REMARKS UPON SOME CASES OF APPENDICITIS AND OTHER FORMS OF PERITONITIS.

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During the past year I have had the opportunity, by the kindness of Mr. Bowlby, of observing the progress of many cases of appendicitis and the like in his wards at St. Bartholomew’s Hospital. From my notes on these I propose to choose instances which illustrate important points in these affections.

It was in my student days that Fitz, of Boston, gave, in 1886, the first precise account of a disease which he called appendicitis. It is true that Addison, in 1836, had drawn special attention to the diseased condition of the appendix found in cases of inflammation in its immediate neighbourhood, a condition to which Dupuytren gave the name of a “phlegmon of the right iliac fossa.” But the power of the peritoneum to rapidly isolate inflammatory effusion was not then understood, and abscesses localised in that region were supposed to occupy the connective tissue. I confess I have no recollection of Fitz’s monograph which first put the disease on its proper footing; but I remember a case labelled by the new name, as I do others of its forerunner, typhlitis, and also of peritonitis pure and simple, more common then than now. Still, I doubt if these latter were as frequent as is now often stated to have been the case, or if errors of diagnosis by our predecessors account altogether for the apparent increase in appendicitis to-day. Indeed, it is a vexed question whether this greater prevalence is real or not. Sir Frederick Treves, in his Cavendish Lecture on the subject says: “Nor is there any evidence to support the suggestion that it (appendicitis) has undergone any remarkable recrudescence, or that it has become more frequent in its appearance. It passed unrecognised in earlier times as gastric attack or gastric seizure, perityphlitis, cramp of the bowels, inflammation of the intestines, iliac phlegmon, and the like. It was the fons et origo of many forms of peritonitis."

On the other hand, I have heard well-known surgeons say that they have little doubt that, of late years, there has been a real increase in the prevalence of the disease, though they cannot account for it. Less responsible writers from time to time announce causes varying from cigarette smoking to the con-
sumption of refrigerated meat, from the effects of influenza to the use of badly enamelled cooking pots. Here, again, the hurry of modern life, bolted food, and constipation, are made to answer for it. But is the rush of existence so markedly greater than it was a dozen or fifteen years ago? Or is the athletic damsel of to-day more prone to constipation than was her sedentary grandmother in the "forties"?

It is a truism that the type of a disease varies from time to time, possibly owing to modifications in bacteria and their products, as well as to the environment of their hosts. When one considers what is the most constant factor in appendicitis and other peritoneal infections, is it not the presence of the *Bacillus coli communis*? In the Hunterian Lectures of last year, Mr. Eccles analysed the results of the bacteriological investigations of many authorities as to the pathogenic organisms present, in such cases, in the lumen of the appendix, its walls and its surroundings. He finds that in 94 per cent. of cases it is the colon bacillus. Far behind follows that of tubercle in 2 per cent.; whilst streptococcus and staphyloccocus are bracketed third in frequency, with a percentage of 1·5 each. May not the virulence of the former organism be enhanced nowadays by conditions of which we are ignorant; and is it unreasonable to look in this direction for some explanation of the apparent increase of this disease?

As appendicitis is now to the fore, so the mere existence of what was called typhlitis has been denied. That perforation of the caecal wall and perityphlitis not primarily due to the appendix still account for a small percentage of cases of peritonitis in the right iliac fossa has been shown by Dr. Russell, of Edinburgh. From what he describes as a comparatively small experience he gives four instances. Three were of acute perityphlitis due to primary lesion in the caecum. Of these, one case on operation showed marked pericæcal inflammation, while the appendix showed none; at the autopsy a few days later faecal ulcers were found in the caecum. The perforation of a similar ulcer was the cause of death in a second. In a third fatal case there was no perforation or ulcer. In none of these was the appendix diseased. In the fourth a burrowing perityphlitis abscess was found, in which the appendix was not involved. Whilst considering that a margin should be left for such cases, he is of opinion that in more than 95 per cent. of acute affections at the caecum the primary seat of trouble is the

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appendix, and that were it not for this vulnerable relic perityphlitis would be of rare occurrence.

