Observations ON MORTALITY IN ACUTE APPENDICULAR DISEASE*

BY

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I make no apology for taking up a subject which has been dealt with repeatedly in recent years. We are faced with the fact that despite improved education, the greater appreciation by the public of the dangers of the disease, the extended hospital facilities, the vastly improved transport service, and the increased number of capable operating surgeons, the death rate in acute appendicular disease is as high to-day as it was twenty years ago. There can be no doubt that early operation is now practised the country over to a degree greatly exceeding that of two decades ago. How are we, then, to reconcile our advocacy of early operation with this persistently high mortality? To my mind the explanation is clear, and permits of no doubt—namely, that the majority of the patients now operated on early are of the class which did not contribute to the mortality, while in the dangerous and fatal type of case operation occurs little, if at all, earlier than it occurred twenty years ago.

Why is it that this type escapes early diagnosis and thus early operation? It is here that I join issue with the teachers of pathology and of surgery. The fundamental facts of the pathology of the acute diseases of the appendix are not presented to-day, either in the textbooks or, so far as I can gather, in lectures, with greater clearness than they were before the early changes in these diseases had been described. By attempting to group all cases as examples of "appendicitis"—that is, an inflammation of the appendix—it is necessary for the teacher to qualify every statement, both in regard to pathological changes and clinical symptoms, in such a way that the subject is shrouded in difficulties and uncertainties, which make for hesitation and delay in diagnosis.

At the Annual Meeting of the Association in 1914 I brought forward clinical and experimental evidence in support of a plea for the recognition of two primary acute diseases of the appendix: (1) acute inflammation of its wall—appendicitis; and (2) acute obstruction of its lumen—a closed-loop intestinal obstruction.† These two diseases differ essentially in their pathology, their clinical symptoms, and, above all, in their danger to life. The profession, and particularly the teachers in the profession, have been slow to appreciate this fundamental distinction, which is, I consider, a primary requisite for correct and early diagnosis.

THE RESPONSIBILITY OF DIAGNOSIS

This falls mainly on the general practitioner, and for him it is all-important to have a clear picture of the early pathology of acute affections of the appendix.

Appendicitis

Inflammation of the wall of the appendix is of common occurrence, and may be the sequel to a blood-borne infection—as when it follows a tonsillitis—or to an intestinal catarrh. It is characterized by a turgid swelling of the wall of the appendix, frequently some ulceration of its lining mucosa, and, in some cases, by a plastic and

occasionally suppurative local peritonitis. Clinically its onset is, relatively speaking, a gradual one, characterized by nausea, pain, and constitutional disturbances in the shape of fever, accelerated pulse rate, dirty tongue, and usually constipation, but occasionally diarrhoea. It is seldom a fatal disease unless complicated by an obstructive factor, by the sudden perforation of an ulcer due to a concretion, by a secondary intestinal obstruction, which is rare, or by an ill-timed operation.

Acute Appendicular Obstruction

This is a very fatal form of intestinal obstruction, because it is of the closed-loop variety which tends to early gangrene (Fig. 1). If we would but realize that

FIG. 1.—Obstructed appendix full of faecal matter; tension gangrene.

a sudden and complete obstruction of the lumen of an appendix, containing faecal matter, will, from the pressure of decomposing content, inevitably lead to tension gangrene within from six to twenty-four hours, and that gangrene will be followed by perforation, with the escape of stinking faecal content into the free and unprepared peritoneal cavity, we would regard the recognition of the onset of such an obstruction as one of the most important and responsible duties of a medical practitioner.

Is there, then, a clinical picture typical of this disease distinct and differing from that of appendicitis? Most assuredly there is. Here is the actual history of a case.

A boy, aged 14, who had previously enjoyed good health, except for occasional colicky pains in the abdomen, went to bed at night, apparently in his usual health. At 2 a.m. he awoke and cried out, complaining of severe colic, and vomited. He indicated to his mother that the pain was worst just above the umbilicus, but spread right across the abdomen. A hot cloth was applied to the abdomen, and in twenty minutes the pain had practically disappeared and the boy fell asleep. At 4.30 a.m. he was again awakened by a return of the severe colic, and again vomited. Heat was again applied, and the doctor was called. When seen at 7 a.m. by the doctor the boy volunteered that the pain had almost gone. *His pulse and temperature were normal*, but his face looked slightly drawn, and there was a slight rigidity of the right rectus muscle in its lower part, and a little tenderness.

The doctor advised a continuance of the hot applications, nothing but water by the mouth, and promised to return two hours later. At 8.30 a.m., and shortly before the doctor returned, the boy had another attack of colic and vomited. When examined at 9 a.m. he was again comfortable; his pulse and temperature were still normal, but his facial expression was slightly anxious, and the rigidity and tenderness in the right lower quadrant of the abdomen were still present, although not pronounced. Removal to hospital was recommended, and the boy was admitted with the doctor's card bearing the diagnosis "acute appendicular obstruction."

