2. The wasting of muscle is frequently not manifest until many years have elapsed since the original injury.

3. The problem of the way in which the atrophy is brought about is discussed.

4. From the medico-legal point of view alone the subject is of very great importance.

I am indebted to Dr. M. E. Disney, medical registrar, for assistance with the case notes at the Bristol Royal Infirmary.

REFERENCES

Alajouanine, T., Gopcevitch, M., and Meillaud, P. (1930). *Rev. neurol.*, **1**, 429.
Brodin, Lhermitte, and Lehmann (1931). Ibid., **1**, 191.
Geyer, H. (1934). *Dtsch. Z. Nervenheilk.*, **134**, 14.
Lhermitte, J. (1930). *Gaz. Hôp.*, **103**, 873.

AETIOLOGY OF ACUTE APPENDICITIS*

BY

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I. The History of Acute Appendicitis

Although appendicitis is usually regarded as a disease of modern civilization a small number of Egyptian mummies have been found to possess unmistakable signs of chronic inflammation of the organ, and in one case, that of a young royal princess, an acutely inflamed and perforated appendix was beautifully preserved.

The first recorded case in modern Europe was in a patient of Mestiver (Germany) in 1759, but the disease does not appear to have been recognized in England until 1812, when Parkinson diagnosed it in London. For some years the aetiology was obscured by the use of the term perityphlitis; but after 1820 appendicitis was increasingly recognized. Between 1820 and 1840 there were thirty-three recorded cases, and during the twenty years 1840–60 the number had risen to 102. In 1918 the *deaths* from the disease totalled 2,416. The figures showing the incidence of the disease at the Bristol Royal Infirmary are of interest. In 1880 out of 2,591 patients there were four cases of appendicitis. By 1905 the proportion had risen to sixty-four cases out of 3,762 patients, and by 1918 it had reached 113 appendicectomies in 4,021 patients.

DISTRIBUTION OF THE DISEASE IN THE COMMUNITY

Both sexes are equally prone to the disease, and the age of greatest liability is from 10 to 30 years. While it does occur in infants it is rare to find the disease before 3 years of age. It is also rare to find it at the other extreme of life, but it has been known to occur at 84 years. The disease is more serious at the extremes of life than it is during the years of its maximum incidence. As regards the influence of social position, the disease is definitely more common among the well-to-do; poorer people, especially those living in institutions, enjoy a relative immunity. For instance, there was only one case in ten years at Portland Prison. At Clifton College, Bristol, twenty cases occurred among 500 boys in five years, while at Müller's Orphanage, also in Bristol, there were only four cases among 950 children in the same period.

NATIONAL DISTRIBUTION

The disease is commonest in the United States of America, where its incidence is nearly twice what it is in Great Britain. Holland and Sweden have an incidence equal to our own, but the disease is less common in Denmark, Italy, Spain, and Greece. In the West Indies it is extremely rare among the natives but relatively common among the white population. In the villages of India and China the disease is unknown, but it is seen in the towns, both among the white population and those natives who adopt a European diet. This in fact is true all over the world. Native peoples, among whom the disease has been previously unknown, become very prone to it as soon as they live on a European diet. In seeking a cause of appendicitis the following summary of historical facts will be found helpful.

1. The disease was present, but relatively rare, in this and other countries until the end of the nineteenth century, since when it has become increasingly common in most countries. The rise in England began in 1890, and was pronounced between 1895 and 1905. From 1905 onwards it has been fairly stationary.

2. The rise was at first most marked in the towns; later the rural districts were affected, until at present the incidence is the same for town and country.

3. Inmates of institutions, living on a plain diet, are less prone to the disease than are more well-to-do people.

4. The privations of the war did not affect the frequency of the disease.

5. Appendicitis is unknown in wild animals, while it is common among animals in captivity.

THEORIES OF CAUSATION OF APPENDICITIS

As with all diseases of unknown causation, theories of the cause of appendicitis abound. One of the earliest was that it was due to the replacement, by the water-closet, of the squatting position in the act of defaecation. Another was that the fault lay with the substitution of stone flour rollers by steel ones, but no pieces of steel have ever been found in the appendix.

Of greater interest is the theory that the disease is due to an alteration in the food habits of the people, resulting in a marked reduction of the cellulose intake. During the last forty years English diet has changed very considerably, and it is of interest to examine these changes to see whether they have any bearing on the causation of appendicitis.

(1) Since 1890 there has been a tremendous increase in the consumption of cocoa and chocolate; but these articles can be dismissed at once, since animals in captivity which develop appendicitis do not eat them.