Again, has not the appendix been credited nowadays with a little more than its due share in the causation of that form of peritonitis which was vaguely termed idiopathic? Doubtless in the vast majority of cases it originates in the appendix, the stomach, or the tubes. Still, from time to time instances occur in which the viscera appear healthy, and there is nothing to show from what source the invading organisms came. The term "primary peritonitis," qualified by the name of the germ if possible, seems here to be justifiable. Professor Osler\footnote{\textit{Medicine}, p 597.} mentions 102 cases of acute peritonitis which proved fatal at Baltimore. In twelve of these the infection was confined to the peritoneum itself. Most were mono-infections, streptococci being present in five cases.

I have used the term "peritonitis" instead of the more accurate one "peritoneal infection," though some have regarded the former as a cloak for ignorance. But it must be clearly borne in mind that peritonitis is an infection of a large serous sac, and that it is not necessarily an inflammation, in the ordinary sense of the term, accompanied by its cardinal symptom, fever. Indeed, an acute infection of this membrane is usually associated with a normal, or subnormal, temperature and a small, rapid pulse. These symptoms indicate the absorption of toxins, for the treatment of which surgery alone—and that not long delayed—is of any avail. Some of the following cases will show this clearly.

A woman, aged 42, was admitted on March 22nd, 1904, with dyspepsia. In 1894 she first suffered from it, and a gastric ulcer was diagnosed. Ever since she has had pain and vomiting after food. On March 19th last she had pain in the epigastrium; next day she was taken suddenly worse in the afternoon and vomited in the night. On the 22nd she vomited again and was brought to hospital. Her temperature was then 100°4, pulse 92, and respirations 32. The lower half of the abdomen was rigid, slightly distended, and tender. The liver dulness was normal in extent. Her bowels had not acted since the 21st and no flatus had passed. The next day the distension was increasing and enemata had no effect. She had not vomited again, her temperature was not raised, and pulse was 100 and fairly strong. On the 24th, there being no improvement in condition, laparotomy was performed. The first incision was made from the ensiform cartilage. No free gas was found on opening the
peritoneum, nor was there flatus in the stomach. No adhesion or thickening of that viscus was felt. The second opening was made above Poupart's ligament and the appendix explored. It did not appear diseased, but some thin pus was found in the peritoneal cavity. A third incision was made in the middle line downwards from the umbilicus. The uterus, broad ligaments and ovaries were explored, nothing abnormal being detected, but more pus was found in the pelvis. The lower part of this incision was left open and the wound drained. After the operation the pulse was 100, but she continued to vomit for two days, and enemata had no effect. She was then given calomel in full doses, and later castor oil. The bowels then acted, and the sickness and distension disappeared. She made an uninterrupted recovery and was discharged five weeks from the date of admission.

A staphylococcus was grown from a specimen of the pus, confirming the opinion formed at the operation from the character of the pus, that it was not due to the *Bacillus coli*; in which connection it is worthy of note that the smell of pus is no criterion of its toxic qualities, as witness the inodorous result of the virulent streptococcus and the stinking pus of the colon bacillus.

The past history of this patient would have led one at first to think that the peritonitis was due to the perforation of a gastric ulcer. One point against this was that the area of liver dulness was not lessened, which was confirmed by no gas being found free in the peritoneal cavity. As the distension and constipation persisted it was considered best to operate whilst her pulse was still good. It will be noticed that the constipation and distension persisted after the operation. Had this been left unrelieved the result would have been disastrous; nothing is more important in the after-treatment of such cases than the regular evacuation of the bowels. This holds good not only in acute cases like this, where the intestines are paralysed as a result of peritonitis, but also in the ordinary cases of appendicitis operated on in an interval of quiescence; or in those cases where the gut, having been freed from some adhesion, is very apt not to recover its tone for a time. The regular use of enemata seems the best method of attaining this result; in suitable cases I have also seen good results from the administration of strychnine.

Following on this case of primary peritonitis due to staphylococcus, comes one where the pneumococcus is the agent of evil. In the same ward, in the previous year, a woman, aged 27, was admitted on February 26th, complaining of headache and general pains. Her temperature was 104.2°, and she had a rigor. On the
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following day signs of general peritonitis followed. A large polymuclear leucocytosis was present. On operation on March 2nd the appendix was found to be constricted at its base, dilated, and full of mucus above. It was ligatured and removed. There was some thin, turbid, dark-coloured fluid in the peritoneal cavity, but no adhesions were found, nor was there any evidence of the perforation of a viscus. A culture of the fluid showed the presence of pneumococcus. Becoming worse, antipneumococcal serum was injected. Later on the pleural cavity was infected, but after resection of a rib and the evacuation of twelve ounces of sour-smelling pus, the further progress of the case was uneventful.

