A Clinical Lecture

ON

ACUTE PERITONITIS DUE TO APPENDICITIS

AND ON SOME CASES OF SIMPLE PERFORATING ULCER
OF THE DUODENUM.

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APPENDICITIS.

THE specimens, gentlemen, which are on the table were removed from the body of a woman, aged 35, who was admitted to the hospital on January 21st, suffering from great pain in the abdomen, vomiting, and collapse. On examination it was found that she had general peritonitis and mitral stenosis. The condition of the heart and pulse was so bad that any operative interference seemed contraindicated.

The case presents several features of great practical importance; I take the opportunity of bringing it before you, and saying something with regard to acute peritonitis.

As I have said, this patient had mitral stenosis. A well-marked presystolic and a slight systolic murmur were present in the mitral area; the heart was beating extremely rapidly (150 to 160), and half of the cardiac pulsations did not reach the wrist; the heart's action was very weak and irregular.

On inquiring into the history, we found that since early life, as long as she could remember, she had been short of breath on exertion; that a month before admission to hospital she had had an attack of rheumatic fever, from which, however, she had completely recovered; that while going about her ordinary household duties she was suddenly seized with acute pain in the abdomen and vomiting. This, as I told you, was followed by the development of general peritonitis and collapse.

Now, two questions of course arose: (1) What was the cause of the peritonitis? and (2) How was the peritonitis to be dealt with?

Acute general peritonitis may be due to a variety of different causes, some of which may be regarded as medical accidents—that is to say, sudden unforeseen lesions, occurring in the course of some previous diseased condition. In the *Lancet* of January 27th there is a valuable clinical lecture by Mr. William Henry Battle on the "Acute Abdomen"; in that lecture he has tabulated all the cases of the "acute abdomen," as he terms it, which occurred in St. Thomas's Hospital during the three years 1900, 1901, and 1902. They were 456 in number; this shows that the condition is a common one. Of these 456 cases, 37 per cent. were cases of appendicitis and its complications; 24 per cent. were cases of intestinal obstruction other than intussusception; 16 per cent. were cases of intussusception; 11 per cent. were perforations of the alimentary tract, stomach or duodenum; 6 per cent. were gynaecological cases; 3 per cent. were cases of abdominal abscess other than appendicitis; and the remainder of 3 per cent. were not classified.

From this statement you will see that the great majority were cases of appendicitis, strangulation of the intestine, and intussusception. The cases of appendicitis and its complications, intestinal obstruction, and intussusception together amounted to no less than 77 per cent. of the total 456 cases.

In making a differential diagnosis, when you may have a variety of different possible conditions, it is of great importance to know not only what are the possibilities but what are the probabilities. A statistical analysis of cases, such as that published by Mr. Battle, is consequently of great value, provided of course that the figures are large enough to warrant general conclusions.

But we do not, of course, make a diagnosis merely on the relative chances or frequency of different diseased conditions; if that were so, it would be easy for any one to make a cut-and-dried diagnosis in every case. We have to take into consideration all the factors that go to form the individual and personal equation of the case. After having ascertained all of the facts we draw a logical conclusion from those facts, taking, of course, into account, as one of the facts on which our judgement is based, the relative frequency of the different possibilities (causes).

Well, in this particular patient we had an old-standing heart lesion (mitral stenosis) and a history of recent rheumatic fever. Now, in cases of old-standing valvular disease a recent attack of rheumatic fever is, as everyone knows, very apt to be attended with a fresh endocarditis. The history showed that this patient, after recovering from the rheumatic fever, was suddenly seized on Friday, January 19th, with acute pain in the abdomen and vomiting, followed by acute peritonitis. She was admitted to hospital two days later, January 21st, 9 p.m. Her condition was then very grave, and in the opinion of Dr. Greer, my house physician, precluded any operative interference.

I saw her the next day and confirmed the diagnosis of acute general peritonitis. The pain was most marked in the left hypochondriac region, and over the left side of the abdomen the skin was excoriated, the result of the application of hot fomentations; the fact that the fomentations had been applied over the upper part on the left side of the abdomen showed that in the early stages of the attack the pain was most marked over this part of the abdomen. I agreed with Dr. Greer that any operative procedure was out of the question; the patient's condition was so desperate that she would in all probability have died on the table if anything had been attempted; her heart was beating very irregularly at the rate of 160 to 200 per minute, and many of the pulsations did not reach the wrist.