(2) *Bananas.*—West Indian natives eat large quantities of bananas, but they do not develop the disease.

(3) *Butter.*—This commodity has always been plentiful.

(4) *Meat.*—There is a very close resemblance between the increase in appendicitis and the rise in imported meat in the period 1895–1905. During these years our meat imports doubled, both beef and mutton being affected. There is no doubt but that the average consumption of meat per head of the population increased parallel to the rise in appendicitis, but if we examine the problem further we find that large sections of the community were heavy meat-eaters before 1895. In 1889, for instance, Pearce's dining-rooms in London supplied 30,000 carmen daily with a lunch containing 5 oz. of meat, and each soldier in the British Army was allowed 12 oz. of meat a day, yet the incidence of appendicitis among them was very low.

(5) *The Reduction in Cellulose Intake.*—Between the years 1890 and 1915 the consumption of preserved meats, butter, tea, cocoa, chocolate, rice, bananas, etc., increased considerably, as has been mentioned, so that the older, coarser, cellulose-containing foods, such as swedes, turnips,

* The successful thesis entered for the Martyn Memorial Scholarship in Pathology in the University of Bristol.

carrots, parsnips, leeks, and cabbages, have been crowded out, and it is suggested that this reduction in the cellulose content of the diet is at least a prominent predisposing cause of appendicitis. The following points are of importance in this connexion:

(i) The time incidence is correct. Both occurred between 1895 and 1905.

(ii) Cellulose foods were in favour longer in the country than they were in the town, whereas nowadays country and town diets are alike.

(iii) In institutions the older, cheaper cellulose foods persisted longer than among the well-to-do.

(iv) Apes in captivity do not get the coarse fibrous food they do in the natural state.

(v) Rabbits on a cellulose-free diet get appendicitis.

(vi) The disease is also very common among the inhabitants of Tristan da Cunha, who live on an exceptionally low cellulose diet owing to the impossibility of growing cereals on the island.

OTHER CAUSATIVE FACTORS

1. It has been suggested that there is a relation between appendicitis and sore throats.

2. Trauma has inevitably been suggested as a cause.

3. Foreign bodies are sometimes found in inflamed appendices and may be at fault. Especially is this so when the foreign body is one, or more, oxyuris.

4. Faecal concretions are present in 15 to 20 per cent. of cases and in 10 per cent. of apparently normal appendices. Krogns found concretions in 35 per cent. of perforated appendices and in 27 per cent. of non-perforated appendices.

5. Kinks of all kinds have been accorded a causative function.

6. The disease has been said to follow chronic intestinal stasis.

II. The Bacteriology of Acute Appendicitis

Aschoff has done a considerable amount of work on the bacteriology of the disease, and gives his results in his monograph on appendicitis. The point he stresses is that the distal part of the appendix, where the inflammation usually starts, has a special bacterial flora of its own, different from that of the proximal part of the organ and certainly very different from the caecal flora. As examination proceeds from the proximal to the distal end of the organ there is first a reduction in the larger bacteria, especially of the spore-bearing organisms of the gas-gangrene group. There is a similar but less rapid reduction in the coliform Gram-negative organisms. The closer one gets to the apex the more Gram-positive organisms there are, but the larger cocci, streptococci, and diplococci disappear, until there only remain fine Gram-positive diplococci and rods (enterococci A and B, haemolytic streptococci). Smears from acute appendices do not usually show a pure culture; some coliform Gram-negative organisms are always present and may even be in pure culture. This bacterial flora is found equally in healthy and acutely inflamed appendices, and the important question arises as to how the fine Gram-positive organisms at the apex suddenly take on an increased virulence, sufficient to cause an acute inflammation of the organ. To this question Aschoff knows no answer. Ricker states that it is due to a circulatory disturbance of nervous origin, causing haemorrhagic infarction of the mucosa through which the organisms can enter. But appendices removed early in the course of the disease do not show this, and Aschoff maintains that no *demonstrable* circulatory disturbances arise.

Stagnation of Faeces.—Aschoff's experience is that acute appendicitis occurs more often in empty appendices than in those containing faecal matter, and he dismisses the presence of faecaliths as being purely fortuitous. He points out that the inflammation begins *distal* to the faecalith, not around it. (These points are discussed later.) Contrary to Rendle Short, Aschoff holds that diet is of no importance, except that perhaps the amount of food taken may be of some consequence. In his opinion figures concerning frequency of the disease among the various classes of people are useless, as they only take into account the cases which come to operation. But he is probably wrong here, for in such a disease the total incidence and the number of cases operated upon run parallel to each other. Aschoff believes that curved or bent appendices are more prone to the disease than straight ones.

