilliland	M	51	2 years	Pain 2 hours after food. Melena Vomiting	Niche	Retrocolic	V.G.	
14.	1927	Lennon	F	65	Many years	Pain. Vomiting. Hemorrhage.	No record	Retrocolic	V.G.	Abdomen explored five years ago
15.	1927	Miss O.	F	42	26 years	Pain 2 hours after food	Niche	Retrocolic	V.G.	
16.	1928	Ashfield	M	23	6 months	Pain. Vomiting. Melena	No record	Antecolic	-	Death due to emaciation and weakness
17.	1928	O'Connor	M	42	2 years	Pain 1-2 hours after food	Retention	Retrocolic	V.G.	Post-op. vomiting. Lavage
18.	1928	Bugler	M	-	2 years	Pain 1 hour after food	Niche and Retention	Retrocolic	V.G.	Typhoid during convalescence
19.	1928	Cox	M	42	4 years	Pain 1 hour after food Loss of weight	No report	Retrocolic	G	Discomfort after greasy food
20.	1929	Haskins	F	46	10 years	Pain. Periods of remission	Niche and hour-glass	Retrocolic	Death	Bad heart
21.	1929	Carson	M	25	6 months	Pain. Vomiting	Retention	Retrocolic	V.G.	
22.	1929	Mackey	M	59	12 years	Pain 1-2 hours after food. Vomiting. Melena	No record	Retrocolic	V.G.	Mesocolic hematoma. Shock
23.	1929	Woods	F	48	2 years	Pain 1-1 hour after food. Vomiting	Niche and hour-glass	Retrocolic	G	Sickness after food
24.	1929	McMullen	F	54	25 years	Pain 1 hour after food	Hour-glass Retention	Retrocolic	V.G.	Previous operation for perforation

LIST OF CASES (*Continued*)

NO.	YEAR	NAME	SEX	AGE	DURATION	SYMPTOMS	X-RAY	OPERATION	RESULT	REMARKS
25.	1929	Mr. E.	M	25	2 years	Pain	No record	Retrocolic	—	Suicide 9 months later. Ulcer developed in presence of gastro-enterostomy
26.	1929	Miss B.	F	51	30 years	Pain. Hemorrhage	Niche and hour-glass	Retrocolic	Fair	Free from dyspepsia. Pains in lower abdomen
27.	1929	McGragh	M	28	3 years	Pain 1 hour after food Loss of weight	No record	Retrocolic	V.G.	
28.	1930	Wallace	M	40	2 years	Pain. Loss of weight	No record	Retrocolic	V.G.	
29.	1930	Watson	M	24	1 year	Pain. Hemorrhage	No record	Retrocolic	G	A little pain at times. Previous op. for perforation. Ulcer in presence of gast-cent.
30.	1930	Hunter	M	52	15 years	Pain 2 hours after food. Hemorrhage	No record	Retrocolic	V.G.	2 previous operations. Gastro-enterostomy. G. Bandone. Duodenal ulcer reappeared
31.	1930	Taylor	F	58	Many years	Pain 20 minutes after food. Vomiting Heartburn	Hour-glass	Retrocolic	V.G.	
32.	1930	Collins	M	38	1 year	Pain 1-1½ hours after food. Hemorrhage	Niche	Retrocolic	V.G.	Severe post-operative hemorrhage
33.	1930	Roy	M	—	2 years	Pain. Vomiting. Melena	Retention	Retrocolic	V.G.	

NO.	YEAR	NAME	SEX	AGE	DURATION	SYMPTOMS	X-RAY	OPERATION	RESULT	REMARKS
34.	1930	Young	M	38	1½ years	Pain 1-1½ hours after food. Vomiting. Hemorrhage.	Retention	Retrocolic	—	Death. Very extensive ulceration. Leakage.
35.	1930	Tate	M	55	3 years	Pain 1½-2 hours after food. Vomiting	Retention	Retrocolic	V.G.	Two ulcers and pyloric anastomotic processes.
36.	1930	Cullen	M	40	6 months	Pain 2-3 hours after food. Vomiting. Wasting	Niche	Retrocolic	V.G.	
37.	1930	Grimason	M	49	3 years	Pain. Vomiting	Niche	Retrocolic	V.G.	
38.	1930	Bodkin	M	—	13 years	Pain ½-1 hour after food. Vomiting	Retention	Retrocolic	V.G.	Operation for perforation, 1927
39.	1930	Greig	M	43	3 years	Pain ½-1 hour after food. Loss of weight	Niche hour-glass	Retrocolic	V.G.	
40.	1930	Swain	F	50	Many years	Pain made worse by food. Loss of weight	Niche hour-glass	Retrocolic	V.G.	
41.	1930	Shannon	F	46	2 years	Constant pain, worse two hours after food. Hemorrhage	Niche hour-glass	Antecolic	V.G.	Transverse mesocolon absent
42.	1931	Bingham	F	59	26 years	Pain ½ hour after food	Niche hour-glass	Retrocolic	Death	Two large ulcers
43.	1931	Mantell	M	58	10 years	Pain 1-1½ hours after food. Loss of weight	Retention	Retrocolic	V.G.	
44.	1931	Carvill	F	35	1 year	Pain. Vomiting. Loss of weight. Water-brash	Hour-glass	Retrocolic	V.G.	



