friends were at first amused, and did not realize that it was not part of the entertainment. They then came to his help and gave him a glass of water, which he was unable to swallow, the fluid regurgitating through the nose. This was followed the next day by paralysis of the left leg, left arm, and left side of the face, including the upper part, and for a month he had to be fed with a tube. This happened in Japan; the doctor treated him by inunction, and after a month he was able to swallow and speak. He was sent to me on his return from Japan suffering with the remains of a left-sided paralysis, including the upper facial muscles, and there was also paresis of the left vocal cord and soft palate. He attended my practice for some years without further symptoms, and gradually improved.

I do not believe there is any reasonable ground for questioning the syphilitic origin of my case, as it occurred as one of a series of syphilitic phenomena which yielded rapidly to specific treatment, and the patient's condition at the present time is not in favour of its being a manifestation of that curiously irregular disease—disseminated sclerosis.

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THE IMPORTANCE OF A LEUCOCYTE COUNT IN DOUBTFUL CASES OF APPENDICITIS.

BY

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THE cases here recorded illustrate in a marked degree the importance of taking a leucocyte count in doubtful cases of appendicitis. This weapon of diagnosis is without doubt daily used, and is, in my opinion, of paramount importance in elucidating vague symptoms referable to the appendix region; it tells whether we may temporize, or whether, on the other hand, we have no time to spare. It is on these cases with a high leucocytosis, and with the constitutional symptoms masked, often both pulse and temperature below 100, that I wish particularly to lay stress; it is these cases which, if watched, so often end in the tragedy of acute suppurative peritonitis; it is, moreover, more common in a first attack, where adhesions do not exist to shut off an appendix distended with infected contents, with usually a stercolith, gradually burrowing its way through the muco-muscular wall of some portion of the appendicular canal; if, in these cases a count shows a leucocytosis of anything between 15,000 and 30,000, the appendix should be at once explored, and the picture usually presented is a semi-gangrenous appendix, the serous coat of which is intact, the lumen distended with foul pus, with one or more stercoliths, but no adhesions, which renders the operation of removal easy and safe.

CASE I.

R. C., aged 14 years, had had no serious illness, but had suffered occasionally from attacks of indigestion.

He was quite well at school on November 13th. He was seen by Dr. Leslie Watt at 9 a.m. on November 14th, when he complained of pain in the right iliac fossa, increased on pressure; he had vomited several times during the night. The pulse was 84, the temperature 98.4° F.; there was no rigidity of the right lower rectus abdominis. He was seen again on the same day at 8 p.m., when he looked flushed, the pulse was 104, and the temperature 100.8° F. There had been no vomiting since the early morning. Hot belladonna fomentations were applied. He was seen again at 8.30 a.m. next day, November 15th. He had had a comfortable night, and looked much better; the pulse was 84, the temperature normal, and pain slight. I was asked to see him at noon; his general appearance was good, and did not in any way suggest the necessity for an operation; pulse and temperature were normal. I took a specimen of blood, and Dr. Pethybridge reported a leucocytosis of 25,000. Immediate operation was urged.

The appendix was found lying free, without adhesions; it was of large size, 1 in. in diameter at about the centre; towards the base it appeared normal, and this was clamped, the stump buried in the caecal wall, the meso-appendix tied off, and the parietes closed in layers without drainage. On opening the appendix it was found tightly distended with foul pus, and there was a large stercolith; the anterior two-thirds of the mucous and submucous coats were gangrenous. Recovery was uneventful.

I have not the slightest doubt that, but for the blood

count, this case would in twenty-four hours have become an example of a fulminating appendicitis.

CASE II.

T. L., aged 46, seen in consultation with Dr. George Hillyar. On November 19th he played golf; at night he had pain in the right iliac fossa. I was asked to see him next day (November 20th). There was some tenderness in the right iliac fossa, but no rigidity of the rectus; the patient did not appear very ill. I was doubtful whether the appendix was at fault, as he had previously suffered from attacks of pain referred to the region of the gall bladder; a specimen of blood was taken: Dr. Pethybridge reported a leucocytosis of 14,800.