Hilgermann and Pohl contend that the disease is caused by oral organisms which have reached the appendix; and while this may be true for the very few cases in which the pneumococcus has been found in pure culture, it probably does not hold generally, since one would usually expect a preceding history of rhinitis and/or pharyngitis from the virulent organisms.

III. The Purpose of the Present Investigation

This investigation was undertaken in order to elucidate, if possible, certain points about which Aschoff says more information is needed and to see whether the information gained would shed any light on the causation of acute appendicitis. In addition, a number of factors about which there is a difference of opinion and some factors of general interest were investigated. The investigation dealt with the following points:

1. The proportion, position, and importance of faecaliths and strictures.

2. The proportion of empty and full appendices affected.

3. The incidence of sore throats preceding the attack of acute appendicitis.

4. The proportion of bent and straight appendices affected.

5. The proportion of cases showing various symptoms, such as constipation, vomiting, etc.

6. The length of time taken for suppuration, gangrene, and perforation to appear in order to determine whether it is possible to construct a time-table of acute appendicitis, so that its condition may be clinically diagnosed from the time of onset of symptoms.

IV. Data obtained during Investigation

Proportion of Bent or Straight Appendices Affected

Bent appendices	15
Straight appendices	5

Incidence of Faecaliths and Strictures

Faecaliths only	14	Multiple faecaliths	3
Strictures „	9	Neither	3
Faecaliths and strictures	4		

Empty or Faeces-containing Appendices Affected

Containing faecal matter	15
Empty	5

Incidence of Various Symptoms

Constipation	8	Pain starting in R.I.F. and later moving to umbilicus	2
Vomiting	18	Diarrhoea	1
Pain starting at umbilicus	15		
„ „ in R.I.F.	3		

Incidence of Sore Throats

Acute sore throat up to 7 days before appendicitis	5
Chronic sore throat	2

Average Time Taken for Various Stages of Inflammation to Develop

Catarrhal inflammation	.. ..	22 hours	(5 cases)
Suppurative „	.. ..	24 „	(3 „)
Gangrene	.. ..	26 „	(4 „)
Perforation	.. ..	42 „	(8 „)

Range of Temperatures found in the Series

Subnormal	2	100.1°–101°	1
Normal	1	101.1°–102°	3
98.5°–99°	5	102.1°–103°	1
99.1°–100°	7		

Range of Pulse Rates found in the Series

80–85..	6	101–105	0
86–90..	4	105–110	1
91–95..	4	111–120	4
96–100	1		

V. Examination of the Results obtained during the Investigation

INCIDENCE OF BENT OR STRAIGHT APPENDICES

75 per cent. of the affected appendices were markedly bent, thus confirming Aschoff's finding that bent appendices are more prone to disease than straight ones. A suggested reason for this will be given later in this paper.

INCIDENCE OF EMPTY AND FAECES-CONTAINING APPENDICES

Aschoff's opinion that diseased appendices are more often empty than full is not upheld, as 75 per cent. of the specimens contained either faecaliths or fluid faecal matter. This finding will be referred to again in the discussion as to the causation of acute appendicitis.

PROPORTION OF CASES ASSOCIATED WITH SORE THROATS

35 per cent. of the patients complained of a sore throat. Of these, 10 per cent. suffered from chronic sore throat, and in each case the appendix was found to be chronically inflamed. In the remaining cases the patients had never had a similar abdominal pain before, and no trace of chronic inflammation could be found in 90 per cent. of the appendices. These figures are suggestive, if nothing more, and it would appear that there is an association between at least *some* cases of appendicitis and a slightly preceding sore throat. The series of cases examined was not, however, large enough for any definite results to be obtained.

AVERAGE TIME TAKEN FOR VARIOUS STAGES OF INFLAMMATION TO DEVELOP

Some authors are of the opinion that a definite time-table for the various stages can be made out, but this is certainly not true of the present series of cases. The average time for gangrene to develop is only four hours longer than the average for catarrhal inflammation in a total of twenty-six hours. Perforation, however, seems to be definitely later than the onset of gangrene (forty-two hours as compared with twenty-six hours). The only inference which can be drawn from this part of the investigation is that where a patient has had a characteristic pain for two days, and has a pulse and temperature of 100 or over, the appendix is probably perforated.

PROPORTION OF CASES SHOWING VARIOUS SIGNS AND SYMPTOMS

A point of interest under this heading is the low proportion—40 per cent.—of cases with constipation, which is usually regarded as being one of the most characteristic symptoms. Vomiting—90 per cent.—was a much more reliable symptom.