FIGURE 1

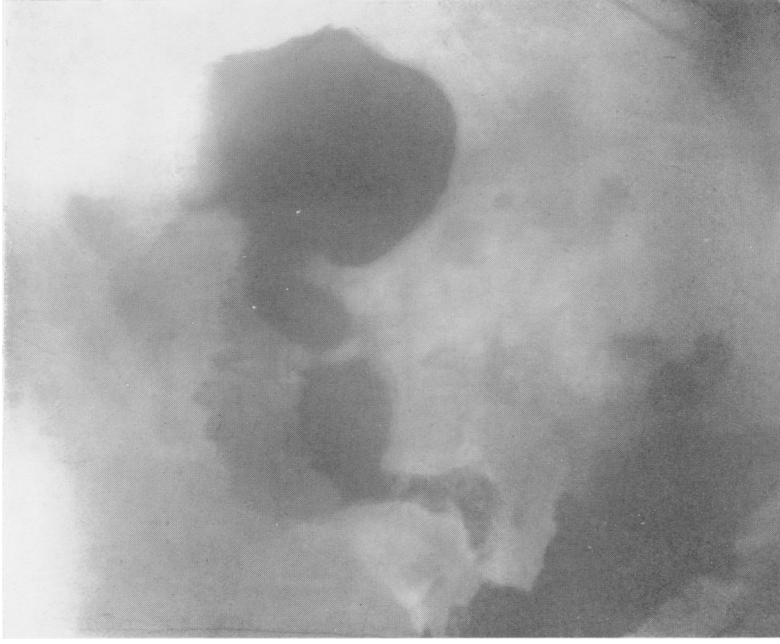


FIGURE 2



FIGURE 3



FIGURE 4

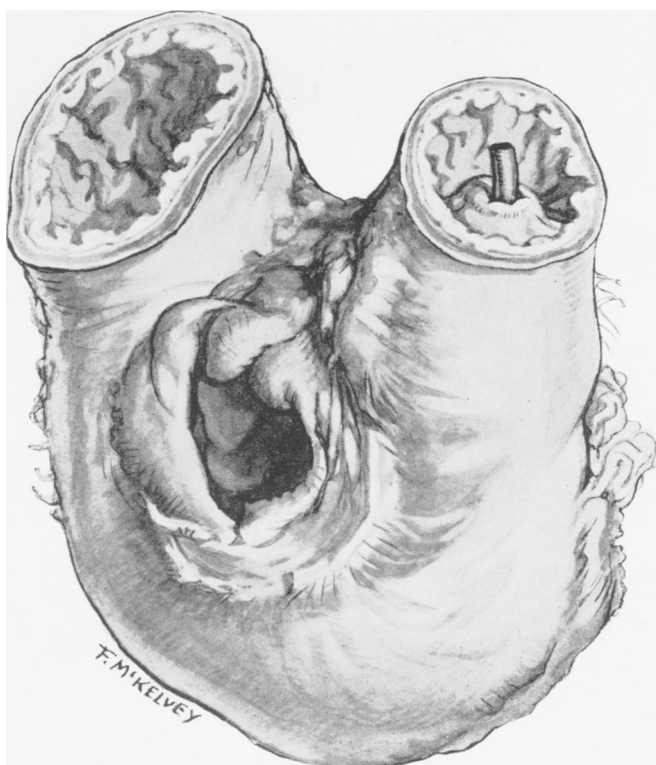


FIGURE 5

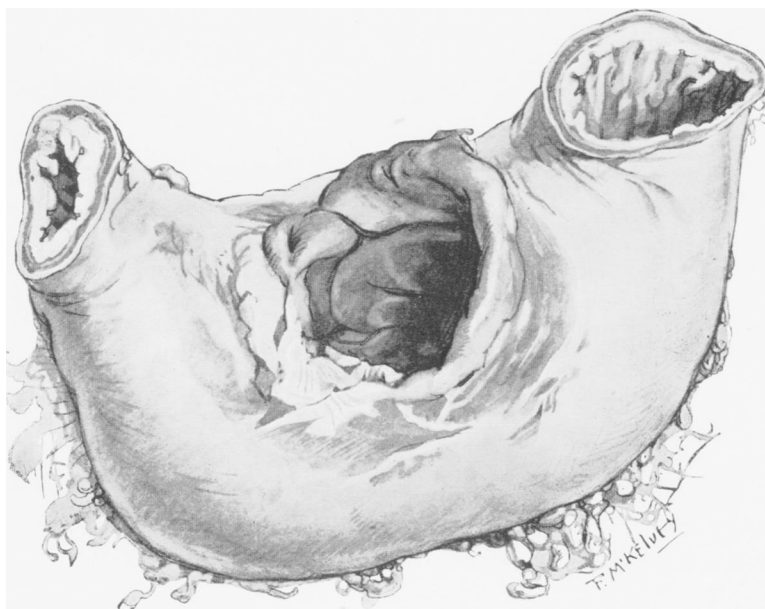
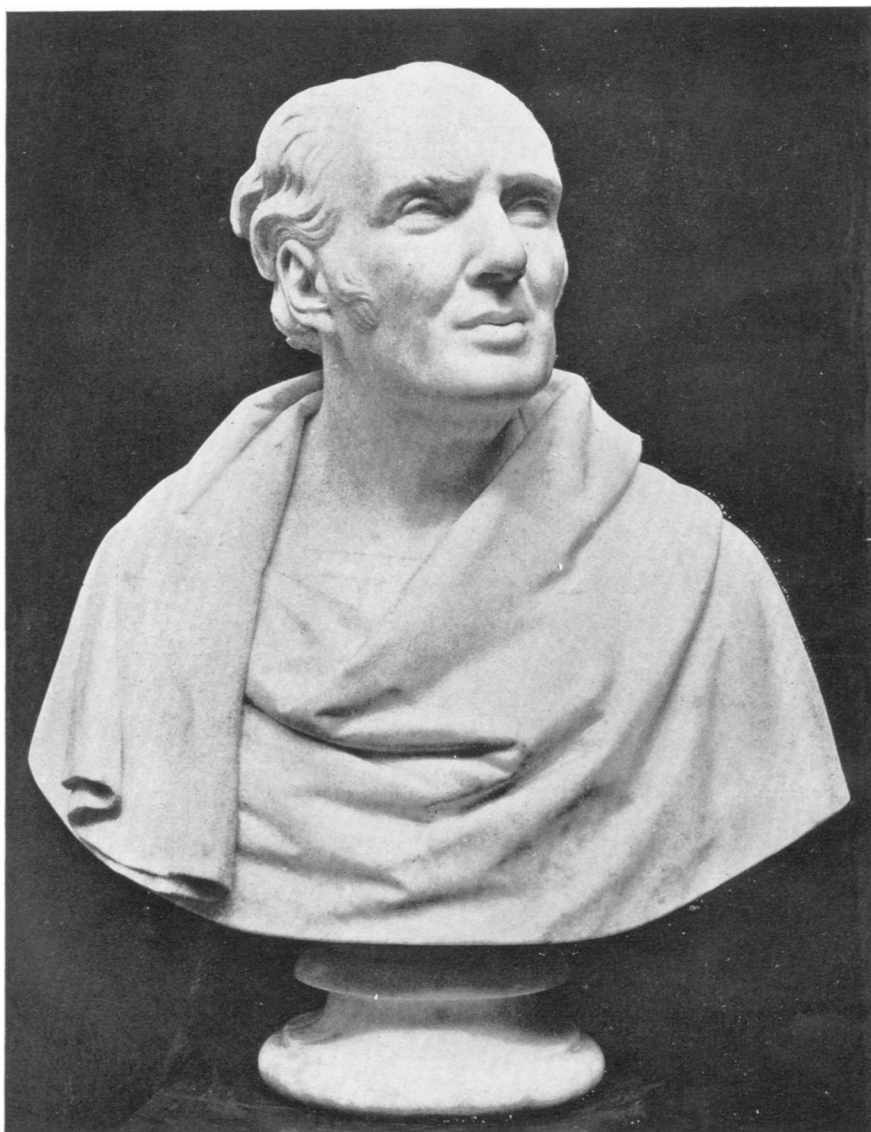


FIGURE 6



DR. JAMES MCDONNELL.

BLOCK KINDLY LENT BY THE
BELFAST NATURAL HISTORY
AND PHILOSOPHICAL SOCIETY

The Founder of the Belfast Medical School

By SAMUEL SIMMS, B.SC., M.D., M.R.C.P., D.P.H.

THE School which this new journal represents had its origin a little over a hundred years ago, and from a small beginning it has expanded, until to-day it holds no unworthy place in the great commonwealth of nations that constitutes the Empire. Its fame is upheld by its graduates who are scattered over the four quarters of the earth, and this is a direct result of the well-laid plans of one James McDonnell, the son of the owner of a little property in the Antrim Glens, where he was born in 1762, about a quarter of a mile from Cushendall on the road to Red Bay.

In 1780 McDonnell's father died, and a few years later he proceeded to Edinburgh, then the medical Mecca of Great Britain, where he graduated in 1784. His thesis was "On the Drowned." He discussed the various methods then known of resuscitation, and suggested as a last resort, transfusion of blood. He then settled in Belfast, where he gradually built up a large practice. The population of the town was about 17,000, and there were about ten physicians in practice, chief of whom was Dr. Halliday, the friend of Lord Charlemont.

The last ten years of the eighteenth century in Belfast were years of crowded life, political and intellectual, and it was during this period that the philanthropy, the foresight, and the enthusiasm of McDonnell succeeded in laying the foundation of the Medical School by the establishment of the BELFAST DISPENSARY. Previous to this the only medical relief in the town to the indigent was rendered by the Belfast Charitable Institution in Clifton Street, which, however, was limited in proportion to its means and accommodation. As a consequence, many of the poor received no medical treatment at all, and to remedy this was the aim of the founders of the Dispensary. In the words of Malcolm, the historian of our Hospital: "Amongst this noble band of philanthropists it cannot now be considered invidious to distinguish the name of one who may, without exaggeration, be considered to have represented this end throughout his active life, all the energy and zeal which animated and cherished this charitable movement—James McDonnell, M.D., of whom it will be sufficient at present to state that in times of the greatest apathy towards his favourite project, and when almost unassisted by an encouraging hand, he continued to tend and watch over it with fostering care and unabated interest, until it had reached a vigorous maturity." The prospectus was issued on 13th April: £50 only was asked for the initiation of the scheme. The first general meeting was held on the 19th, and the medical officers, Dr. McDonnell and Dr. White, were appointed on 16th May. A house was taken in West Street (off Smithfield), and the infant charity started forth on its way. Its aim was to supply free medicine and free medical attendance to the indigent poor, and also to visit them in their homes when occasion required. The days of attendance were Mondays, Wednesdays, and Fridays at 10 a.m., and the Dispensary was open till 3 p.m. The charity was popular with the class for whom it was established, and in a little over four years 2,406 patients had received medical or surgical advice, and medicine valued at

£120 per annum, which is at the rate of 4s. 4d. per person, or £21. 13s. 4d. per hundred. Of these, 1,740 were cured, 336 relieved, 50 reported as incurable, and 280 died or made no report.

In the years following 1792 there is little evidence of Dr. McDonnell's movements. It is known, however, that he was occupied with the Dispensary and with his private medical practice.

A further step was taken in 1797, on the incentive of Dr. McDonnell, to found a public hospital. He was long convinced that it was necessary to have a hospital where fever cases, which were very prevalent amongst the poor, could be properly attended. Typhus was a great scourge of the town for many years, and it was impossible to control the infection and nurse these patients in their own homes. On 14th April this idea was publicly adopted by a Committee, and on the 17th a dwelling-house was taken in Berry Street at a rental of £20 per annum. Six beds and other requisites were ordered. A nurse was appointed, and on the 27th the physicians and surgeons of the Dispensary were asked to attend at the new institution. The new hospital—the *first hospital in Ireland for fever*—was thus inaugurated. It was reported on the 1st June that ten patients had been received into the hospital, cured, and discharged.

This new institution, The Belfast Fever Hospital and Dispensary, was in this way established, and the future expansion and foundations of the Medical School can be traced to this small hospital of six beds, opened in May, 1797. The first appeal for public assistance was issued on 6th October. It stated that sixty patients had then been admitted to the hospital, with only one death, and the importance of this work to the community was stressed. The original subscriptions for the hospital had amounted to £58, and at the date of the appeal nearly £53 had been expended on the sixty patients.

In a printed letter of John Templeton (1766-1825), the Belfast naturalist, dated September, 1797, it is stated: "Our benevolent friend, the doctor, willing to do every service in his power to mankind, lately set on foot a fever hospital; the institution was certainly good, but it has proved what many people prognosticated—'a seminary for diseases,' since it has only been three months established, the housekeeper, the apothecary, the dispensing surgeon, Alexander McDonnell, and Dr. Boiragon, also the doctor himself, had all caught the fever by their attendance. They are all happily recovered, but the last-mentioned, and he is also convalescent." This incident perhaps was a factor in the temporary abeyance of the charity, as the extreme infectiousness of typhus and its mode of propagation was not then understood.

In September, 1799, a meeting was held to revive the hospital, and £113 was collected by means of a charity sermon of the Rev. William Bristow. Three houses in West Street at the corner of Smithfield were taken, and turned into a hospital. Dr. James McDonnell was appointed physician, and Mr. Bartholomew Fuller as surgeon. From this time forward for the next eighteen years, the Belfast Hospital and Dispensary increased in importance and in the affections of the people of the town. The guiding hand was that of McDonnell, who devoted his time, his

services, and his talent to making the Hospital a success. From December, 1799, to October, 1800, 148 patients were admitted, with 12 deaths, and from that date to 8th August, 1801, 249 were admitted, with 18 deaths. Thus in a little over a year and a half, there were 398 cases, mainly of typhus. For many years the only cases that the hospital could receive were those of fevers, their urgency generally excluding medical and surgical cases. Up to the year 1807 the hospital and dispensary was entirely supported by voluntary contributions, but in that year the Irish counties were enabled by Act of Parliament to grant certain sums for maintenance of these charities. The sum granted in this year amounted to £193. 7s. 6d., and the progress of the hospital was so assured that at the close of the year the question of extension was considered, but temporarily abandoned.

In 1810 the first decisive step was taken towards the erection of a new hospital building. A portion of ground was selected in Frederick Street, a lease obtained by Dr. McDonnell, Wm. Clarke, and Wm. McS. Skinner from the Marquess of Donegall, and a Building Fund was commenced. In the following year the Building Fund was increased by means of *Charity Sermons*, and the labours of collectors, and on 5th June, 1815, the first stone of the new building was laid by the Marquess of Donegall. The foundation stone has a long inscription beginning with *Hoc nosocomium ægrotis et arti medicæ sacrum*.