This case was discussed at a meeting of the Clinical Society last January. Professor Marsh was unable to state whether the case was one of primary appendicitis, but he considered that pneumococcal peritonitis must be added to the list of conditions which imitate appendicitis.

As to the connection between this form of peritonitis and lobar pneumonia, it was stated that of 182 fatal cases of the latter disease at Guy's Hospital in the last five years, in which necropsies had been made, only five showed any evidence of infection of the peritoneum, and that was slight. At the German Surgical Congress of 1903, Dr. von Brunn read a paper on this variety of peritonitis. In his experience the symptoms were comparatively mild; in the early stages it was liable to be mistaken for appendicitis, a point of difference being that in peritonitis there was more often diarrhoea.

From the pneumococcus and the staphylococcus we come to the tubercle bacillus. This may give rise to conditions in the abdomen of which, though they have existed for a long period, the symptoms may exactly resemble the sudden onset of a typical attack of acute appendicitis.

J. R., aged 27, was admitted on November 25th, 1903, with the following history. After being in his usual health, and having suffered from nothing of the kind before, he was seized on the night of November 11th with sudden pain in the abdomen. He was in bed at the time. The pain was over the whole of the abdomen and continued for two hours, when he was violently sick. Next day he went to work. He took nothing but milk, and in a few days the pain gradually disappeared. During most of this time his bowels had not been open, though before coming to hospital they yielded to castor oil and enemata. On admission his temperature was 99° and pulse 64. There was slight pain and tenderness in the abdomen, a small hard mass being felt in the right iliac fossa. He
was kept in bed for a fortnight, during which time there was no return of symptoms, and on December 11th laparotomy was performed. The appendix was not found, but the hard mass was found to consist of a caseating mesenteric gland, which was removed. The sutures were taken out on the tenth day and he was discharged on January 5th.

Here you have several symptoms which are very characteristic of the ordinary acute appendicitis. The onset was quite sudden. It occurred at night, which is very often the case. Indeed, in children a history of recurrent attacks of stomach-ache when in bed, some hours after food, should always lead one to think of the appendix as a possible source. Again, it should have been stated that he had been for a long walk that afternoon—his occupation not being an active one. It seems almost as common for an attack to be preceded by some unwonted exertion as it is by an error in diet. And yet, with all this, the condition found on operation, allowing for the period of rest, could not well have been more chronic.

The following is another case illustrating the way in which tubercular peritonitis may simulate appendicitis, and what an advanced condition it may reach with very few symptoms of its widespread nature.

G. T., aged 5, admitted April 28th, 1904, with the history that last October she was suddenly seized with an attack of acute pain in the right iliac fossa, accompanied by frequent vomiting. She had similar attacks at intervals for the next two months; then she apparently completely recovered, except that constipation persisted. Ten weeks ago she had measles, followed in a month by another attack of vomiting and pain in the same region; lastly, a fortnight ago, yet another, though slight, attack. She was a delicate-looking child, though in good condition; her temperature and pulse were normal; she complained of pain in the right iliac fossa, where there was some resistance on palpation, but no swelling. After being kept quiet for three weeks, during which time there was no return of symptoms and her pulse and temperature were normal, the abdomen was opened on May 20th. The intestines were found to be densely matted together, and the cecum to be universally adherent. The appendix was not searched for. Further operation being hopeless, the abdomen was closed. She progressed uneventfully and was discharged on June 7th.

In spite of evidence of such extensive disease the symptoms had been those of typical attacks of recurrent appendicitis, in the intervals of which the child was in fairly good health and com-
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plained of nothing but constipation. The tubercle may have invaded the peritoneum from the mesenteric glands, the caecum, or the appendix. The enormous mass of adhesions found here is very characteristic of tuberculous peritonitis; in some cases they are found to bind all the abdominal organs together as if they had been fixed by plaster of Paris.