In 75 per cent. of the cases pain began at the umbilicus, shifting later to the right iliac fossa. This figure corresponds exactly to the incidence of faecaliths (75 per cent.). Of the cases with faecaliths 90 per cent. showed pain commencing at the umbilicus, which would indicate that the initial distension of the appendix is greater in these cases than in those where only a stricture is present. The pain due to the distension is referred to the midline at the umbilicus, because the gut is developed in the midline (Arey).

Two cases showed the interesting condition of pain commencing at the right iliac fossa and later becoming localized around the umbilicus. There is no explanation of this phenomenon in the literature, but both appendices revealed the presence of chronic strictures, and it may be that the localized pain in the right iliac fossa was due to the organ being unduly near the peritoneum of the abdominal wall, so that it was infected early. Simultaneously the stricture was in effect becoming thicker from the mucosal hypertrophy which always occurs and was damming back any faeces in the distal part of the appendix. This would produce distension and consequently pain around the umbilicus.

One case showed diarrhoea instead of constipation. No aperient had been taken.

RANGE OF TEMPERATURE AND PULSE IN THE SERIES

It was confirmed that in acute appendicitis it is unusual for the temperature to rise above 100°, with a corresponding increase in the pulse rate.

INCIDENCE OF FAECALITHS AND STRICTURES

Perhaps the most important point on which the results of this investigation differ from those of Aschoff is in the incidence and importance of faecaliths. Aschoff holds that their presence is purely fortuitous, and that the inflammation begins not around them but distal to them. In the present series faecaliths were found in 70 per cent. of cases and strictures in 45 per cent.; only 15 per cent. were free from either. In 20 per cent. both faecaliths and strictures were seen. Aschoff's finding that the tissue around the faecalith was not affected was not upheld in a single case. In every instance there were inflammatory changes of various degrees in the tissue around the faecaliths. In some cases there was chronic thickening, in others catarrhal inflammation, while two appendices were actually perforated around the faecalith. Of their importance, then, there can be no doubt, and the suggestion is made that they play an important part in the causation of acute appendicitis, upon the following lines.

For some as yet unknown reason faecal matter in the appendix becomes inspissated to form a hard structure —the faecalith. It is possible that its formation is linked up with the reduction in the cellulose intake, suggested by Mr. Rendle Short to be the cause of the present widespread nature of appendicitis. I would suggest: (*a*) That if the caecal contents are bulky from the presence of much cellulose the faeces will not enter the appendix as easily as do the very fluid faeces associated with a low cellulose diet. Hence faecaliths would only be able to form in a smaller number of appendices, because a larger proportion would not contain any faeces for them to form from. (*b*) That once the faeces had entered the appendix the mucosal glands absorbed water from them. Now in the case of the faeces from a low cellulose diet the result is a hard mass which easily becomes impacted, but where the faeces contain a high proportion of cellulose the result is a softish fibrous mass which can easily be extruded and which would never evoke the inflammatory response which the hard faecalith calls forth.

Once the faecalith has been formed it is regarded by the appendix as a foreign body, so that an inflammatory

response is set up around it. If the faecalith is not very big the peristalsis of the appendicular musculature may be sufficient to force it into the caecum, after which it passes along the intestine. But the inflammatory response it has evoked results in the formation of fibrous tissue—a stricture—and it is noteworthy that all the strictures in the present series were found in the same situation as the faecaliths—that is, at the proximal end of the organ. If, however, the faecalith is too large to be forced into the intestine the inflammatory response will be all the greater. The resulting oedema and compression of the surrounding tissue will compress first the lymphatics and veins, and later perhaps the arteries which drain and supply the apex of the organ. Hence it is usually at the apex that the mucosa is first seriously damaged. Once this has happened the small Gram-positive cocci and rods which Aschoff consistently found can make their way through the mucosa, become increasingly virulent, and set up an acute inflammation.

BIBLIOGRAPHY

Arey, L. B. (1934). *Textbook of Embryology*, Philadelphia.
Aschoff, L. (1932). *Appendicitis*, London.
Short, A. Rendle (1920). *Brit. J. Surg.*, **8**, 171.

MASSIVE SPONTANEOUS INTRAPERITONEAL HAEMORRHAGE

BY

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Massive intraperitoneal haemorrhage in the male in the absence of trauma or gross visceral disease is rare, and for that reason it was thought justifiable to put a further case on record.