Dr. McDonnell now became the leading physician of Belfast, a position which he maintained for over thirty years, until ill-health forced him to retire. He was clearly the leader in the foundation of the new hospital, and his activity was everywhere visible. The years 1816 and 1817 were pre-eminently "hard years," and they were associated with a revival of the dreaded typhus. The building of the hospital was pushed on, and at length the wards were opened on 1st August, 1817. The epidemic was at its height, the patients were transferred from the old hospital, "though the walls were wet and the staircases scarcely secure," and no application from town or country was refused. This hospital of one hundred beds was further taxed in 1818 by the admission of 1,530 patients, and in 1819 by 1,258. From 1817 till 1821 no patients were admitted to the hospital except fever cases; it was probable that in these epidemics a fourth of the population was infected. It was the intention from the foundation of the hospital that its advantages should not be confined merely to the inmates. "The physicians and surgeons of Belfast should be invited to place their pupils there, to acquire experience by observing its practice, and in the course of a few years it might become a school of physic and surgery of no trifling importance to the young medical student of this neighbourhood and of the Province of Ulster." This, however, was not adopted until January, 1820, when the Committee recognised that pupils should be admitted into the hospital, and that each of the medical attendants might introduce one pupil to act as clerk or dresser. The first registered medical student of the hospital was Mr. B. Bingham, who was registered on 21st December, 1821; from that date up to 1850 over four hundred students passed through the hospital. In 1822 "The Belfast Medical Society" was revived by four physicians, one of whom was Dr. McDonnell. The original Society founded in 1806 had lasted to 1811, when it was dissolved. It was now again revived, a valuable medical library was collected

and located at the hospital. In process of time, this Society became the parent of the Ulster Medical Society, which to-day possesses its original Minute Book. For some years many pupils attended the hospital, but the question of promoting systematic clinical lectures only became prominent at the close of 1826. Dr. McDonnell, however, had advocated in 1824 that the hospital should provide the nucleus of a medical school. In 1827 it was decided that such lectures would be a great advantage to the pupils and to the hospital, and on 3rd June the senior physician, Dr. James McDonnell, gave the first clinical lecture on "Systematic Medicine." The Belfast School of Medicine had started on its way. Its author had now reached the summit of his medical career: he had come to Belfast many years previously, he had founded a Dispensary, he had founded a Hospital, and in the lecture-theatre he had become a teacher of his profession. The full fruits of his plans were not yet matured, but some years later (1836), before his death, the Medical School was completed by the establishment of a medical department in the Academical Institution. The Medical School of Belfast was founded in the old General Hospital, improved by the formation of the medical department of the Academical, and completed in 1848 by the establishment of the Medical Faculty in the Queen's College.

Feeling that the time had arrived when he should retire from his active duties, he resigned, and was appointed to the consulting staff of the hospital in 1828. In the same year, on 18th April, he was presented with a service of plate, valued at £700, inscribed as follows: "To James McDonnell, Esq., M.D., who during a period of nearly forty years has devoted his time and eminent talents to the work of humanity, whose gratuitous advice has always been at the service of the poor, and to whose exertions this town has been principally indebted for that invaluable institution, the Fever Hospital and Dispensary, this service of plate has been presented by the nobility, Ladies and Gentlemen of Belfast and its vicinity, as a tribute of their respect and esteem. A.D. 1828." This testimonial was a proof of the high position he occupied in the minds of the community, and it was certainly well deserved. For many years after this he still continued to practise his profession and to take an active part in scholarly work. His residence throughout his long life was No. 13 Donegall Place. He was of medium height, and his countenance was open, impressive, and cheerful. He was quick and able in speech, and had a good style of composition. In medical science he was against the practice of employing wet nurses, and urged that children should be nursed by their own mothers. He was interested also in the various phenomena associated with irregularities of the pulse, and was skilled in the treatment of fevers.

He survived until 5th April, 1845, when he died in his eighty-second year. He was then the oldest member of the profession in the city. His funeral was attended by the Mayor and members of the Corporation. It left Belfast at 9 a.m. for the journey to the churchyard of Layde, about one mile north of Cushendall, where his remains rest amongst those of his illustrious ancestors. A beautiful Celtic cross was erected to his memory, which cannot ever be forgotten (*Si monumentum requiris circumspice*), for to his foresight we are indebted to-day for the Belfast Medical School.

P.S.—My original article contained references to the ancestry of Dr. James McDonnell, to his boyhood days in the Glens of Antrim, to contemporary opinions on his method of practice. I mentioned also his association with the United Irishmen, how he organised the Belfast Harp Festival of 1792, and how he promoted the Belfast Reading Society, out of which grew the Linen Hall Library; how he was a foundation member and first president of the Belfast Literary Society, 1801, and how he organised the philanthropic Belfast Harp Society in 1806. It seemed to me that these additional records gave one a view of the man himself, but the editorial shears have excised the above and left only a record of his medical work. The above deficiencies then must not be attributed to lack of knowledge on the part of the author.—S. S.

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REVIEW

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THIS little volume has now reached its second edition, which would show that there is a definite place for it on the student's bookshelf. Those of an inquiring turn of mind would often be glad to hear more of men whose names are attached to so many anatomical structures, and for such the larger works on the history of Medicine and Anatomy are often out of reach. Here Dr. Hunter's book is of great value.

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Simple Achlorhydric Anæmia in Children

By F. M. B. ALLEN, M.D., M.R.C.P. (LOND.), and G. P. McCULLAGH, M.B., B.SC.,
from the Belfast Hospital for Sick Children.

FOR some time past attention has been directed in the periodicals of this country and America to a condition variously described as simple achlorhydric anæmia (1), cryptogenic achylic chloranæmia (2), pseudo-pernicious anæmia (3), hypochromatic anæmia (4), idiopathic secondary anæmia (5), and chronic chlorosis (6). It is true, however, that Faber (7) in 1914 drew attention to the condition, and that various continental observers have emphasised its distinctiveness from time to time. It seems to us that the term "simple achlorhydric anæmia" is the most suitable, as this includes and emphasises the common clinical features of the condition.

The disease is said to be more common in females with an age incidence of 35 to 40 years. Anæmia is the predominant feature, but it is associated with gastro-intestinal symptoms, particularly of flatulence. The anæmia is characteristic in that the red blood cells are only moderately reduced, if at all, whereas the hæmoglobin reaches very low readings, twenty-five per cent. not being unusual. Dyspnœa is prominent, and the patient, although the red cells may number over four millions, is unable to attend to her duties; whereas the patient having pernicious anæmia with a red cell count of under three millions may be capable of carrying on with his work. According to Davies (8), the "gastro-intestinal symptoms consist of discomfort and fullness after food, a poor appetite, and flatulence. These are, like the anæmia, of long standing, and may dominate the picture for a number of years. The anorexia may be more marked in the morning, and improve towards evening. Discomfort is worse after meat, and less after carbohydrate foods; in consequence, the diet becomes one of bread, potatoes, milk puddings, and tea, the patient being led by her symptoms to believe that other foods are injurious. Many of the patients, when asked, remarked on their loss of desire for any change of diet; they had not touched meat for years, and they were satisfied if they ingested small and frequent carbohydrate feeds." The complexion is sallow, resembling pernicious anæmia, but distinguished from secondary anæmia by an icteric tinge. According to some observers, there is an atrophy of the mucous membrane of the tongue, but never associated soreness. The nails are split longitudinally, and are commonly flat, or even sometimes spoon-shaped.

The gastric secretion is of particular interest, as there is absence of free hydrochloric acid, associated with, it is said, rapid emptying of the stomach contents and increased viscosity of the gastric juice. Such is a brief summary of the condition which is gathering many excellent clinical descriptions around it.

During the course of a routine investigation of two cases of anæmia who were in the ward in the Belfast Hospital for Sick Children early this year, it was found that the blood examination showed a marked reduction in hæmoglobin associated with achlorhydria. One case, Winifred G., aged ten years, had a red cell count of 5,837,500, white cells were 6,875, and hæmoglobin 35 per cent. The differential

count was—polymorphonuclears 71 per cent., lymphocytes 17 per cent., mononuclears 11 per cent., and eosinophiles 1 per cent. The other patient was Thomas C., aged eight years, who had a red cell count of 5,775,000, white cells 4,375, and hæmoglobin 30 per cent. The differential count showed polymorphonuclears 67 per cent., lymphocytes 26 per cent., mononuclears 6 per cent., and eosinophiles 1 per cent. In both cases there was total absence of free hydrochloric acid in the fractional test-meal analysis, and in both the anæmia had been of long standing.

The examination and appearance of these cases recalled to memory a family of seven children, three of whom were examined and investigated in 1926 and 1927, and who could not be placed in any recognised group in the accepted classification of the anæmias of infants and children. At that time, however, a test-meal was not examined. These children, Muriel W., Gladys W., and Harold W., were shown at the meeting of the Association of Physicians of Great Britain and Ireland at Belfast in 1927. The provisional diagnosis of a congenital family anæmia was made, for although the appearance was very like pernicious anæmia, the blood examination negated that conclusion. Conditions such as achloric jaundice and syphilis were excluded. In view of the recent interest in achlorhydric anæmia, it was decided to investigate these children further, and within the past few weeks a number of blood counts have been done, and fractional test-meals examined. Free hydrochloric acid was found to be absent in two of the children, and very much reduced in the other. It would have been interesting to have been able to report on the blood and gastric contents of all the other four children and of the parents, but this was not possible. However, we are able to present the blood examinations of two of the normal children, and a test-meal was made on another. These showed normal bloods and a normal test-meal. In June, 1927, Gladys W. was thirteen years of age, and the blood examination then showed 5,105,000 red cells, 6,400 whites, and hæmoglobin 40 per cent. Muriel W. was nine, and had 6,050,000 red cells, 6,600 whites, and hæmoglobin 35 per cent. The van den Bergh and Wassermann reactions were negative, and the fragility of the red cells was normal. It was decided to keep the children on liver and a simple iron tonic, which has been kept up constantly for over four years. In March, 1931, they showed red and white cell counts practically the same, but the hæmoglobin had risen in Gladys to 64 per cent. (70 per cent. in August, 1931), and in Muriel to 80 per cent. Fractional test-meals showed complete absence of free hydrochloric acid in Gladys, and marked diminution in Muriel. The boy, Harold W., was eighteen years in 1927, and showed a count of 3,450,000 red cells, whites 7,600, and hæmoglobin 30 per cent. The differential count showed polymorphonuclears 63 per cent., lymphocytes 31 per cent., mononuclears 3 per cent., eosinophiles 2 per cent., and basophiles 1 per cent. The van den Bergh and the Wassermann reactions were negative, and the fragility of the red cells was normal. In 1931 the reds were 3,030,000, whites 14,600, hæmoglobin 35 per cent., and differential counts the same. A fractional test-meal was examined, and total absence of free acid was demonstrated.