He was at once removed to a nursing home, and, assisted by Dr. Hillyar, I made an incision over the right rectus; the appendix was found much swollen, coiling round, and plastered to the fundus of the caecum; the parietes were sutured without drainage. The appendix contained a stercolith and 2 drachms of foul pus. This body had a marked resemblance to a gall stone, but it proved to be a stercolith. The patient made a good recovery and left the nursing home in seventeen days.

CASE III.

D. T., aged 8, seen in consultation with Dr. W. L. Hodge. On August 30th he was watching a cricket match; when he returned home at 7 p.m. he complained of abdominal pain; he vomited during the night and had several loose motions; the pain, which was paroxysmal and extended over the abdomen, subsided. At 8 a.m. next day (August 31st) the temperature was 100.6°, the pulse 120; acute abdominal pain returned at noon and continued till 4 p.m. and was more localized in the right iliac fossa; his condition then improved. The temperature was 100° and the pulse 98; the tongue was moist. A leucocyte count was made by Dr. Pethybridge, who reported 22,000.

Immediate operation was performed and I found the appendix of large size and gangrenous, and the gangrene had invaded the serous coat, although rupture had not taken place; infection of the fossa was evident, necessitating drainage. The base of the appendix was divided and buried in the caecal wall. The patient left the nursing home in four weeks and rapidly regained health.

In this case the symptoms were apparently improving and the patient's father, a medical man, was in favour of delay before the blood count was made. I am of opinion that with delay of twenty-four hours the result would probably have been different.

OBSERVATIONS ON THE FUNCTIONS OF THE COLON, WITH SPECIAL REFERENCE TO THE MOVEMENTS OF ENEMATA.

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A STATEMENT by Professor Rutherford Morison to the effect that enemata will travel along the whole of the large intestine and that succussion of the fluid injected can be obtained in the caecum, directed my attention specially to the subject and led to the following observations. He made this remark before x-ray apparatus was in use, and therefore before the method of examination by bismuth enemata was carried out as an aid to diagnosis of disease, and at the time, I believe, its truth was generally doubted.

It has now been definitely proved that in animals as well as human beings an enema will travel through the whole course of the large bowel, and the object of my paper is to illustrate this and to record some interesting observations, the result of personal experience with the fluorescent screen, in investigating a large number of cases when bismuth and barium enemata were employed for diagnostic purposes.

Cannon¹ has shown experimentally that in cats enemata will not only find their way into the caecum, but when injected in large quantities will travel through the ileo-caecal sphincter into the small intestine; and on one occasion he observed that the enema injected entered the stomach, for the animal actually vomited a portion of it. He found, however, that the ileo-caecal sphincter remained competent when small enemata were employed, and that to pass the caecum an enema had to be injected under considerable pressure.

Gruttyner's² observations, published in 1894, seemed to prove that a salt solution containing powdered charcoal when injected into the rectum was carried up the whole

length of the alimentary tract into the stomach, but his results were not confirmed by later observers. Hertz³ affirms that an injection of bismuth when run into the rectum at low pressure may pass into the ileum, and the same statement has been made by other observers.

My own observations made on patients go to show that enemata invariably travel to the caecum, but rarely any further in normal cases, and that when they do pass the ileo-caecal valve there is, as a rule, some pathological condition present, a point illustrated by the following cases of appendicitis in which intestinal obstruction developed some days after operation for removal of the appendix. The first case was that of a boy aged 14; an appendix abscess was opened and drained, and the appendix removed, and the following day enterostomy was performed for acute intestinal obstruction. After this it was noticed that when an enema was given the salt solution flowed freely out of the enterostomy wound. He succumbed on the fifth day after the second operation, and *post-mortem* examination showed that the enterostomy opening was situated 7 in. above the ileo-caecal valve, and that there was no fistulous communication between the large and small gut. This, of course, proved that the water had not travelled by a short-circuit route.

The second case was a boy, aged 10, who was operated upon for acute perforative appendicitis. The appendix was removed and drainage of the peritoneal cavity was established. Acute obstruction of the small intestine occurred on the eleventh day after the operation, and this necessitated an extensive enterectomy, followed by a small bowel anastomosis; this leaked, and was followed three days later by faecal fistula. Rectal injections of water escaped through the fistulous opening in the abdominal wall. In this case half a pint of water had to be administered per rectum before it appeared on the belly wall.

