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1931–32

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## OBSERVATIONS ON ENLARGEMENT OF THE MESENTERIC LYMPH - GLANDS<sup>1</sup>

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

<sup>1</sup> This was the first paper of the first issue of the Ulster Medical Journal, but while it acknowledges Mr Irwin's Presidency of the Ulster Medical Society, it does not definitely state that this was his Presidential Address.

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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-caecal 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

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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 leukocytosis 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 coeliac 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-caecal valve, the appendix, and the caecum, are drained by the ileo-caecal 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 caecum 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-caecal glands do not discharge into the superior mesenteric glands. There is a lymph-shed between these two adjacent sets of lymph vessels. The ileo-caecal 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 caecum 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-caecal valve, the caecum, 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 coeliac 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-caecal group

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of lymph-glands extending upwards from the ileo-caecal 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 caecum where the stream of contents from the ileo-caecal valve strikes it, and (3) in the ileo-caecal 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-caecal 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-caecal 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-caecal 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-caecal 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 coeliac 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-caecal type of intussusception, gross enlargement of the ileo-caecal 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

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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 anaesthesia.

## NATURE AND ROLE 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-caecal 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-caecal 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-caecal 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-caecal 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

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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-caecal 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

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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 axilla, 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-caecal 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.

If 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.

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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-caecal 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-threequarter playing school football in the winter 1926x27, 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-caecal 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-caecal 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.



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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-caecal 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. Alkalis do not relieve. Appetite good. Bowels constipated. Micturition and menstruation normal. Loss of weight recently.

On examination, patient is pale and anaemic. 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.