Attention has been drawn to the frequency of attacks of appendicitis in women at the menstrual period, this organ sharing in the general congestion. Here is a case where the disturbance occasioned by that process simulated very grave disease, the symptoms ending with the onset of the flow.

A young woman, aged 18, came in on the evening of April 8th, complaining of great abdominal pain, which came on suddenly in the forenoon of April 5th, accompanied by vomiting. For the past two years she had had attacks of pain and vomiting at intervals, and last summer brought up a pint of dark blood. On this occasion there was no hæmatemesis. There was great pain in the abdomen and imperfect movement, also tenderness in the epigastric region. Her pulse was 132, temperature 102.4°. A blood count gave 17,200 leucocytes. Widal's test was negative. Next morning her temperature fell to 98.2° and pulse to 96, pain was much less, and menstruation was reported to have set in the previous night. The following day all pain had disappeared, and she was discharged quite well on the 14th.

The symptoms, coupled with the past history of hæmatemesis, pointed very much to perforation from a gastric ulcer, possibly leaking into a shut-off cavity. But the often useful rule that a rise of both temperature and pulse is not so likely to mean perforation as a fall of temperature with a rapid pulse held good in this instance. It should have been said that on admission friction under the left breast was said to have been heard by one observer. Had pleurisy been present and spread to the diaphragm the symptoms would very likely have been referred to the abdomen, but they would not have passed off so rapidly as the above did.

We now come to the case of a young woman where nothing was found to account for her symptoms. A young widow, aged 25, with one child, has had for the past year pain in the epigastric and right hypochondriac regions, extending sometimes to the lumbar area. At times it comes on acutely and interferes with her duties. It is much worse after a hard day's work, or if she catches cold. She has no pain when lying down. She has also had pain down the right leg. Once or twice she has been unable to micturate all day,
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and on the following day has passed a large amount of water. Three weeks ago she had a very severe attack of pain and was recommended by her doctor to come to hospital, which she did on November 28th. His impression was that the trouble was caused by a floating kidney.

She is a particularly strong, healthy-looking, country woman. Temperature normal, pulse 76. There is pain on palpation all over the abdomen, more particularly in the epigastric and right hypochondriac regions. The abdominal aorta is felt to be pulsating in a marked manner, otherwise nothing can be felt. There was the same negative result on examination by the rectum and vagina.

On December 4th an incision was made downwards from the umbilicus, the abdomen was opened and the kidney explored, with no result. The appendix was then brought outside the wound. It appeared to be healthy, but it was considered advisable to remove it. The wound was then closed. She made an uninterrupted recovery, and was discharged on December 23rd, stating that she was quite free from pain. A month afterwards she wrote to say that she was perfectly well and had had no return of her symptoms. The appendix was examined and found to be free from disease. In some positions of the appendix it is very near to the kidney, and the symptoms of the one are very liable to be referred to the other. Renal pain is rarely referred to the epigastrium; that due to the appendix not uncommonly so. The pain down the leg she complained of is sometimes met with in appendicitis, due to the spread of inflammation to the sheath of the psoas. In this case the examination by rectum and vagina revealed nothing, but it should not be omitted in such cases: the appendix may hang down into the pelvis, or a small twisted ovarian cyst may be the cause of the symptoms. The fact that nothing was found and yet her symptoms disappeared after operation may be accounted for in two ways. In a recent article Mr. Arbuthnot Lane explains this as follows: "Where faecal matter is allowed to accumulate in the intestines, one of the most favoured spots is the cecum and the ascending colon. From irritation by faecal masses its walls become inflamed and adhesions form between it and the adjacent wall of the abdomen externally, as well as in the neighbourhood of the kidney. When you cut down, expecting to find a stone, you...

1 Lancet, January 2nd, 1904.
carefully separate the kidney and its pelvis from the surrounding structures, explore the organ, and find nothing. Nevertheless, all pain and discomfort ceases from that date. The adhesion which probably caused a kink in the bowel at the hepatic flexure has been broken down and the obstruction removed." Though no marked adhesions were found in this case, it is possible there may have been very slight ones. I have seen two years' persistent pain ended at once by the breaking down of a tissue-paper-like adhesion between the stomach and the abdominal wall, an adhesion which was far too weak to affect the shape of the organ in any way. The other explanation is that she might have been cured by hypnotic suggestion. The pulsation of the aorta and the history of periodical retention of urine are in favour of a nervous origin, which her appearance did not in the least suggest.