The third case was that of a labourer, aged 24, who was admitted to hospital with general peritonitis of a week's standing. A fortnight after his admission the appendix was removed. The patient developed a faecal fistula which, at a subsequent operation performed with the object of closing the same, was shown to originate from a loop of small intestine. After this, whenever saline was introduced by the rectum, it came out of the faecal fistula in the belly wall; this took place after 10 oz. of fluid had been injected into the rectum.

It was inferred that in these cases there was paralytic distension of the caecum and adjacent small gut, rendering the ileo-caecal valve and sphincter incompetent.

When employing bismuth or barium enemata for diagnostic purposes it was my practice to place the patient on his back and to inject the fluid slowly and rhythmically by means of Higginson's syringe. Where there was no organic obstruction of the large bowel it was found, as a rule, that bismuth reached the caecum in ten minutes or under (a result rarely obtained before a pint was introduced). The more rapidly fluid was pumped in the sooner the caecum was reached, provided the pressure was not sufficiently great to cause discomfort to the patient and the return of the fluid. The rule was that $1\frac{1}{2}$ or 2 pints were necessary to reach the caecum, and this agrees with the observations of Haenschhof, who found that the colon was traversed after 1 litre was introduced—namely, 1.76 pints. Hertz noticed that after more than $1\frac{1}{2}$ pints were used with a pressure of $1\frac{1}{2}$ ft. of water, or even less, the colon contracted and rejected its contents.

At times, no doubt, fluid travels along the bowel at a more rapid rate—for example, in two instances the caecum was reached in eight minutes and the splenic flexure in three and two minutes respectively, in the former case when 6 oz. had been introduced.

I also found that whilst a pint of barium-charged fluid arrived at the caecum in eight minutes, a similar quantity of a fluid of thinner consistency (sterilized milk or water) reached the proximal end of the colon just as quickly—for example, a man aged 40, upon whom caecostomy had been performed for acute colitis, was given an enema of 18 oz. of sterilized milk, and in six minutes this fluid appeared from the caecal fistula. On another occasion the same patient was given an enema of similar nature—12 oz. in amount—but it did not reach the caecum before twenty-five minutes.

Hey Groves⁴ made a similar observation in a lad aged 19, with a faecal fistula, the result of acute appendicitis. He found that on injecting from time to time a coloured fluid into the rectum it made its appearance in the caecum after 15 oz. had been given, the amount varying only within limits of an ounce or two. He also observed that the fluid appeared at the caecal fistula not in a continuous stream, but in jets, occurring from four to five times a minute, and from this he concluded that antiperistalsis was responsible for the backward movement of the injection.

With regard to the question, To what extent, if at all, does antiperistalsis take place? it may be mentioned that in none of the cases above referred to could I see any indication of reversed peristalsis. Cannon,⁵ in 1902, published observations made on cats after bismuth meals. He found that antiperistalsis definitely occurred and that such movements were limited to the proximal half of the intestine. In 1904 this observation was confirmed by Elliott and Barclay Smith,⁶ who demonstrated it in the rat, guinea-pig, dog, hedgehog, and ferret, and concluded from their observations that "antiperistalsis is a characteristic feature of the muscular activity of the colon."

Hertz⁷ investigated antiperistalsis in the human colon, but discovered no movement which suggested antiperistalsis, and he came to the conclusion that the passage of fluid from the rectum to the caecum was brought about solely by hydrostatic pressure. This opinion is supported by *post-mortem* investigation. For example, at an autopsy on the body of a man a measured quantity of water was introduced through the anus with a Higginson syringe, and it was found that between 2 and $2\frac{1}{2}$ pints had to be injected before the fluid reached the caecum. This result I have demonstrated on at least two occasions.