Olga W. is a normal child, a sister of the last three cases, and twelve years of age. Her red cells number 5,000,000, whites 16,000, and hæmoglobin 90 per cent.

Polymorphonuclears are 65 per cent., lymphocytes 28 per cent., mononuclears 4 per cent., and eosinophiles 3 per cent. Her fractional test-meal was normal.

Dorothy W., also a sister, aged twenty years, shows red cells 4,900,000, white cells 12,480, and haemoglobin 90 per cent. Polymorphonuclears are 70 per cent., lymphocytes 20 per cent., mononuclears 9 per cent., and eosinophiles 1 per cent. The fractional test-meal was not done.

It seems a reasonably fair assumption that the three affected children had absent (or in the case of Muriel W., diminished) free hydrochloric acid in 1927. Mills (5) mentions that there is sometimes a history of anæmia in other members of the family, and Witts (9) discusses the familial occurrence of simple and pernicious achlorhydric anæmia sufficiently to exclude any reference here.

Witts says that he excluded cases under ten years, as a symptomatic and transient achlorhydria is not uncommon in infantile anæmias, but we believe, as these cases show a blood syndrome along with achlorhydria similar to the condition in adults, that the condition is the same occurring in children. The examination of the family W. would suggest that the achlorhydria is not a transient phenomenon. There does not seem to be any other reasonable diagnosis, as treatment for simple secondary anæmia was unsuccessful until very large doses of iron or iron and liver were given. In addition, every possible source of bleeding was investigated, and other diseases which might have accounted for the condition were excluded. It is admitted that the nail changes are absent, and gastro-intestinal symptoms are present chiefly as deficient appetite, but this might be expected, as all observers emphasise that these are present only after a prolonged period. Lack of energy and lassitude, "being easily tired," were the chief subjects of complaint.

Another case came under observation quite recently which may also be one of the same condition. A boy, Hugh H., aged eight years, has red cells numbering 3,888,000, whites 5,300, and haemoglobin 35 per cent. His test-meal revealed total absence of free hydrochloric acid, and in five weeks, after treatment with increasing doses of iron, his haemoglobin reached 60 per cent.

The interest of these cases seems to be that the condition of simple achlorhydric anæmia described by previous observers in adults, does occur in children: three cases in separate families are noted, as well as three cases occurring in one family, the other four members of which are unaffected.

SYMPTOMS.

The appearance of the children suggested pernicious anæmia as met with in adults, the colour being sallow with an icteric tinge. Nutrition is good, but little complaint was made of gastro-intestinal symptoms, although the appetite favoured a carbohydrate dietary. Lack of energy and lassitude were marked, and these formed the main source of complaint along with the obvious anæmia. Changes in the nails have not been noted in these cases, but the spleen has been palpable in three of them. In two of the cases there was an intermittent low pyrexia for some weeks during the stay in hospital, for which there was no obvious cause. The tongue has been clean in all cases, and no complaint of soreness has been made. As regards the fractional test-meal, free hydrochloric acid has been absent in five,

and markedly reduced in one, but there has not been any excess of mucus, probably accounted for by the factor of youth. This possibly is associated with the absence of the gross gastro-intestinal symptoms described in adults. Histamine tests have not been made upon the gastric secretion in any of the cases.

TREATMENT.

Owing to the superficial resemblance to pernicious anæmia in 1927, the children W. were advised to take liver as well as a simple iron tonic. The improvement was so obvious that the mother insisted on them continuing with the liver constantly during the intervening four or five years, and the blood examination now made in 1931 shows the satisfactory progress made. According to Witts, liver alone is not of much value, but combined with iron he concluded the effect was better. The other three children who came under observation this year were treated with at least twice the ordinary dose of iron. Idozan, on account of its palatability and lack of digestive difficulties, was much favoured and quite effective. Mills recommended iron combined with copper, and some of these children have been taking a colloidal preparation of iron and copper since discharge from hospital. Josephs (10) has made a valuable contribution in this connection, that the effect of iron is first on the reticulocytes, and then on the hæmoglobin, with a latent period before the effect on the hæmoglobin is manifested, and he states that copper accelerates hæmoglobin formation, but has no influence on the reticulocytes. It is interesting to recall that the preparation of iron which is most successful in raising the hæmoglobin content is reduced iron, and it is possible that this good effect is very largely due to the copper impurity which it is known to contain. The importance of this latent period in iron-therapy is noteworthy, and it seems to be exaggerated in simple achlorhydric anæmia. We found that there was very little response to iron for some time, and that a period of as much as three weeks might elapse before there was any definite clinical improvement. In practice we need to remember that the effect is not immediate.

PROGNOSIS.

In adults this has not been considered good as regards a permanent cure, there being little tendency to spontaneous recovery. Admittedly the hæmoglobin can be raised and the condition of the nails cleared up, but continuous treatment is always necessary. Two of the children W. were observed and treated before they had reached puberty, and it is possible that in children who are treated before puberty a better ultimate result may be possible. Although the other three cases which have not reached puberty have not been sufficiently long under observation to prove this suggestion, their response lends considerable support to it.

DIAGNOSIS.

Observation of the achlorhydria associated with anæmia of the type described is characteristic. The colour-index is never above unity, and always much under this figure. There does not seem to be any reduction in the red cells, and nucleated reds were never seen. The presence of achlorhydria distinguishes the condition

from ordinary secondary anaemia. In all cases syphilis has been excluded by a negative Wassermann reaction; in the family W. this was negative in both parents as well. Adamson and Smith (6) maintain that the condition now described as simple achlorhydric anaemia is similar to those cases of chlorosis of the chronic type which do not respond to treatment with iron, and they regard the syndrome as chronic chlorosis. The blood picture is certainly very similar, and so are some of the clinical symptoms, to those cases of chlorosis which used to be fairly common in hospital wards. The recognition of certain distinctions has always been a criterion for the isolation of a new entity in medicine, and to deny the identity of a new syndrome in simple achlorhydric anaemia in which there is such a definite feature as achlorhydria does not seem justified. Witts (9) has been unable to demonstrate achlorhydria in cases of chlorosis; in fact, he states that the free hydrochloric acid is either normal or raised. The absence of a positive van den Bergh reaction and the normal fragility of the red cells excludes congenital acholuric jaundice.

There is room for considerable speculation and interest regarding the children Muriel, Gladys, and Harold W., as they appear to be the only members of the family who are affected, and they each have either total or greatly reduced free acid in the gastric contents. The condition does not seem to be related to the "familial infantile pernicious anaemia" described by Fanconi (11), which is associated with pigmentation, cutaneous haemorrhages, microcephaly, and hypoplasia of the testes. Parkes Weber, who saw these three children in 1927, considers them comparable to Fanconi's cases in a consideration of "Some Familial Anaemias and Splenomegalies" (12).

SUMMARY.

The condition of simple achlorhydric anaemia as occurring in children is described in six cases.

The features are those of an anaemia of varying intensity, particularly reflected in diminution of the haemoglobin, associated with achlorhydria.

The symptoms are those of poor appetite and marked loss of energy. Nutrition is well maintained.

The blood reveals no abnormalities apart from the diminished haemoglobin.

The response to treatment in those cases before pubescence has been good in two children, who have been re-examined after an interval of over four years. Three cases have been seen during the past nine months, and their response has also been satisfactory. One case seen after puberty has not been so satisfactory. The observations of others regarding the efficacy of iron in large doses, and its combination with small amounts of copper, have been confirmed.

Three of the cases recorded occurred in one family, the other members of which appear to be normal.

We are indebted to Dr. M. Brice Smyth for facilitating our access to some of the cases, and to Dr. Eileen Mercer for some of the laboratory observations. The boy Harold W. was at one time under the care of Dr. S. Boyd Campbell, to whom we are also indebted.

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REVIEWS

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THIS small but valuable contribution to our knowledge of the pathology of pain is based on the personal experience of Mr. John Morley as a clinician. He starts from the experience that current doctrines of referred pain and clinical conclusions based on them do not agree. He claims that the deep and superficial tenderness and muscular rigidity of the abdominal wall, observed in cases with inflammatory disorders in the abdomen, are not reflex, but are the expression of the cerebro-spinal nerves of the parietal peritoneum. He brings forward clinical evidence in support of this view, and then discusses the diagnostic significance of the conclusions reached. The book is eminently readable, and few practitioners will lay it aside until they have completed the final page.

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Infections of the Beard Region

By IVAN H. McCaw, M.B., B.CH.

Dermatologist to Royal Victoria Hospital, Belfast, and
Belfast Hospital for Sick Children

WHEN one is consulted by a man who has a skin disease on the beard region only, one immediately thinks of three diseases—impetigo contagiosa, sycosis, and ringworm. All three diseases are contagious, and may be contracted at a barber's, hence the term "dirty shave," the old term for ringworm of the beard being "barber's itch." On the other hand, it is more frequent to find these diseases in men who shave themselves, and it is sometimes problematic from whence the infection comes.

In order to make a definite diagnosis, there are some very important clinical points which must be noted. First of all, how long has the disease been present? In impetigo, the duration is usually given of a few days up to a week or two; if it is ringworm, a few weeks is usually the history; whereas a patient with sycosis says that the disease has been present with exacerbations and remissions for months or years. Secondly, what is the type of lesion one sees in each case? Therefore, I shall attempt to describe briefly these three diseases.