As regards the cause of the peritonitis I was disposed to think that it was probably an embolism of the mesenteric artery. We had a patient with mitral stenosis; we had a history of recent rheumatic fever likely to produce fresh endocarditis; we had the sudden occurrence without any previous abdominal symptoms, of severe abdominal pain and vomiting, followed by acute peritonitis, the pain being chiefly located in the upper part of the left side of the abdomen. It seemed to me that it was probable that there was some connexion between the cardiac condition and the development of these fresh symptoms. I consequently concluded that the lesion was probably an embolism of the mesenteric artery. I have seen a few other cases of the same kind. It is a rare accident. All of you who know anything about mitral stenosis know that one of the risks in that condition is the production of embolic infarction. A little piece of clot becomes detached from a thrombus in the left auricular appendix, or—but this is much less frequent except in cases of ulcerative endocarditis—from a vegetation on the stenosed valve; it is swept into the circulation and carried to some distant vessel; it may be carried to the middle cerebral artery, producing hemiplegia and aphasia; to the spleen, producing acute abdominal pain and localized perisplenitis; to the kidney, producing perhaps pain over the kidney, and, if it is a large infarct, haematuria and albuminuria; to the mesenteric artery. If a large branch of the superior mesenteric artery is plugged, the blood supply is cut off from a large portion of the intestine; this causes gangrene of the gut and general peritonitis.

Let me briefly describe the leading features of a typical case which I met with a few years ago:

Case of Aphasia in which Death was due to Embolism or Thrombosis of the Superior Mesenteric Artery with Resulting Gangrene of Several Coils of the Small Intestine and Peritonitis.

Mr. A., aged 70, was seen in consultation on March 4th, 1898, suffering from aphasia due to embolism of the branch of the left middle cerebral artery supplying Broca's convolution. From the aphasic point of view, the case is, I think, one of the most interesting and remarkable cases which has ever been recorded; but this point does not concern us at present; any of you who would like to study it will find it recorded in *Brain*, vol. xxi, 1898, p. 343. The interest of the case for my present purpose is that the patient died from gangrene of the small intestine and peritonitis, the result of an embolism or thrombosis of the superior mesenteric artery.

After I saw the patient on March 4th, 1898, he slowly improved so far as the speech defects were concerned. On April 8th he was at business all day and seemed in good health. On April 9th he complained of pain in the region of the stomach. I saw him the next day (April 10th). He did not look ill, but there was considerable tenderness over the abdomen. The pulse was slightly quickened—about 80 per minute—but there was no temperature. Up to this date, from the onset of the aphasic attack, the pulse had been slow (45 to 50 per minute). On April 11th the temperature went up to 100°, and the pulse to about 96. He complained of intense

pain over the abdomen and vomited frequently. The abdomen was moderately distended and very tender all over. General peritonitis was obviously present. During the attack the bowels were more than once freely opened by medicine. There was no intestinal obstruction and no apparent cause for the peritonitis. On April 12th and 13th he remained much *in statu quo*. On April 14th he vomited a small quantity of blood. On April 15th he died. He remained mentally quite clear up to the end. On the day of his death he drew and signed a cheque quite correctly; the writing was good.

Post-mortem Examination.—This was made on April 16th. Several coils of the small intestine were black and gangrenous, and there was general peritonitis. There was no constriction and no hernia, either externally or internally. The portion of mesentery going to the affected part of the small intestine was markedly swollen and oedematous. The gangrene of the intestine was obviously due to blocking of the mesenteric artery. The abdominal aorta was somewhat atheromatous. The heart was moderately dilated, the valves healthy, the muscular substance markedly fatty and degenerated. The thoracic aorta was atheromatous. The kidneys were slightly enlarged and somewhat granular. (The urine was tested on several occasions during the course of the patient's illness, and was always free from albumen.) A few recent small petechial hæmorrhages were scattered over the mucous membrane of the stomach. The posterior end of Broca's convolution (third left frontal), the subjacent white matter, and the island of Reil were completely destroyed and softened; the posterior end of the second left frontal convolution was slightly affected.