It was frequently noticed that when the bismuth or barium were clearly shown in the caecum—that is, when the large bowel had been traversed and its outline revealed—there was little or nothing to be seen to mark the iliac colon, due possibly to tonic contraction of this segment of the bowel. At two points at least the passage of fluid along the colon is apt to be retarded—namely, at the junction of the rectum and pelvic colon, and at the splenic flexure, as Hertz has pointed out—at least with regard to the former site. Experience in the use of the sigmoidoscope throws light upon this, as the anatomical kink may be so acute that at times, even under general anaesthesia, the instrument cannot be passed beyond the rectum. In one case in which there was no obstruction, as shown *post-mortem* (for the patient died after an abdominal operation a fortnight later), the barium, even when 2 pints were injected, could not be coaxed beyond the splenic flexure.

Whatever may be the force at work in passing fluids injected in quantity along the colon, whether hydrostatic pressure or antiperistalsis, there must still be some doubt as to the means by which small enemata travel along the bowel, as I think they certainly do.

A nervous hypochondriacal man complained of constipation. His bowels were cleared with a dose of castor oil and by enemata, and at 9 a.m. an enema of potato-starch solution (5 oz.) mixed with bismuth was injected into his rectum, where it was seen by the fluorescent screen. Three hours later it was seen to be still confined to the pelvis. Six hours after the injection it was found to have left the pelvis, and the outline of the descending colon, up to the splenic flexure, was plainly seen, whilst there was still a mass in the rectum. The patient was not examined again for eighteen hours—that is, twenty-four hours after the enema had been administered, and it was then found that the solution occupied the whole of the large intestine up to the hepatic flexure. On examination thirty hours after the injection the bismuth had gone back, so that the ascending colon was occupied down to the level of the iliac crest. Forty-eight hours after the injection it could no longer be made out in the transverse colon; what remained of the mass lay chiefly in the rectum, and only a thin coating could be seen in the descending colon. Although the solution was never seen in the caecum, it seems to be a fair assumption that it reached there some time between the thirtieth and the forty-eighth hour.

Bearing upon this question, the observations of Bond⁸ are of interest. He found that indigo granules were present in the mucus from a caecal fistula from twenty-four to forty-eight hours after the dye had been introduced

into the rectum. He states that this back flow occurs independently of muscular contraction, and is brought about by a reversed mucous current along the bowel.

Advantage was taken of the opportunity to investigate the relation between the small bowel and the colon in some 5 or 6 cases of ileo-colostomy (division of the lower end of the ileum and implantation of the proximal end into the pelvic colon low down). An enema of barium sulphate was given in the way already described, and its course watched as it travelled along the bowel. In cases in which the operation had been performed recently—within a month or so—the barium was seen to travel up the large intestine, and in one case to the caecum, but there was no indication that it entered the small bowel.

On the other hand, in cases in which the operation had been performed one or two years previously, the solution ran up into the small bowel, and was seen in distended loops of small intestine lying on the right side of the abdomen; it was not until a considerable amount of the fluid was so disposed that the large intestine proximal to the anastomosis began to show a shadow.

This observation suggests that in cases of ileo-colostomy in which the operation has been performed some considerable time—a year or more—the dilated coils of small intestine adjacent to the colon assume somewhat the functions of the large gut.

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THE PRE-PERITONITIC STAGE OF ACUTE APPENDICITIS.

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If the mortality from appendicitis is to be reduced almost to vanishing point, it is essential that the earliest signs and symptoms of the condition should be appreciated clearly; the view is accepted in America and is gaining ground in England that every case of acute appendicitis should be operated on within the first twenty-four hours from the onset, or as soon after as is possible. There are two reasons why cases are operated on later than this—either the patient may think that the symptoms are not serious enough to seek medical advice, or his adviser may think the symptoms not typical of appendicitis or not serious enough to demand operation. It is clear that we have no remedy against the first cause save the education of the public, but in regard to the second something can be done by way of explaining that the so-called typical symptoms of appendicitis as given in the textbooks often indicate a somewhat advanced stage of the condition, and that it is impossible to say at the beginning of an attack whether it is likely to be mild or severe in type.

It is desirable and in most cases possible to diagnose appendicitis before peritonitis has set in, or at least before there is any more than that slight amount of congestion of the peritoneum which is commonly associated with any inflammatory process within the gut.