Impetigo contagiosa commences as a vesicle which is produced by the action of streptococci which have gained entrance to the epidermis through a slight abrasion, often during or after shaving. The roof of the vesicle, being very thin, soon breaks, allowing clear serum to exude, carrying streptococci with it. Very soon afterwards, the exuded serum dries to form brownish or amber-coloured crusts, which on removal leave raw, moist surfaces. Streptococci spread to the surrounding skin, producing fresh lesions, so that in this way and by shaving, part or the whole of the beard region may be affected. After a week or two, a typical picture is seen of raw, oozing surfaces and various-sized brownish crusts, which appear to be simply stuck on the skin. Impetigo being a superficial infection, may not be absolutely confined to the beard region, but may spread to non-hairy parts of the face and neck or on to the ears. The hairs and hair-follicles are not as a rule affected, unless the disease is complicated by staphylococci, which produce pustular lesions. In the latter case, the patient is liable to be left with the more chronic disease, sycosis, after the impetigo has been cured. As a rule, subjective symptoms are not marked, itching being occasionally complained of, and as a rule the neighbouring lymphatic glands are enlarged.

Treatment is simple, but must be thoroughly carried out, otherwise the disease may last for a few weeks. It consists of removing the crusts with any antiseptic lotion twice daily, and the application of ordinary white precipitate ointment, or of Lassar's Paste containing two to five per cent. white precipitate. A useful application after the crusts have been removed is the French "eau d'alibour," which is camphor water containing 1 in 1000 copper sulphate and 1 in 300 zinc sulphate. Antiseptics should be added to the shaving water, and only the areas of the beard

unaffected by the disease should be shaved, after which the razor and brush must be sterilised. Precautions must be taken for disinfecting articles of clothing which come into contact with the disease, such as handkerchiefs, pillow-cases, etc.

The next disease in order of duration is ringworm of the beard—*tinea barbae* or “barber’s itch.” Of this there are two varieties, one a superficial, circular, scaly type, like ordinary ringworm of the scalp, and the other a deep-seated, pustular, painful condition. The first variety is caused by a fungus called the *trichophyton endothrix*, which enters the hair-follicles and hairs in the affected area, and sometimes produces slight inflammation. This variety has to be distinguished from circinate impetigo, but microscopic examination of hairs taken from the affected area will reveal the fungus.

The second, or suppurative, variety is the more common, and is caused by a fungus called the *trichophyton ecto-endothrix*, which is of animal origin. Hence one frequently observes this type of *tinea barbae* in farmers, cattlemen, or others whose occupation brings them into contact with infected animals. The disease commences as a perifollicular pustule, but soon the surrounding follicles become infected, and usually by the time one is consulted about the disease there is a raised, red, crusted, deep-seated, granulomatous mass, which is tender and painful. When the crust is removed, it reveals pustules pierced by opaque swollen hairs, which are quite loose, and may be extracted painlessly and without any sense of resistance in the follicles. Usually there are several such areas, ranging in size from that of a sixpence to a half-crown or larger. The lesions resemble abscesses, but if incised, only a small bead of blood-stained serum oozes out. On examining the loose opaque hairs microscopically, the fungus will be seen and the diagnosis settled.

The treatment of the dry, scaly type is best accomplished by depilating the affected areas by means of X-rays, and the application of parasiticide ointments, such as ammoniated mercury, oleate of mercury, chrysarobin, iodine, sulphur, etc., or if X-rays are not available, the affected hairs should be all carefully epilated and the ointment well rubbed in.

The suppurative type is best treated by applications of boric-starch poultices every few hours for some days, to remove the crusts, and epilation with forceps of all the hairs in each lesion. The latter is not difficult, because the diseased hairs are quite loose, some of them coming away with the crusts, X-rays not being necessary. After all the crusts and hairs have been removed, iodine ointment or ammoniated mercury ointment must be regularly applied until all the inflammation has subsided. The healthy parts of the beard region should be kept shaved, antiseptic precautions being taken in doing so. This suppurative type of ringworm, like kerion scalp ringworm in children, confers immunity to all forms of ringworm later in life.

The third disease, sycosis, is the most chronic of the three, and the most disappointing as regards treatment. It is a pustular folliculitis usually caused by the *staphylococcus aureus*, and commences as a minute red spot at the mouth of a hair follicle, which rapidly develops into a pustule. Neighbouring follicles soon become infected, and the result is an area of small follicular pustules, each pustule being pierced by a hair. Later on, when the lesions become numerous and close together,

the whole affected area is red and dotted over with small pustules and crusts. One or both sides of the face may be affected, or the chin only, or the whole of the beard and moustache regions. On the upper lip it is often caused by chronic nasal catarrh, the discharge from the nose infecting the skin and hair follicles. The patient complains of burning, itching, or pain if the affected hair is touched or moved. This latter symptom is a very important one in distinguishing sycosis from suppurative ringworm, because the patient complains of pain if one tries to pull out an affected hair.

The treatment of sycosis is difficult, because the disease is deeply situated in the hair follicles, and it is practically impossible for antiseptics to penetrate sufficiently to kill the staphylococci. Therefore, one tries to prevent the disease from spreading by means of antiseptic lotions, such as perchloride or biniodide of mercury, or the "eau d'alibour" which I have already mentioned. Ointments of ammoniated mercury, oleate of copper or mercury, are also used, and if there is much inflammation, boracic ointment. Opinions are divided as to whether the patient should shave or not. In cases where there is not too much pain or inflammation, the patient should shave daily or every second day, using antiseptics in the shaving-water and antiseptic shaving-soap, the razor being sterilised after use. It is occasionally useful to apply a very thin layer of the ointment before applying the shaving-soap. The patient should be told to empty the affected hair-follicles by extracting the hairs, but, needless to say, this advice is not always carried out, on account of the pain that is occasioned thereby.

Depilation by means of X-rays is not altogether satisfactory, because it is difficult to administer the correct dose of X-rays evenly to all parts of the beard region on account of its contour, and the disease may reappear when the hair grows again. Repeated large doses of X-rays will produce permanent alopecia in the area treated, but this is to be avoided on account of the disfiguring and disastrous X-ray scar which will result.

Vaccine treatment is often disappointing, either with stock or autogenous vaccines, but recently I have been obtaining encouraging results by giving the vaccine intradermally, as advocated by Drs. Barber and Forman of Guy's Hospital (*Proceedings Royal Soc. Med.*, August, 1931, pp. 1356-1360).

Before concluding, I should like to mention briefly the important points in the differential diagnosis of these three diseases. Firstly : The duration of the disease, which is a few days to a week or so in impetigo, a few weeks in ringworm, and months or years in sycosis. Secondly : In impetigo, superficial amber-coloured crusts are the essential lesions, whereas in sycosis and suppurative ringworm the lesions are follicular pustules, the latter disease being deep-seated and swollen. Thirdly : In impetigo the hairs are not affected, in sycosis the hairs are painful to extract, while in suppurative ringworm the hairs come out without pain or sense of resistance, and show the fungus on microscopic examination.

There are several other skin diseases which may involve the beard region, for instance acne vulgaris, eczema, lupus erythematosus, seborrhoeic dermatitis, etc., but I have confined my few remarks to those diseases which usually involve the beard region only.

A Plea for the Myxœdematous

By EILEEN M. HICKEY, B.SC., M.D., D.P.H.

Mater Infirmorum Hospital, Belfast

ANY person who has had the pleasure of teaching students clinical medicine must have been struck by the difficulty they experience in eliciting a true history and an accurate account of the patient's symptoms. It is obvious to the teacher that the main reason for his inability to extract information is that they are to a large extent unaware that this information exists. Long after student days are over, one is apt to carry some of this early frame of mind into the consulting-room. This is probably more true with regard to certain diseases than others. Myxœdema is one of the most typical examples—not merely the mild cases that might possibly present some difficulty in diagnosis, but the actual fully-fledged example of the disease.

When myxœdematous patients arrive in the consulting-room, they have often surprisingly few complaints. They do not feel well, they have not much energy, they cannot walk far, they are easily tired, they feel short of breath, dizzy, etc. In other words, their vague complaints might be the complaints of almost anyone who had been leading a life rather too strenuous for their capabilities, physical or mental. They do not mention that their skin has become dry, their hands coarse and lined; they forget that their hair is falling out, and that they rarely feel warm. In fact, they rarely mention any of the classical features of the disease.

It is true that the facies of the myxœdematous patient is most characteristic. If ever a disease was writ large that all who run might read, it is certainly myxœdema. The unfortunate victims, or, to quote a textbook description, these toad-like caricatures of humanity, have their symptoms stamped on their broad, swollen features. The two points that would appear to be most characteristic in sufferers from this disease are the growling voice and the thick bluish underlip. This lip is less blue perhaps than the lip of severe cardiac disease, but always thicker. It is not a feature of the nephritic facies, but it could not be confused with the bluish, pallid lip of the severe anæmias. The voice is unmistakable—it recalls Red Riding Hood's wolf, and cries aloud for chalk. Having noted these two points, if the patient proves to have in addition a slowly-beating heart, the diagnosis is clearly beyond question, and the clinician can already feel a thrill at the thought that this pathetically ill, swollen mass of humanity will, in a very few weeks, trip lightly into the consulting-room.

It is a notable fact that one hears more sincere expressions of gratitude from these people than from almost any other type of patient. According to the more intelligent and highly educated amongst them, it is impossible for anyone who has not been similarly afflicted to appreciate the full horrors of their condition. A "living death" is a favourite description. Some have had to be led about, even carried upstairs. Work of any kind became an impossibility. Hopeless dreariness was their outlook; all joy and laughter had gone from their lives. Their very

affections died, their nearest and dearest ceased to be more to them than the man in the street.

To the casual observer it might appear that there are few conditions, if any, that could be confused with this malady. Unfortunately, however, this is not the case, and such widely differing diagnoses as nephritis, hyperpiesis, brain tumour, myocarditis, disseminated sclerosis, anæmia, are amongst the labels that some of these unfortunates carry about for years, much to their detriment. These diagnoses have no doubt been made owing to too much stress being laid on certain symptoms of the disease. One is apt to forget that these people are liable to dizziness, staggering gait, headaches, serious eye changes, epileptiform attacks, slowness of speech; that they may have a coincident secondary or even primary anæmia and a myocardium suffering as a result of such. Their pulse may be above the normal rate, they may have varying amounts of albumin in their urine, and the blood pressure may be raised. The writer can recall two cases of myxœdema who passed through typical attacks of acute nephritis, and another who has the blood-picture typical in every respect of pernicious anæmia.