In my experience, gangrene of the intestine due to embolic or thrombotic plugging of a large branch of the mesenteric artery always results in death. I do not know if any case of this kind has been successfully operated upon.*

I repeat that embolism of the mesenteric artery seemed to me to be the most likely cause of the acute abdominal symptoms in this particular case; and, if this diagnosis was correct, it was additional reason against operative interference, for the chances of successful operation are much less in the case of gangrene of the intestine due to embolism of the mesenteric artery than in cases of ruptured gastric or duodenal ulcer or to a ruptured appendix. The possibility of a ruptured appendix or a ruptured ulcer of the stomach of course occurred to us, but for the reasons I have already stated, embolism of the mesenteric artery seemed to me a much more probable cause in this particular case. But, as I have already told you, we did not entertain the question of operation; the extremely grave condition of the patient, and the fact that the course of the peritonitis appeared to be embolism of the superior mesenteric artery, seemed to all of us who saw the case to preclude operative interference.

The patient remained much *in statu quo* until January 25th, four days after admission, then hiccup developed, and she died on January 26th, the temperature going up very rapidly just before death from 95° to 102.8° on the morning of the 26th.

* In a recent number of the BRITISH MEDICAL JOURNAL (February 17th, 1906, p. 366) there is a valuable paper by Mr. George Heaton on *Some Acute Abdominal Disorders which resemble in their Symptoms Acute Intestinal Obstruction*. In that paper Mr. Heaton records a case of gangrene of the intestine due to blocking of the superior mesenteric artery and makes the following remarks on the condition: "It is an uncommon condition, but some 214 cases have been collected by Drs. Jackson, Porter, and Quinby (*Journal of the American Medical Association*, 1904, 1 and 2). In the large majority of these cases there has been some endocarditis found as the cause of the embolus. When there is thrombosis of the mesenteric vein, it has usually followed some enteritis of the intestine or intra-abdominal suppuration, but is in some cases secondary to a portal cirrhosis. The attack is usually quite sudden. The patient, who is generally a male, is seized with acute abdominal pain in the umbilical region. With the pain there is much collapse and sickness. The temperature is, as a rule, subnormal throughout the course of the disease, and extreme restlessness is often a marked symptom. The bowels are in some cases opened repeatedly, and in these cases large quantities of blood may be passed, but in the majority of cases constipation is absolute. The abdomen soon becomes distended and tympanitic, but later on evidence of free fluid may make its appearance. The pain throughout is colicky in character, and comes on in paroxysms, as in intestinal obstruction. The patient's general condition becomes rapidly worse; his pulse, frequent from the first, becomes more and more rapid, and towards the close may be uncountable. The prognosis is extremely bad, and the fatal issue in the acute cases is not often delayed beyond the eighth day. The diagnosis from acute intestinal obstruction is extremely difficult. The large majority of the recorded cases have been operated upon as such, and the condition only recognized at the operation or at the post-mortem examination. The points which in a doubtful case would make one suspicious of an embolus or thrombosis being the cause of the symptoms are the presence of cardiac mischief, or some source for an embolus, a previous history of embolism in other parts of the body, copious hæmorrhage from the bowel, and a very low temperature, with extreme collapse and colic. An abdominal section, however, even if undertaken in mistake, gives the patient his only chance in this as in obstruction. The bowel supplied by the thrombosed vessel almost invariably becomes gangrenous, and a resection of the loop supplied by the thrombosed vessel gives the patient his only chance, though it is a very remote one."

Post-mortem examination showed general peritonitis due to ulceration and rupture of the appendix vermiformis. The heart presented the conditions which we had diagnosed, a marked stenosis of the mitral valve and a few small vegetations indicative of recent endocarditis. The left auricular appendix was filled with an old adherent thrombus. All the conditions for the production of an embolism were therefore present, but there was no embolism, as we had supposed.

It not infrequently happens, in consequence of the presence of old disease or of a history of symptoms which in reality have nothing to do with the present condition of the patient, that one is thrown off one's guard, and led to form an erroneous diagnosis. This is a point that has to be remembered in practice. Let me give you another illustration:

Case of Acute Peritonitis due to Ulceration and Rupture of the Appendix Vermiformis, in which the previous history and symptoms (more especially vomiting of blood) seemed to me to indicate the rupture of a duodenal ulcer.