It must be understood that the pre-peritonitic stage does not always coincide with a simple catarrhal state of the mucous membrane; quite commonly there is deep ulceration of the interior of the appendix, and sometimes even gangrene of part of the wall extending through to the peritoneal coat.

In diagnosing the pre-peritonitic stage of acute appendicitis the following points must be noted:

(a) The History just Prior to the Onset of Pain.

There is often a history of some unusual irregularity of the bowels. Constipation is of less significance than diarrhoea, which is especially common in children. The occurrence of diarrhoea is likely to mislead; it is probably

due to activity in the caecum and colon of the virulent microbes which cause such damage in the appendix. This early diarrhoea has to be distinguished from the later variety due to irritation of the rectum by pelvic peritonitis or a pelvic abscess. Frequently also there is an account given of flatulence or indigestion for a few days prior to the onset of the attack; in a patient who has never previously been subject to pain after food this history should be sufficient to put one on guard.

(b) The Symptoms of the Attack.

The early symptoms are pain, nausea or vomiting, local deep tenderness as felt from the abdomen or by the rectum, sometimes rigidity or local distension, and generally fever. The pain in the majority of cases is first referred to the epigastrium and is later localized in the right iliac fossa. But this localization is not by any means constant in the pre-peritonitic stage. Sometimes the patient asserts that the initial pain is felt all over the abdomen, or it may be hypogastric in distribution. When the appendix is in the retro-caecal position the pain may be from first to last felt only in the right iliac fossa. The causes of the early pain are probably two. First, and most important, is the exaggerated peristalsis of the appendix excited by the relative or absolute obstruction to its lumen by a concretion, a kink, or swollen mucous membrane; bacterial infection causes the accumulation of irritating products, which lead to a distension of the appendical lumen. Secondly, there may be bouts of pain caused by painful peristalsis of the caecum. Vomiting generally, but by no means always, occurs in the pre-peritonitic stage, and usually the patient only vomits once or twice. Nausea is nearly always present, even though there be no vomiting. The degree of nausea and the amount of vomiting in this stage appear to depend on two factors—first, the amount of distension of the inflamed appendix; and, secondly, the reflex nervous susceptibility of the patient. Vomiting is thus more prone to occur in children, or in patients whose digestive tract is easily deranged. It may be taken as an important general rule that the severity and frequency of the vomiting at the onset indicate the degree of distension of the appendix, and consequently the immediate risk to the patient that perforation may occur.

Local deep tenderness is probably to be elicited from the onset of the attack, but at first it is frequently masked by the more generalized referred pains. When the latter have subsided the local deep tenderness is easily elicited. Quite frequently there is no superficial tenderness in the early stage.

Rigidity of the abdominal muscles over the appendix is not a constant symptom. In most cases well-marked muscular rigidity coincides with commencing peritonitis, though I have certainly seen it when no peritonitis has been present; but it is quite a common experience (I believe it is the general rule) not to get rigidity in the pre-peritonitic stage. I am certain that the failure to realize this fact is responsible for many delayed operations and lost lives. An appendix may be on the point of bursting into the general peritoneal cavity without a single adhesion to limit infection, though at the same time the abdominal wall may be flaccid and allow of free manipulation without any rigidity appearing. This fact must be known to every surgeon of experience, but in general it is certainly not fully appreciated, nor do the textbooks make the point clear. Rigidity is taught to be one of the earliest signs of acute inflammation of the appendix, whereas in quite a large proportion of cases it is almost completely absent in the earliest stage and in some cases is absent even though pelvic peritonitis exists.

Fever may not be present at the beginning of the attack, but nearly always develops before twenty-four hours have passed. As a rule, the temperature does not go much above normal, two or three degrees being the average elevation. Mistakes are liable to happen owing to the fact that the temperature is not elevated at the time of the onset of the pain, and thus the more serious disease may be mistaken for an attack of simple intestinal colic. In any suspected case the temperature should be taken every two or four hours, and if it rise in a gradual manner it is a point in favour of appendicitis. If at the very beginning of any attack of acute abdominal pain the temperature is raised, the presumption is against appendicitis.