Speaking of the nervous origin of symptoms, here is a case the sequel of which it is difficult to explain except on that theory. A strong, robust lad, aged 17, came to hospital on February 2nd. His story was that on January 16th, after lifting heavy boxes, he had a severe attack of pain in the right side of the abdomen. He kept at his work for the next few days, the pain being intermittent. On the 29th he was worse and a few days later he came to hospital. He had never had anything of the kind before, but had suffered at times from stomach-ache; had always had good health. On admission temperature was 98.2° and pulse 72. The abdomen was not distended and moved well on respiration. There is an extremely tender spot an inch below and two inches to the right of the umbilicus. His bowels are open freely. On February 4th he complained of a severe increase of pain in the right iliac fossa, which was very tender: nothing was felt. His temperature was normal, and his pulse 96. Two days afterwards the appendix was removed. It appeared to be quite healthy, but contained a small quantity of faecal matter. In the next five days he went on perfectly well, and then suddenly his temperature ran up from 98° to 103.6°, he had a rigor, and complained of great pain in the abdomen. The wound was opened up at once and everything found to be normal; it was closed, and he has gone on perfectly well since. This sudden disturbance is hard to explain. In an adjacent ward I saw the same thing occur in a girl who had been in bed for months with sacro-iliac disease, with no rise of temperature. One day, with no apparent cause, she had a rigor and her temperature ran up to 105°. In a few hours it was normal and remained so. An opportunity does not often occur of examining what proved to be an aseptic wound
five days after operation. No adhesions were found and the various layers had united separately. Apart from this episode the case is fairly typical of a large class of such, in which no signs of inflammation are found on operation and the appendix appears to be normal, perhaps containing, as in this instance, a little fecal matter. There was no history of attacks of pain localised in the appendix region, though there was of "stomach-ache." The onset was sudden, and attributed to exertion. To this class belongs the next patient.

J. S., aged 26, was admitted on December 8th for abdominal pain in right iliac region, which he stated came on suddenly ten days ago, when he was at work. He had no vomiting then, but was very constipated. He kept to his bed till coming to hospital, the pain being intermittent. He has had two previous attacks of abdominal pain, one in December, 1902, the other in June, 1903, but on both these occasions it was not localised to any particular region of the abdomen. He has never had any vomiting, but has always been constipated at these periods. This was the case on this occasion, when his abdomen was found to be rigid, and there was tenderness in the right iliac fossa. Temperature and pulse normal. After being kept quiet for a week, the pain soon disappearing, the appendix was removed. There were no adhesions or signs of peritoneal inflammation, but the organ was swollen and kinked; He made an uninterrupted recovery.

Resembling the two previous cases, in that no signs of inflammation were found in the appendix on removal, but differing from them in that there had been several definite attacks of localised pain, is the following one. A girl, aged 16, comes into hospital on February 7th with this history. During the past two years she has had four attacks of abdominal pain. The first was in June, 1902. As so often happens, it came on suddenly at night. It was low down in the right side of the abdomen. She vomited, and for several days afterwards was constipated. There was also a history of fever. She felt unable to do her work, but did not take to bed till that evening. She stayed in bed two days and got up feeling quite well. Up to the present time she has had four such attacks, all much of the same character. The last one, a month ago, was the worst and she remained in bed a week. She finds that she is unable to eat anything indigestible without bringing on an attack. A long walk or a bicycle ride has the same effect. After a week’s rest her appendix was removed. There were no adhesions or signs of previous inflammation, but it was a little swollen and kinked, being bent almost at a right angle by a very short mesentery.
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Now these three cases agreed, in that no signs of inflammation were found on removal of the appendix; in two it was kinked, in the third there was faecal matter in the tube. In symptoms they differed somewhat; the girl had a history of several attacks localised in the right iliac fossa; one boy had a history of "stomach-ache," and typical cases of appendicitis are often, especially in children, preceded by what are considered to be attacks of indigestion; the other boy had a distinct history of attacks of pain which were felt all over the abdomen. What was the cause of these symptoms?