Case Record

The patient, a man aged 52, was admitted to hospital with a history of a sudden onset of acute abdominal pain six hours previously. This had come on in the umbilical and hypogastric regions and doubled him up. It had since radiated to the right side of the chest and occasionally into the shoulders. On deep breathing the pain in the right hypochondrium was worse. He had had a similar attack of pain a week before. This was confined to the umbilical region and had not radiated. It had lasted three days. During the whole of the week he had had two or three attacks almost daily, the pain lasting a few seconds. There was no history of indigestion and none of trauma, but some frequency and scalding of micturition was experienced. He had been on a diet for high blood pressure and had lost 2 stone.

The previous medical history—for which I am indebted to Dr. R. O. Knowles of Birkenhead, who sent the patient into hospital—showed him to have suffered from bronchitis for some time. He had had two attacks of pulmonary oedema, as well as anginal attacks, and his systolic blood pressure was 200 mm. Hg. For this he had been dieted. He was a fairly well built man, with an emphysematous chest. On admission he was pale and somewhat cyanosed, cold, and clammy. His pulse was 96 and temperature 96.8°. The whole of the abdomen was very tender, this being especially marked in the upper abdomen and right subcostal regions. There was no real rigidity and no loss of liver dullness. In view of the shocked condition of the patient, his history and build, it was thought that he might have an acute pancreatitis. However, though there was doubt as to the actual causative lesion, there was no doubt that the case was one of acute abdominal emergency and should be explored. The pulse had remained stationary during the period before operation, being 98, and his temperature had risen to 98°.

Under spinal anaesthesia the abdomen was opened through a mid-line supra-umbilical incision and was found to be full of blood. A considerable amount was evacuated, and the hand was passed to the splenic region as it was thought that there might have been a spontaneous rupture of the spleen. Unfortunately, the spleen could not be brought into the wound, but nothing abnormal was felt on palpation. The liver, gall-bladder, pancreas, stomach, and intestines were normal; there was no retroperitoneal mass of clot, which was looked for as it was thought that the bleeding might have come from an aortic abdominal aneurysm. The kidneys felt normal, and there was no retroperitoneal collection of blood in the vicinity. The pelvis was also normal. There was staining of the mesentery and omentum, but no intramesenteric clots were present. As gross aneurysm appeared to have been excluded attention was again given to the spleen, as the quantity of blood there seemed to be greater than elsewhere. An attempt was made to bring it into the wound for inspection; this was successful, but in doing so an adhesion was torn. The spleen appeared to be normal; there was a tear in the capsule about one inch by one-quarter inch, certainly due to operative trauma. There was no pulping of the spleen or any subcapsular haematoma. As the patient's pulse was becoming very weak the abdomen was closed, a drain being left in. His pulse improved in volume once the abdomen was closed. A transfusion of one pint of citrated blood was given an hour later. After the operation his pulse rose to 150 and his temperature to 102°, and in the first twenty-four hours the pulse dropped to between 120 and 136, the temperature at the same time varying between 99.4° and 101.6°. However, thirty-six hours after the operation his pulse became weaker; he became much more dyspnoeic; and he started vomiting, dying soon after.

PATHOLOGICAL REPORT

At the necropsy the abdomen still contained a considerable amount of blood, with what appeared to be a greater proportion in the splenic region and an accumulation in an inguinal hernia sac. The stomach was dilated, and the small intestine also slightly distended but otherwise normal, as also were the liver, pancreas, kidneys, suprarenals, and bladder. The spleen was normal in size and texture. There were two tears in the capsule, each about one inch by one-quarter inch, which were considered by me to have been secondary to the operative trauma. The abdominal aorta showed only very slight evidence of atheroma. The heart was enlarged. The orifices of the coronary arteries were normal. There was slight blood-staining on the upper surface of the diaphragm. Though the individual splanchnic vessels were not carefully dissected the absence of any retroperitoneal clot was thought to exclude any aneurysmal rupture.

Comments

Cases of massive intraperitoneal haemorrhage in the male in the absence of trauma or gross visceral disease appear to fall into two main groups:

1. An older group of patients, aged 44 to 60, with marked arteriosclerosis and high blood pressure, the haemorrhage coming from a ruptured splanchnic vessel. Six cases have been described which can be included in this category; of these, two were not operated on, being discovered post mortem (Moorehead and McLester, 1936), and four were operated on. In three of the latter (Starcke, 1923; Green and Powers, 1931; Buchbinder and Greene, 1935) the bleeding point was found, the vessel was ligatured, and the patients recovered. In the fourth case (Hilliard, 1918), where no bleeding point was discovered, the patient died.

2. A younger group, in which the vascular system is healthy and no bleeding point is found. Three cases come under this heading: Churchman, 1911—a man aged 48; Hartley and McKechnie, 1934—a man aged 31; and Bruce, 1937—a man aged 34. Of these only the last-named survived laparotomy.