When seen in hospital at 10.30 a.m. his condition had not altered, except that his pulse rate was now 90; his temperature was still normal. The abdomen was opened at 11 a.m. A large appendix, tensely distended and stippled with greenish points of commencing gangrene, was carefully delivered through the wound and removed without bursting. The abdomen was closed without drainage. The proximal end of the appendix, for half an inch, appeared normal. Just beyond this, a concretion could be felt impacted. On opening the appendix about two drachms of stinking fluid faecal matter escaped from the part distal to the impacted concretion, and the mucosa of this part was dark green in colour and gangrenous.

* A British Medical Association Lecture (abridged) delivered to the Darlington Division on November 20th, 1930.

† I have since learned that Van Zwahlenberg of Riverside, California, and Campbell of Belfast had come to similar conclusions.

In this case, owing to an intelligent appreciation of the pathology of the condition, the medical man hurried the boy to hospital and thereby saved his life. Had he temporized and waited for the constitutional signs of inflammation, the appendix would have burst, spilling fluid faecal matter into the free peritoneal cavity and determining a fatal peritonitis (Fig. 2).

FIG. 2.—Rupture of obstructed appendix into the free peritoneal cavity.

Appendicitis is a misnomer for this condition; it is a form of intestinal obstruction, and gives rise to the signs and symptoms to be expected when a coil of bowel or a cul-de-sac, such as the appendix, containing faecal matter, becomes suddenly obstructed. The case cited above exemplifies the condition in its purest form. There are, however, cases in which, in the course of a true appendicitis, the obstructive factor supervenes and adds the new danger of tension within the lumen to inflammation of the wall. In such cases the obstructive factor may determine gangrene and perforation, but with less danger to life than in the primary obstruction, for the obvious reason that the primary inflammation has called forth a plastic peritonitis, and, when finally perforation does occur, it is into an area of the peritoneum more or less walled off by adhesions of omentum and intestinal coils.

If we analyse the mortality in the light of the pathology above described, we find that well over 90 per cent. of the deaths occur in the obstructed cases, usually designated as cases of "gangrenous perforated appendix." It is certainly true that in many cases the medical man is not called in until the gangrene and perforation have occurred, the trouble having been regarded as intestinal colic, and treated by what turned out to be nothing less than a lethal dose of castor oil. It is therefore necessary to teach the public the danger signals, and particularly the significance of recurring colic. But how can we expect the public to learn a lesson which is not yet taught in most medical schools?

In reading over the papers published during the past few years on the mortality from "acute appendicitis" I find no mention of appendicular obstruction or its significance, from either the pathological or the clinical standpoint.

THE RESPONSIBILITY OF THE OPERATING SURGEON

While the mortality in acute appendicular disease is attributable, for the most part, to delay in diagnosing and dealing with the obstructive type of case, the responsibility does not end with diagnosis. There are certain principles which should guide our operative practice in regard to:

1. When to operate and when not to operate.
2. How to avoid doing harm while doing good.
3. Management of complications.

When to Operate

All are agreed that when a diagnosis of acute disease of the appendix has been made within forty-eight hours of the onset of symptoms, immediate operation is advisable; in the obstructive case it is imperative. Where the diagnosis is made between the second and fourth days after onset, where signs of a spreading peritonitis are present, and where perforation has in all probability occurred, operation is surely indicated. Where the patient is seen four or five days after the onset, however, and has a palpable lump, but relatively little constitutional disturbance, immediate operation is unquestionably meddlesome. Nature has coped with the initial dangers, and resolution is proceeding quietly. If the surgeon blunders in, breaks down organizing adhesions and causes haemorrhage, obstructive intestinal complications not infrequently supervene, and a patient who was recovering falls a victim to ill-timed surgery. Honesty in reflection will bring to the minds of most surgeons cases of this order which they would willingly forget.

Those who state that there is no stage at which one should not operate may be skilful mechanics; they are not surgical pathologists. Similarly, in the type of case seen seven or eight days after onset, where a diffuse peritonitis has occurred, and where the abdomen is distended and tympanitic but the pulse rate is slow, we can recognize a more or less successful reaction to a widespread peritoneal infection. The original focus now plays a minor part. In such cases there may be a large pelvic abscess which demands evacuation by as direct a route as can be found. Gross disturbance of intestinal coils, in order to discover the now buried and innocuous appendix, is surely not only meddlesome but entirely unwarranted.

In both the types of case just mentioned, conservative treatment, under careful observation, on the lines laid down by Ochsner, while tedious, is undoubtedly logical and sound in principle. McNeill Love and Hamilton Bailey in this country have recently emphasized these points. In America a greater discrimination and more thoughtful study of these cases have clarified the issue and confirmed the view that there is a time not to operate as well as a time when it is reprehensible to delay operation.

Minimizing the Harmful Effects of Operation

A certain degree of trauma and exposure of fresh surfaces to infection is inevitable in any operation for a deep-seated infective focus. A small incision is not the means by which to avoid trauma. The first criterion must ever be an adequate exposure of the appendix. This is especially important in the obstructive type of case where the distended and gangrenous organ may burst unless freed and brought to the surface in full vision of the operator. Even should twenty minutes be taken to free and deliver such an appendix unruptured, the time will have been well spent. Care, and not speed, is the essential in such cases.