It has been stated that every instance of so-called appendicular colic, slight appendicitis, catarrh of the appendix, and so forth, is really dependent on an actual inflammation of the appendix, and that of a bacterial origin. The absence of signs of inflammation and of bacteria in the organ when removed, are accounted for by the suggestion that they have disappeared in the quiescent interval usual before operation. Yet where there has been inflammation, and the patient has been kept quiet for two or three weeks after the temperature has fallen, one is sometimes surprised to see, not how few, but how many signs of past trouble are present. The adhesions may be so vascular and oedematous that it would appear better to have delayed even longer before operation. It is difficult to understand how, if the former class of cases were due to inflammation, it had not spread through the walls of the appendix to the peritoneum and caused adhesions around it.

The explanation of others, who say that many of these transient cases are not inflammatory in origin, but are caused by the efforts of the distended appendix to get rid of its contents, resulting in colic, seems to me more reasonable. It is known that when non-stripped muscle is stimulated to strong contraction violent pain results, as in biliary and renal colic. This would explain the sickness and pain, and also the sudden cessation of these symptoms when the offending matter is expelled. One does not assume that an attack of local peritonitis accompanies every case of biliary colic. The appendix may be distended by its own viscid secretion, or by a concretion formed in situ round a nucleus which may, or may not, have come from the cæcum. The condition found in these three appendices will explain the obstacle of the egress of their contents. In one, the straight one, there was present faecal matter, which is foreign to the healthy appendix. It had not been there long enough to become hard, but was still able to block the tube. In the others

Hunterian Lecture, Lancet, March 14th, 1904.
the organ was kinked by a short mesentery, in one case nearly to a right angle. Even then, under ordinary circumstances, it might allow the passage of its contents into the cæcum. But given any condition which still further reduces the lumen of the already narrowed tube, a spasm of the muscular wall ensues and severe pain, referred to widely-spread areas, is felt. Such a cause would be catarrh of the intestines spreading to the appendix, as does that of the duodenum to the gall-ducts. And catarrh may be caused by errors in diet, by constipation, or by exposure to cold and wet; there is often a history of such in these cases. Again, a twist or kink in the appendix may be aggravated by severe muscular exertion increasing the pressure on the abdominal contents; it is noticeable how often an attack follows unusual efforts. A curious instance of mechanical obstruction is related by Dr. McDougall in the Lancet of February, 1903. His patient in ordinary health was suddenly seized with a severe attack of abdominal pain followed by profound collapse. The abdomen was opened as soon as possible, but nothing was found except a barley corn, apparently of recent entry, impacted in the appendix. Sir Frederick Treves says that "the greater proportion of cases of appendicitis recover spontaneously." Doubtless this class contains many of such cases as the above, where there has been no implication of the peritoneum. But it can easily be seen how such an event may supervene at any time. The engorged appendix may become strangulated and gangrenous; or short of that, its walls may become so damaged as to permit of easy passage of bacteria to the peritoneal cavity; perforation in the ordinary sense is no more needed than in intestinal obstruction preceding peritonitis. The great difficulty is that the transient symptoms of these cases, where the appendix is only temporarily obstructed, are often exactly simulated by others where structural charges are already present. Here is a case in point.

A farm labourer, aged 35, came into hospital on March 9th. His story was that up till February 1st he had never been ill, nor was there any history of even abdominal discomfort. On that day he had his tea as usual, at 4.30, and went to bed at 9.30 feeling quite well. After he had been in bed for an hour he was seized with violent pain in the right iliac fossa, which kept him awake all night. In the morning he vomited once, he felt ill, and was constipated for the next three days, but from that time had felt quite well. He appeared to be in perfect health in every way, and nothing abnormal was detected in his abdomen. A few days later
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his appendix was removed. It was very tense and distended, like a sausage, with a very tight constriction close to the cecum, formed by adhesions. Their condition warranted the belief that they were of much older date than the late mild attack, which was the first of which there was any record at all. And yet from the state of the organ there was the risk of strangulation and gangrene at any time. So that, though one cannot help believing that there are many cases in which, at first, inflammation does not play any part, yet their symptoms may be so exactly simulated by others, even apparently first attacks, in which the gravest possibilities exist, that these should never be lost sight of.