One of the saddest features of these cases is that eye changes have often supervened before the disease is diagnosed, and that they do not yield to treatment like the rest of the condition. To see the large, pale, sodden tongue, that can barely be protruded, transform itself within a few weeks into the normal red active muscular organ, is to witness one of the miracles of medicine. But to see the eyes that can no longer read, and to which faces are but at best a blur, is to weep over the limitations of medical science.

One last point that should be stressed as strongly as early diagnosis is the question of adequate dosage in treatment. It is a common thing to find that the case has been correctly diagnosed, and thyroid prescribed, but that the patient after taking the prescription for months, and feeling no benefit, has eventually discarded it. It is somewhat difficult subsequently to persuade these people that the line of treatment was perfectly correct, and must be recommenced in an intensified form. A case that the writer has seen had faithfully continued an inadequate dose for some years, and has unfortunately irreparable eye damage now.

Like every other serious condition that doctors encounter, myxœdema demands both early diagnosis and adequate treatment in order to obtain results which rank amongst the most dramatic in medical science.

FORTHCOMING PAPERS

It is hoped to publish in the April number of THE ULSTER MEDICAL JOURNAL amongst other papers, the following:—

“Recent Advances in Calcium Metabolism in Relation to Clinical Medicine,” by Professor W. W. D. Thomson.

“Diagnosis and Treatment of Ante-Partum Hæmorrhage,” by Mr. C. H. G. Macafee.

“Heart Block following Coronary Thrombosis,” by Dr. S. B. Boyd Campbell.

“The Functional Divisions of the Large Intestine,” by Dr. R. H. Hunter.

Estimation of Uric Acid in Blood for Clinical Purposes

By FLORENCE BEATTIE, M.Sc.

from the J. C. White Laboratory of Biochemistry, Queen's University, Belfast

AN increase of uric acid in the blood has been noted as the first change which occurs in cases of impaired kidney function; but on the whole question of variations in the blood, uric acid has not received the attention it deserves. This is due possibly to the lack of a convenient method for its determination. The well-known methods of Folin and Benedict give reliable results, but possess certain disadvantages for extra-laboratory use. A new method recently devised by Flatow has been found to give accurate results. Its technique is simple, and requires only small quantities of blood. The method depends on the fact that uric acid is oxidised to allantoin at room temperature by potassium ferricyanide. A protein-free filtrate from the blood serum is treated with an excess of ferricyanide, and the consumption which occurs is determined by back titration with sodium indigo-sulphate. Details of the method as are follows:—

Three c.c. of haemoglobin-free serum are placed in a test-tube with 9 c.c. of distilled water, and mixed with 3 c.c. of 1.65 per cent. solution of uranium acetate. After thorough mixing, this is filtered through a dry filter-paper. 3 c.c. of the filtrate (this is equal to 0.6 c.c. of the original serum) are placed in a wide test-tube with 1 c.c. of a saturated sodium carbonate solution, and 0.6 c.c. of potassium ferricyanide (0.03914 per cent. accurately made up). 0.1 c.c. of ferricyanide solution reacts with 0.01 mg. of uric acid. After three minutes the indigo-sulphonate solution (prepared by five-fold dilution from a stock 0.22 gr. per litre, which also contains 0.3 per cent. sodium fluoride as preservative) is run in slowly from a finely graduated burette. The titration should be carried out against a white background, and is regarded as finished when the faint bluish colour remains for half a minute. The indigo-sulphonate solution, which is not very stable, must be standardised against the ferricyanide for each uric acid estimation, so that 1 c.c. is bleached by about 0.2 c.c. ferricyanide.

The amount of uric acid present follows at once from the amount of ferricyanide used up, but for simplicity the following formula may be used—

$$H = \frac{F}{M} \times \frac{A - B}{A}$$

(where H is mg. per cent. uric acid, F is the number of c.c. of ferricyanide used, M is the number of c.c. of undiluted serum taken, A and B the amounts of blue solution required in the standardisation and in the uric acid estimation respectively). The total duration of a uric acid estimation from the time of the protein removal is about five minutes.

REFERENCE.

Biochem-Zeit., 1926, Vol. 176, p. 178.

Notes on a Case of Intra-Cranial Hodgkin's Disease

By ROBERT MARSHALL, M.D., D.P.H., F.R.C.P.I.

from the Royal Victoria Hospital, Belfast

THE following notes are placed on record because of the extreme rarity of the case. Hodgkin's disease apparently very rarely invades the interior of the cranium. Reeves (1) in 1927 described a case where the orbit was involved; in 1874 Hutchinson (2) described a case showing lymphadenomatous tumours of the brain and other parts; and in 1887 Beale (3) published notes of a case where the cerebral membranes were involved.

H. M., a married woman of 32 years, was admitted to a surgical ward of the Royal Victoria Hospital, Belfast, in March, 1928, complaining of swollen glands in her neck, which she first noticed some three months before. A gland was excised, and submitted to histological examination: it was reported to be more suggestive of tuberculosis than of Hodgkin's disease. In April, 1929, she was re-admitted, this time on the medical side: she still had her glandular swellings in the neck, but since the previous October had been suffering from headache, and of some numbness of her lower lip. She was treated by "deep" X-rays, and the swelling in the neck at first showed some diminution in size. She was discharged from hospital on 4th June, but was re-admitted on 24th August, complaining of abdominal discomfort, and of a sensation of pains and needles in her right arm and leg. Her spleen was now found to be palpable, and there was impaired resonance between her scapulae, although X-ray showed no definite evidence of glandular enlargement in the chest. Her cervical glands were larger, and a "biopsy" was again performed; the report differed from the previous one, as it read: "Early fibrosis in parts, and giant-cells scattered throughout—Hodgkin's disease." She stayed in hospital one week, and then elected to go home, only to be re-admitted on 26th September, complaining of severe headache, which she then said had commenced after the removal of the gland earlier in the month, and which was occipital in site, but accompanied by pain over the right fifth cranial nerve area; she had diplopia, dizziness, and frequent vomiting which, she said, slightly relieved the headache. In addition to her previous physical signs, the right disc was noted to be hazy in outline, suggesting early papilloedema. She soon developed all the signs of an intra-cranial lesion affecting the third, fourth, fifth, and sixth right cranial nerves. There was no proptosis. Her headache could only be alleviated by increasing doses of morphia. On 26th December she had a convulsion, with marked spasms of arms and face; this was followed by a period of noisy delirium, after which she became completely comatose, and died on 29th December.

The post-mortem notes of Dr. J. C. Davison and the pathological report of Professor A. Murray Drennan are appended:—

POST-MORTEM REPORT.

The body was emaciated in its trunk and upper limbs; the lower limbs were well nourished and showed no gross wasting; both corneæ were opaque. Rigor mortis present.

The skull cap was removed; the whole brain was slightly œdematous, especially on the right side. On section, the cerebral hemispheres and cerebellum showed no lesion. The brain weighed $2\frac{3}{4}$ lbs.

Emerging from the optic foramen on the right side and lying in the middle fossa, extending to the pituitary fossa and anterior clinoid process, there was a soft mass of tissue: the part actually within the cavity of the skull measured $2'' \times 1\frac{1}{4}'' \times \frac{3}{4}''$, and involved second, third, fourth, and fifth right cranial nerves. On opening into the orbit, the orbital fat was found to be infiltrated with the same tissue.

Similar tissue was found in flat masses behind the sternum, and along the thoracic part of the vertebral column; in these regions the tissue was of a tough and almost cartilaginous consistency.

The left pleural cavity contained much fluid. The right pleural cavity contained much less fluid than the left. The lungs themselves were congested at the bases, but showed no other sign of disease. The pericardium contained a little fluid.

The heart was very small (weight 6 ozs.), but was otherwise normal.

The liver weighed 3 lbs.; on section it appeared a "nutmeg" liver.

The spleen showed marked perisplenitis (weighed 18 ozs.); it was grossly nodular and much enlarged.

The pancreas was embedded in and pushed forward by a huge mass of enlarged glands, which spread downwards along the anterior aspect of the vertebral column, embedding the abdominal aorta, and dividing into two directions to follow the great iliac vessels. On removal as completely as possible, this mass of tissue surrounding the abdominal aorta was found to weigh $1\frac{1}{4}$ lbs.

The kidneys and suprarenal glands were apparently normal. Each kidney weighed 5 ozs.

Cause of death: Hodgkin's disease (with masses in abdominal glands, spleen, and orbit).

MICROSCOPICAL FINDINGS.

Only portions of spleen, pancreas, liver, and orbital mass sectioned.

Spleen.—A few portions show only engorged pulp in which there are many pigment-laden cells. The greater part is altered by lesions of the Hodgkin type—some of these show as fibrous knots or circles along the line of the arterioles, more show as areas of necrosis with fibrosis around, and punctuated here and there with hæmorrhages. The cellularity varies—the dense fibrous parts have only a few nuclei of the fibroblasts showing; the more cellular areas of fibrosis show fibroblasts, a few lymphocytes and plasma cells, and a variable number of large clear cells, amongst which are actual giant cells with convoluted or lobed nuclei (the D. Reed type of cell). Eosinophiles occur in the surviving pulp and at the margins of the necrotic cells, but are not numerous.

The appearances as seen in this organ are those usually labelled lymphadenoma.

Pancreas.—A mass of similar tissue to the above-described lymphadenomatous tissue occurs at one side of the pancreatic tissue, and surrounds the splenic artery and adjacent structures. It invades the adjacent pancreas, causing atrophy of the pancreas. Elsewhere the pancreas is not involved, and has the usual appearances, plus considerable congestion of its blood vessels.

The Tissue from Orbit.—All parts of this show the same general appearances, viz., those already described in the spleen. The number of large cells varies in different areas, in places numerous, in places scanty—the latter usually where the fibrous tissue is dense. Some parts show large numbers of eosinophiles.

The Liver.—Shows only some venous engorgement and fatty change; no lymphadenomatous invasion.

The histological appearances in the spleen, mass near pancreas, and mass in orbit, are those of lymphadenoma. The only unusual feature is the distribution of the lesions.

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- (2) HUTCHINSON, J. H., "Case of adenoid (Hodgkin's) disease: enlargement of the cervical glands with multiple lymphadenomatous tumours of the brain, spinal cord, etc.," *Trans. Coll. Phys. Phila.*, 1874-5, 3s, 47-67.
- (3) BEALE, E. C., "A case of lymphoma affecting the larynx, eyelid, and cerebral membranes," *Lancet*, 1887, II, 749.