Where a walled-off abscess is found in the right iliac region or pelvis, the appendix should be removed only if it is accessible without gross disturbance of adhesions. A patient will rarely die when an abscess has been drained; he may readily be precipitated into a dangerous condition if a determined operator feels it incumbent on him to remove the appendix regardless of Nature's barriers.

The peritoneum has a wonderful capacity for dealing with infection; not so the abdominal wall. For many years I was struck by the fact that after operations for a perforated appendix with peritonitis, where the peritoneum had been drained, the patients were seriously ill for several days, during which little or nothing came

from the drain. About the fourth or fifth day the general condition of the patients improved, and some foul discharge came away alongside, and not through, the drain; came, in fact, from the abdominal wall. In such cases the edges of the wound felt indurated and were tender, and, if stitches were taken out, foul sloughy material escaped. Anaerobic infection had been locked up in the abdominal wall, and had led to necrosis of fat, aponeurosis, and muscle. It was this that had accounted for the pronounced post-operative constitutional symptoms. Surely it must be that this added abdominal wall infection in some cases turns the scale between recovery and death.

Since adopting the plan of leaving the abdominal wound open, packing with gauze, in which is incorporated a small Carrel tube (Fig. 3), through which small quantities

FIG. 3.—Open treatment of abdominal wound. Carrel tube seen on section in deeper layers of gauze packing.

of hydrogen peroxide are injected every few hours, I have found that sloughing is obviated, constitutional symptoms are absent, and healing is not delayed. Post-operative hernia is also less common, as no muscle or aponeurosis is lost by necrosis. The avoidance of all catgut sutures, and the use of the silkworm-gut looped suture for peritoneum and muscle, leaves no dead organic material in the wound as pabulum for bacteria (Fig. 4).

FIG. 4.—Method of using "looped sutures" of silkworm-gut in place of catgut for closure of deeper layers of wound.

In stout subjects the open treatment of the abdominal wound is especially necessary, as anaerobic infection is tolerated badly.

Post-operative Intestinal Obstruction

This accounts for a number of deaths in patients who have successfully dealt with the peritoneal infection. Where the obstruction is organic in nature and due to a binding down of lower ileal coils to the appendix bed, the patient will complain of some peristaltic pains associated with his distension and vomiting. In such cases, following gastric lavage, a tube enterostomy, or an ileo-colostomy, must be undertaken without delay, and the chloride loss from vomiting must be made good by subcutaneous saline infusions. Where, with distension, there is no clinical evidence of ineffective peristalsis, and paresis

of the gut exists, continuous gastric drainage through a duodenal tube, the intravenous injection of 30 c.cm. of 5 per cent. sodium chloride solution, to replace loss of chlorides and stimulate peristaltic contraction, the application of heat to the abdomen, and the use of pituitrin, may excite peristalsis. Enterostomy has a limited value in this type, but permits of feeding and the replacement of loss of fluid and salt.

The Value of Caecostomy

In a strictly limited number of cases, where at operation a grossly distended caecum is found, a valvular tube caecostomy is unquestionably indicated. The comfort which this gives, the ready supply of fluid, and the absence of flatulent distension, make this a valuable addition to an appendix operation in selected cases.

CONCLUSIONS

1. The mortality from "appendicitis" remains as high as it was twenty years ago, because, while facilities for treatment have improved, diagnosis in the fatal type of case has not.
2. There is now very strong evidence that the fatal cases of acute appendicular disease are primarily cases of obstruction and not inflammation of the appendix. It is necessary to distinguish between these two types of acute disease, the clinical symptoms and essential pathology of which differ widely. The obstructive cases must be regarded and treated as belonging to the same category of urgency as internal strangulation of the intestine.
3. Free exposure at operation is essential in the obstructive cases.
4. While immediate operation is expedient in the early stages of appendicitis, delay is advisable in the resolving case, the danger of operation being greater than that of expectancy.
5. Where a large localized abscess is present, simple drainage with minimal disturbance is the safest line of treatment.
6. In cases with perforation and peritonitis, infection of the abdominal wound may be an important factor in determining a fatal issue. Open treatment of the wound is therefore advisable.
7. Post-operative obstruction accounts for a number of deaths. A timely enterostomy or ileo-colostomy may prove a life-saving measure.

THE FAMILIAL INFLUENCE IN GASTRIC FUNCTION

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In the course of some recent investigations into the question of heredity in gastric cancer we had occasion to examine, by means of the fractional test meal, the chemical and motor functions of the stomach in several members of each of a number of families. During this investigation we were struck by the frequency with which members of the same family gave closely similar results. This has been commented upon by several writers, notably Hurst,¹ who has on several occasions observed that duodenal ulcer with its accompanying "type" of stomach tends to run in families. The same has been noted with regard to achlorhydria and, to a less extent, "gastric diathesis." In 1923 one of us,² showed two instances of the close resemblance in the test-meal charts of parent and child. Since then we have collected two