Before passing on from these non-suppurative cases to those in which pus has formed, I will digress for a short space to describe Mr. Bowlby's method of operating during the quiescent interval of such cases, as it differs from the plan I have usually seen described in the text-books. The great point of it is that the rectus muscle is used as a buttress for the scar. An incision is made from the umbilicus through the anterior layer of the sheath of this muscle, parallel with its outer edge, but half an inch internal to it. The rectus is then retracted towards the middle line, whilst a further incision is made through the posterior layer of the sheath and the peritoneum. On closing the wound these two layers are united by one series of sutures, the muscle slips back into its place over them, and the anterior layer of the sheath and the aponeurosis are taken up together, and lastly the skin. In a fortnight the patient is out of bed, and, as far as the scar is concerned, could play football in a month. No belts or trusses are needed. This method of opening the abdomen also has the advantage that the incision can be prolonged, if necessary, without dividing muscle fibres, and should there be need to explore other organs it affords a ready access to them.

I will now consider some cases which have gone on to suppurative.

A. B., a carpenter, aged 18, was admitted on Sunday, November 22nd, 1903, with the following history. On the previous Thursday he was quite well, and there was no history of similar attacks. At 9 p.m. on Friday he felt a pain in his right groin and vomited several times during the night. On Saturday he went to work, but the pain continued and he had to give it up. He walked to the hospital next day. His temperature was then found to be 102.8°, though his pulse was only 96. He looked very ill. There was a slight swelling in the right iliac region and the skin over it was red, hot, and
very tender. In the evening, after a few hours' rest in bed, his
temperature was down to 98°8, but his pulse had risen from 96 to 112. Next morning his temperature was 100°6 and pulse 104. In
spite of having had nothing by the mouth he was vomiting. In the
afternoon his temperature was 98°8 and pulse 116, rising to 120 shortly before operation. His abdomen was distended and no flatus passed. By an incision over the swelling some thin pus was
evacuated, which appeared to be shut off from the general peritoneal
cavity. No search was made for the appendix. The wound slowly
granulated up and he recovered after a long illness. This case well
indicates the great importance of the relationship between pulse
and temperature. If the temperature is falling at the same
time that the pulse is running up, and especially if there be
vomiting, it points to immediate operation. It will be noticed that
the appendix was not sought for. To have done so would have
to run the risk of breaking down adhesions and of flooding the
peritoneum with pus. The gut in such cases is apt to be soft and
frangible. It is best to merely drain. The débris of the appendix, or
concretions, if any, will probably come away in a few days. A few
of these very acute cases of suppuration recover, without operation,
after a desperate illness. In such there is not the same indication
to operate for the removal of the appendix, after recovery, as there
is in the milder cases. The chances are that it has already been
destroyed by the intensity of the disease, and that everything is
matted together in its neighbourhood. By interfering you run the
risk of doing more harm than good.

Now comes a case where, though pus was present in large
quantity, most of the signs of it were absent for a long time. An
everly stout man, weighing about 20 stones, with a hand's
breadth of fat covering his abdomen, came to hospital on May 14th,
complaining of pains in the abdomen, especially in the right lower
quadrant. He stated that they began four days before, and had
been gradually getting worse. He had not vomited at all, but had
been constipated for some time. His temperature was 100°2, pulse 84. Nothing could be felt in the abdomen owing to fat.

His temperature rose to 102° one night, but after his bowels had
been cleared out by enemata it did not rise above 99° at night, and
a degree less in the morning. Pain passed off and he appeared to be
doing well. On the 22nd a blood count gave 22,000 leucocytes,
and the next day fluctuation was detected. An incision was made
and half a pint of pus was let out. He made an uninterrupted
recovery.
In this case the temperature came down after enemata, the pulse was 70 to 80 throughout, and there was very little pain. The indication given by the leucocytosis proved to be correct. Opinions differ about the value of this sign. It has the charm of novelty, and perhaps too much is expected of it at first; but if taken in combination with other signs it may be most useful. Sometimes it misleads; I have seen a case where there was a leucocyte count of 27,000 and a temperature chart pointing to pus. Instead of a pelvic abscess a haematoma was found. In another case in which it was absent a large abscess was evacuated next day. Perhaps the safest line to take is that its absence proves nothing, though its presence is confirmatory. Especially is this the case where the blood counts show a progressively increasing leucocytosis. Speaking generally, the more intense the poison and the greater the resistance, the higher will be the leucocyte count.