The Ulster Medical Society

THE opening meeting of the Society was held on Thursday, 15th October. Mr. S. T. Irwin was installed President for the Session 1931-2, by the outgoing President, Mr. Henry Hanna. Before giving his Presidential address on Enlargement of the Mesenteric Lymph-glands (page 5), Mr. Irwin referred to the generosity of Mr. Hanna in presenting a beautiful cup to replace the Robb Golf Cup, which had been won outright. He then spoke of the loss which the Society had sustained in the deaths of a number of its Members and Fellows.

One of these was a distinguished Honorary Fellow, Lorrain Smith, who in 1894 was appointed to the newly-created Lectureship in Pathology in the old Queen's College. When this Lectureship was replaced by a Professorship, he was the first occupant of the Chair. In 1904 he was called to be Professor of Pathology in the University of Manchester, and later still he held what may be regarded as the most distinguished post in the subject of Pathology—the Professorship of Pathology in the University of Edinburgh.

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During the ten years he spent in Belfast he took a prominent part in the life of the School and the work of this Society. In 1904 he was called to the Presidential chair. His departure from Belfast at this time prevented him from actually holding the position. He did, however, leave the impress of his dominating but engaging personality on the School and the young graduates of the time. He was a firm believer in pathology, then in its infancy, as the basis of all medical study, and infected his students with enthusiasm for the subject. He was elected an Honorary Fellow of the Society in 1906.

James Huston was a double graduate of the old Royal University of Ireland, obtaining the B.A. in 1888, M.B. in 1895, and proceeding to M.D in 1908. He was one of our oldest Fellows, and a former vice-president of the Society. He was held in high esteem in the town and district of Carrickfergus, where he practised.

On 4th August died William Burns.

Probably he would have desired no more elaborate epitaph. Qualified in 1901, he did special plague duty in India for two years, before settling down to a large and busy practice in his native city of Belfast. He played a prominent part in the recent negotiations with the Government, when medical benefit was extended to Northern Ireland. Deeply interested in all social problems, and widely read, he held strong views on many public questions of the day. These views he always stated clearly, and when he had spoken there was no possibility of ambiguity as to what he meant. Constant in his attendance at our meetings, frequently taking part in our clinical and scientific discussions, his outspoken but always friendly criticisms will be missed.

No more tragic death has occurred in connection with our School for many years than that of Pauline Campbell (née Heron). Stricken with what she knew to be a mortal disease, in the midst of the joys of early motherhood, and in the presence of a young and loving husband—seldom has fate been more heartless. She graduated as recently as 1924, with first place and first-class honours.

John Robert Davison was one of the oldest Fellows of the Ulster Medical Society, which he joined in the year 1887. In that year he obtained his M.D. degree, and at once settled in practice in Belfast. Though he belonged to the older school of practitioners, he kept himself wonderfully abreast of modern methods. He had a very human sympathy for his patients and their ailments, as well as a genial disposition which rapidly gained, and for years held, a very large practice.

In 1917 he read a thoughtful and practical paper on "Tuberculosis—a Public Health Problem." This paper is still within the recollection of some of us. We shall all miss his friendly presence.

It is given to few busy practitioners of medicine to leave behind them such a record of public service as that of the late Richard Whytock Leslie. A member of the Senates of the old Royal and our own Queen's Universities, a member of the Council of the British Medical Association, for thirty-five years a member of the staff of the Ulster Hospital for Children and Women, Medical Officer to Campbell College from its foundation in 1894, as well as a former President of the Ulster

Medical Society—to mention only the more outstanding of the positions which he held. As President of this Society in 1912-3, many of us still remember his interesting and exhaustive address on “Infectious Diseases Incidental to School Life.”

An easy, graceful, and fluent speaker, with a wide knowledge of affairs, both medical and general, as well as a high code of professional honour, he held in a remarkable manner the respect and esteem of his professional brethren.

He gave ungrudgingly of his time and energies to university and medical affairs. When the Royal University was abolished he was granted the honorary degree of LL.D., a token of appreciation of his valuable work on its Senate.

The second meeting of the Society was held on Thursday, 29th October. In the unavoidable absence of the President, the chair was occupied by Professor W. J. Wilson, vice-president. Three short papers were presented. The first was by Doctors F. M. B. Allen and G. P. McCullagh: “Achlorhydric Anaemia in Children.” The second was by Dr. Ivan H. McCaw: “Infections of the Beard Region.” These two papers are published elsewhere in this number of the Journal. The third paper was by Dr. R. H. Hunter: “The Functional Divisions of the Large Intestine.” It is hoped to publish this paper in a future number.

The third meeting of the year was held on Thursday, 12th November, when Dr. Brailsford of Birmingham gave a most instructive paper, entitled: “Radiological Appearances and Diseases of the Hip-Joint.”

The President, Mr. Irwin, was in the chair. Dr. Brailsford's paper was constructed around a large collection of extremely interesting radiograms, illustrating all the common conditions and many of the rarer diseases affecting the hip-joint. He first described the variations in the normal hip-joint which are sometimes seen, including the accessory bone, the *os acetabuli*, which may be mistaken for a fracture. He next dealt with congenital dislocation of the hip-joint. He was of the opinion that the results of operation and manipulation were by no means uniformly satisfactory, and that arthritis of the affected joint was less likely to occur in untreated cases at a later date. He next described the condition of adolescent *coxa vara*, and pointed out the complete separation of the head and neck which occurs in extreme cases, and which may lead to a mistaken diagnosis of fracture. He described the triangular area of irregular calcification which occurs in the neck of the femur in this condition. He then passed to slipped epiphysis, and gave a very complete account of the current theories regarding its causation and pathology. Dr. Brailsford thought that trauma could not be excluded in all cases as a possible cause. A description of Perthe's disease followed. He next dealt with the conditions generally termed tubercular infection of the hip-joint, and showed several examples of the earliest types of the disease. From Dr. Brailsford's remarks it would seem that in Birmingham it is more common to find a very early erosion in bone, or a small abscess in bone, than one sees in Northern Ireland. Dr. Brailsford then discussed infective arthritis, osteo-arthritis, and syphilitic diseases involving the hip-joint, and concluded by showing examples of some of the rarer

diseases, such as Paget's disease, secondary carcinoma, Ewing's tumour, osteomalacia, and chondro-osteodystrophy.

The fourth meeting of the Session was held on Thursday, 26th November. The President, Mr. S. T. Irwin, was in the chair. Mr. John F. Ward, B.Sc., of Crooks Colloidal Laboratories, London, read a paper, entitled: "A Description of the Colloid, and Its Adaptation to Vital Process." Mr. Ward first traced the history of colloid chemistry from its origin with the work of Graham seventy years ago, and then drew attention to its very wide extension within recent years. He pointed out that the original classification of all substances into the colloid or non-diffusible and crystalloid or diffusible was now too limited for our present knowledge. Faraday was the first to demonstrate that many metals could be produced in a colloidal form. A cinema-film was shown, illustrating the properties of solutions of colloidal gold, silver, and sulphur. The Tyndall beam was used to demonstrate their non-homogeneous character; on more minute examination under the microscope the actual particles could be seen as a scintillating cloud of bright bodies in rapid Brownian movement. The effect of adding protecting substances, such as gelatin, to the colloidal-metal solution, was shown; the metal could no longer be so readily precipitated by the addition of salts. That the colloidal particles were electrically charged was proved by their behaviour in an electric field, and in this way the greater precipitating power of the more highly charged salt ions, such as calcium, was explainable. The speaker went on to compare the relative sizes of bacteria, colloidal particles, and the most minute living bodies—the filterable viruses. Utilising the different sizes of various dye molecules, he gave a beautiful demonstration of the methods of separation by membrane filtration. A second film was shown to illustrate the preparation of colloidal gold solution and its use in Lange's gold test for G.P.I. and lues of the central nervous system. The extreme care in the preparation of the gold solution was stressed.

The lecturer next proceeded to describe the peculiar bactericidal action of metals when brought in contact with actively growing cultures. Various theories for this action were discussed. The question of the nature of the metal calcium in the blood and the mechanism of its regulation was a most important one. Less than one-millionth of a grain of irradiated ergosterol was sufficient to prevent rickets in a rat fed on a diet deficient in vitamin D. The potency of such a minute amount showed the importance of vitamins in the animal body. The standardisation of preparations containing these vitamins was described, and the standards at present used for vitamins A, B, C, and D were defined.

In conclusion, a film illustrating the technique used in the intravenous injection of sterile gum saline was shown.

The Annual Dinner of the Society was held in the Medical Institute on Thursday, 3rd December. There was a very large attendance of Fellows and Members. The guests included His Grace the Governor of Northern Ireland, the Lord Mayor of Belfast, the Vice-Chancellor of Queen's University, Belfast, the Rt. Hon. Mr. Justice Brown, Mr. McConnell (Dublin), and Dr. Cunningham (Londonderry).

Londonderry Medical Society

THE first meeting of the Londonderry Medical Society for the session 1931-2 was fixed for 16th October, but owing to the illness of the President-elect, it was postponed until 30th October. On this date, however, it was again found to be impossible to carry out the original programme, but at short notice several members came forward with brief notes, case histories, etc., and the meeting, which was favoured by a large attendance, turned out to be most instructive.

The following office-bearers were elected for the ensuing session:—Dr. J. G. Cooke, the President-elect, was unanimously elected President; his enforced absence through illness was commented upon by the outgoing President, Dr. H. W. Cunningham, and the hope was expressed that at the next meeting Dr. Cooke would be able to deliver his Presidential address. The Hon. Secretary and Hon. Treasurer, viz., Dr. J. A. L. Johnston and Dr. J. Watson, were re-elected.

Dr. Killen opened the meeting with a description of a case of sympathetic ophthalmia, which was successfully treated with neo-salvarsan. Discussion centred on the diagnostic significance of the relative large mononuclear leucocytosis, which in this case was found to be thirty-four per cent. in a differential count.