I will conclude the series with the worst case which I have ever seen recover. It illustrates not only that general peritonitis may be present for two or three days without many of the characteristic signs, but also that pus may form in large quantities during apparent intervals of improvement and give no indication of its presence.

A young blacksmith, aged 22, was admitted on January 14th, complaining of constipation with considerable abdominal distension, but not much pain. His temperature was 101° and pulse 96. He was greatly relieved by enemata, which brought away large fecal masses, and on the afternoon of the 15th his temperature was 99° and pulse 80. The distension remained, but he had no sickness or pain, and there was no indication for operation. For the next two days his progress appeared satisfactory, his bowels were open freely and he was much more easy; pulse about 70 and temperature 99°. On the evening of the fourth day he was suddenly seized with great pain in the abdomen, and showed all the signs of intense toxæmia. The abdomen was opened and a large quantity of pus evacuated. He was almost pulseless, and it was doubtful if they would get him off the table alive. However, they did so, and he rallied in such an extraordinary manner that for the next six days he appeared to be improving rapidly, his pulse being about 100° or under, and his general condition far better than could have been expected. He was then taken very much worse and a large sub-diaphragmatic abscess was found and evacuated, the pleural cavity being also involved. Then, again, for four days his temperature fell to normal, his pulse was 100 or under, he was quite free from pain, and very
cheerful. On the 27th he vomited, the abdomen was distended, there was tenderness over the pubes, and he had pain when micturating. An incision was made over the pubes and a pint and a half of pus let out. After this there were no more collections of pus and he recovered slowly. In seven weeks' time he was sent to the convalescent home, all the wounds having closed except the thoracic one. When I saw him two months afterwards he had put on several stones in weight, and except that the upper opening had not quite closed, was apparently well: an example that there may be a chance for the most hopeless.

The absence of symptoms in this case was very marked. Here was a man whose pulse was not increasing in rapidity, who was not vomiting, and whose bowels were freely open, and yet he had general peritonitis and a large collection of pus in his abdomen. The same lack of symptoms is to be seen in the intervals between the operations when pus was forming in large quantities. The toxins seem to have had a cumulative effect. Sub-diaphragmatic abscess is not an uncommon complication where the appendix has been the origin of mischief. The other localised collection of pus in his pelvis was faithfully indicated by the pain in micturating just prior to his last relapse.

In relating the above cases I have tried to deal with the points of interest in each as they occurred. To recapitulate briefly: To start with, there were two cases which show that, though we no longer speak of "that heaven-sent peritonitis which was called idiopathic," still, from time to time, instances occur in which none of the viscera appear to have been the starting point of the mischief, and so, perhaps, we are justified in speaking of primary peritonitis; a term which can usually be qualified by the name of the offending organism. Then came two cases of chronic tuberculous disease, the one local and the other general to the peritoneum, whose symptoms resembled those of acute appendicitis of the milder type. Next was one where the disturbance caused by menstruation simulated a perforation of the appendix or stomach, the history of haematemesis adding to the probability of the latter.

The symptoms of another young woman, apparently due to the kidney or appendix, disappeared after the abdomen had been opened, though nothing was found. After this were four cases illustrative of catarrhal appendicitis, so called. They showed that in some cases there is every reason to believe that the symptoms of this variety may be due to temporary blocking of the appendix, and not to inflammation; that, on the other hand, inflammation may
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readily follow if this condition is prolonged, and that it is very hard to say when it is not present, a case being quoted of apparent appendicular colic, and a first attack, where old adhesions were found with the possibility of the gravest results.

Finally came three cases where suppuration had taken place. They showed the importance of the pulse as opposed to that of the temperature, the help that leucocytosis may give in diagnosis, the latency of symptoms during pus formation, and the possibility of recovery in the gravest cases.

The lesson to be learned from these cases seems to me to be that the symptoms of disease within the peritoneum are protean; that one must regard the mildest of such cases as fraught with the gravest possibilities, and the most quiescent interval with danger; and that of all signs the one to be most relied upon is the condition of the pulse.