Dr. Eaton initiated a discussion on the surgical problems which arise from the swallowing of various forms of foreign bodies, such as artificial dentures, pins, pebbles, etc., and illustrated his remarks with a very interesting series of radio-grams, and showed convincingly the necessity of these in solving the problem of what surgical measures have to be taken in any given case. As may be expected, this subject proved to be a fruitful one for discussion.

Dr. McGinley related the case of a patient who was brought into the Letterkenny Hospital as an emergency. The woman was obviously ill, and examination of the abdomen revealed a large tumour in the pelvis, which appeared to be an ovarian cyst. On vaginal examination, this could be distinctly felt in the pouch of Douglas. On operation, the cause of this tumour was found to be an enormously distended gall-bladder, the fundus of which extended right into the pelvis. On removal it was found to contain two pints of fluid and pus and hundreds of small gall-stones. In the shocked state of the patient before operation, it was impossible to determine if there was any jaundice present or not, and there was, of course, no time for any clinical examination to be made. The patient made a complete recovery.

Dr. Alexander then followed with a description of an operation for flail-foot on a patient who had had infantile paralysis. A demonstration of the result was given, showing how the manifest improvement was obtained.

Dr. Johnston concluded the proceedings with a demonstration of the clinical bacteriological methods used in isolating *B. typhosus*. A description was given of an outbreak of typhoid fever which occurred in Donegal this autumn, one nidus being in Lifford and the other in Ramelton, separated by fifteen miles, and the suspicion arose as to the possibility of there being a carrier. The steps taken to prove this possibility were next gone into, which resulted in a keen discussion regarding the carrier question, and the problems arising in their treatment.

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

PORTADOWN AND WEST DOWN DIVISION, B.M.A.

A MEETING of the Portadown and West Down Division of the B.M.A. was held in the C.B. Café, Scotch Street, Armagh, on Wednesday, 28th October. Professor W. J. Wilson, Queen's University, Belfast, read a paper on "The Place of the Laboratory in Diagnosis and Treatment." A few of the points to which Professor Wilson drew special attention were :—

(1) That the diagnosis rests with the medical practitioner, the laboratory worker furnishing him with a additional sign which, taken in conjunction with other signs and symptoms, may be helpful to him in his treatment.

(2) Material for histological examination should be sent in a bottle containing ten per cent. formalin. Tissue for cultural examination should be wrapped in sterile gauze surrounded with oiled silk.

(3) For blood cultures, 5 to 10 c.c. of the blood should be injected into a bottle containing about 100 c.c. sterile broth.

(4) For serological tests, 2 to 5 c.c. should be sent in tubes, and the use of glass capsules and capillary tubes abandoned.

Such tests include the Widal test for the typhoid-paratyphoid group, the Weil-Felix reaction for typhus, the diagnosis of undulant fever, the Wassermann and Meinicke reactions for syphilis and its sequelæ.

The serum in these tests should be clear and free from hæmoglobin, hence the syringe and needle used in its collection should be dry or rinsed out with normal saline solution. It should not contain alcohol or ether, neither should it be heated. If glass capsules are used, their ends should be closed with melted sealing-wax.

In the interpretation of the result of a serological test, the doctor should remember that a negative result obtained before the tenth day of the disease is of little significance.

(5) For the examination of blood diseases, films should be sent on a glass slide.

(6) For a determination of blood-urea and blood-sugar, the blood should be prevented from clotting by the addition of a trace of powdered potassium oxalate. A tiny portion from the point of a penknife should be added to the dry and empty tube employed to receive the blood. In the blood-sugar test, the addition of excess of the potassium oxalate might lead to hæmolysis and vitiate the results. In both cases, 5 or 10 c.c. of the blood should be sent.

(7) As much morbid material as possible should be attached to swabs sent for microscopic and cultural examination.

A positive result of the direct examination of a diphtheritic swab is of great significance; a negative result of little. Vaginal, cervical, and urethral swabs from cases of gonorrhœa in women, usually give negative results as regards the discovery of the gonococcus.

(8) For the discovery of the *Spirochaeta pallida*, a swab from the chancre is useless; serum from the base of the chancre should be collected in capillary tubes.

(9) The first portion of cerebro-spinal fluid should be kept apart from that received for examination, as the first portion not infrequently contains blood from puncture of a vein.

(10) The isolation of typhoid and paratyphoid bacilli from faeces is now a much easier task than formerly, and is now as reliable as blood culture in the diagnosis of enteric fever before agglutinins appear in the blood.

(11) Urine should be collected with as aseptic precautions as possible, even for examination for tubercle bacilli. The presence of pus in urine unaccompanied by *B. coli* is very suggestive of tubercle infection, even where the tubercle bacillus cannot be found. The presence of contaminating *B. coli* might lead to a wrong diagnosis of *B. coli* infection.

(12) As regards Public Health examinations of water and milk, it should be remembered that the bottles and corks should be sterile, and that for bacteriological examinations six or eight ounces of water and two ounces of milk are sufficient.

For a chemical examination of water, a Winchester Quart bottleful is necessary.

A negative result as regards the finding of the typhoid bacillus in water or milk is of no significance, and does not rule out these materials as having been vehicles of infection.

Dr. T. B. Pedlow reported a case of undulant fever which he had met with in his practice. He commented on the frequency with which this fever occurs in Denmark, an agricultural country very similar to ours, and how seldom it seems to occur here. Is it not recognised? Or are Irish people immune?

Dr. H. C. C. Deane showed a demoid ovarian cyst containing hair and a well-developed tooth.

CHARLES J. BOUCHER, *Hon. Secretary.*

NATIONAL HEALTH INSURANCE NOTES— BELFAST AREA

MEDICAL BENEFIT has now been in operation in Northern Ireland for one year, and, I think everyone is agreed, with advantage to both patients and doctors. The fact that there have only been three complaints of a minor nature before the Medical Services Committee speaks well for the efficiency of the service.

It is unfortunate that Insurance practitioners of Northern Ireland should only have had one year of payment at the full capitation rate of nine shillings, but it is to their credit that they at once unanimously agreed to the ten per cent. reduction brought about by the economy proposals, and I believe their readiness to shoulder their share of the national burden will react in their favour when the time comes to ask for a reconsideration of the present capitation rate, which is utterly inadequate for the services rendered. The more efficient the service is made, the stronger will be the claim of the practitioners when a reconsideration of the terms is made.

The question of the high cost of prescribing is still disturbing the Ministry of Labour, as the recent circular issued by the Central Practitioners' Committee to all Insurance practitioners shows. In our next issue I shall deal with these difficult problems.

I regret to have to record the deaths of Doctors W. Burns, R. W. Leslie, J. R. Davison, and J. A. Hutchinson, during the first year of Medical Benefit. The profession, as well as the public, will feel their loss, as they took a deep and active interest in everything for the welfare of our profession.

The Medical Guild of Belfast Insurance Practitioners held its annual meeting in the Institute on 3rd November last, and one felt sorry that the attendance was not worthy of such an important Society. Dr. D. Gray, the outgoing chairman, kindly entertained the members to tea before the meeting.

I congratulate Dr. Robert Thompson on being appointed chairman for the ensuing year. The Guild has made a wise choice, as Dr. Thompson has had many years' experience of the office, being chairman of the Public Health Committee of the Belfast Corporation. Dr. William Calwell was elected to the vice-chair, and Dr. D. Rodgers is again secretary and Dr. Cahill treasurer. These office-bearers should ensure a successful year for the Guild.

The Medical Guild at its meeting on 31st November passed a resolution that Insurance practitioners should not accept transfers from a practice that had been sold, unless the special consent of the purchaser had been given. On this, of course, rests the value of the practice to both purchaser and vendor. The Ministry of Labour has consented to amend the notice sent out to insured persons on the retiral or death of a practitioner. The use of paragraph A on the medical card under such circumstances was unfair to the purchaser and vendor, and its deletion causes no hardships to insured persons.

S. McCOMB.

COUNTY AREAS

It seems that the time has now arrived when we medical men in Ulster must, as a matter of self-preservation, of economic and ethical necessity, co-operate so as to be able to speak with one voice in the defence of our interests. By mutual and friendly understanding and co-operation we can help each other, and thus avoid friction, either between ourselves or with the Insurance authorities. To those who have not already done so, it is advisable to form Guilds, so that each district can exchange views, and express to each other difficulties or grievances as they arise, in order to have them solved in a proper manner. If each practitioner takes a line of his own without consultation or sympathy, or in a spirit of rivalry, confusion must inevitably result, with the consequences reacting most upon himself. A free and informal discussion of this nature took place at the October meeting of the Lisburn and District Medical Guild. Matters affecting the interests of panel practitioners were raised. The proposed mode of procedure to be adopted soon, in cases of doubt or of attempted malingering, was also considered. The reduction of remuneration owing to the present financial stringency was discussed, also the importance of not over-prescribing, so as to save the Insurance Fund any overcharges. With due regard to the interests of our patients, it behoves all of us to see that such overcharges do not occur as far as we can prevent them. But to attain this end, it is impossible to avoid the obligation of each prescriber to master the details of pricing. These appear to need drastic overhauling, being complicated, and in some particulars not a little foolish. The average charge per head for drugs is now much too high, but with time and experience a considerable saving is to be expected.

J. S. PEATT, 14 Railway Street, Lisburn.

OBITUARY.

JAMES ALEXANDER LINDSAY, M.A., M.D., F.R.C.P.(LOND.).
Emeritus Professor of Medicine, Queen's University, Belfast.

MANY generations of Belfast medical students will learn with regret of the death of Professor James A. Lindsay, which occurred on Tuesday, 15th December, 1931. "Jimmy" Lindsay, as he was affectionately known to his students, probably exercised a greater influence on the Belfast Medical School than any other figure of his time; while the gentleness of his disposition and the kindliness of his nature earned for him the deepest affection of the members of his classes. He was a man of wide culture far beyond the confines of his profession, and he contributed in a material degree to the fame of the Belfast Medical School, adding lustre to its history as one who always strove for the highest ideals of his profession.

Professor Lindsay was a past President of the Ulster Medical Society, and of the Ulster Branch of the British Medical Association; he was a Fellow of the Royal Academy of Medicine and of the Royal Society of Medicine, to all of which he brought distinction by his contributions to their discussions.