About a month ago I saw in consultation a man, aged 50, who was suffering from acute peritonitis. He had been quite well on the last day of the year; on the evening of that day, somewhat late at night, he had taken a hearty supper. He went out on the morning of the first day of the year to see his friends and make calls; he came back to lunch at midday saying that he was not well; he complained of pain in the abdomen and vomited. For the first thirty-six hours of the attack the abdominal symptoms were not severe. I saw him on the afternoon of January 3rd, two days after the symptoms had developed. He was then suffering from acute general peritonitis; the abdominal symptoms had become greatly aggravated a few hours before my visit; his pulse was quick; he was vomiting blood—coffee grounds and streaks of blood mixed with mucus.

On inquiring into the history I found that for some months before the development of this acute attack he had occasionally suffered from abdominal pain and flatulence, that the abdominal pain occurred some hours after eating, and that it was (usually) relieved by eating. There had never been vomiting.

Now, here again I was thrown off my guard, partly by this history of previous digestive trouble, but chiefly by the fact that the patient was vomiting blood. The hæmatemesis seemed to suggest that the most probable cause for the acute peritonitis was the rupture of an ulcer of the stomach or duodenum. The facts that the pain used to come on some hours after eating, that it was usually relieved by eating, and that there had been no vomiting (until the acute symptoms developed), seemed to me to indicate a duodenal rather than a gastric ulcer. The vomiting of blood seemed to me to be strongly in favour of a gastric or a duodenal ulcer. The patient's medical attendant, however, was inclined to think that it was a case of appendicitis. We both agreed that immediate operation was necessary. A surgeon was immediately telephoned for. He agreed with the medical attendant that the cause of the peritonitis was probably appendicitis; he opened the abdomen, and found a ruptured appendix.

In subsequently discussing the subject with Mr. Stiles, he informed me that in the later stages of cases of appendicitis, in which septic peritonitis is developed, vomiting of blood is of frequent occurrence. Though the occurrence of hæmatemesis in the later stages of acute appendicitis with peritonitis is mentioned, it is not sufficiently emphasized in the textbooks which I have consulted. I am anxious you should be acquainted with it. It is a misleading symptom. I am anxious that you should profit by my inexperience and by my mistake, so that if you should, after entering practice, happen to meet with a case of acute peritonitis with hæmatemesis, you should not too hastily conclude that the peritonitis is due to the rupture of a gastric or duodenal ulcer. It may be due to a ruptured appendix.

SIMPLE OR PERFORATING ULCER OF THE DUODENUM.

In the remarks which I have just made I have referred to duodenal ulceration. I wish now to say a few words with regard to that condition.

You all know that ulceration of the stomach (simple or perforating ulcer of the stomach) is a very common condition. It is much more common in women than in men, and usually occurs in young women, anæmic (chlorotic) women; it is the most common cause of vomiting of blood (hæmatemesis), and is an important cause of acute perforative peritonitis—the "acute abdomen," as Mr. Battle terms it. A similar form of ulceration occurs in the duodenum. "Simple" or "perforating" ulcer of the duodenum is much less frequent than simple or perforating ulcer of the stomach, but is probably much more common than is usually sup-

posed. It differs in some of its features very materially from the same form of ulceration in the stomach. As I have just said, it is less frequent; it is much more common in men than in women: it occurs at a somewhat later age than simple ulcer of the stomach in women; simple ulceration of the duodenum is most frequently developed between the ages of 30 and 45, whereas simple ulceration of the stomach is most frequently developed in young women between the ages of 16 and 30. Of course, simple ulceration of the stomach may be met with at any age. We recently had a case in hospital in which the patient was a man aged 70: in that case the diagnosis was verified by *post-mortem* examination. So, too, simple ulceration of the duodenum may occur at any age, that is, in young people. I am speaking of the relative ages at which the two conditions are usually developed. I repeat that simple ulceration of the duodenum occurs much more frequently in men than in women, and more frequently in middle-aged than in young men.

In my experience, simple ulceration of the duodenum is a more serious condition than simple perforating ulceration of the stomach. It is more difficult, I think, to heal, perhaps because the healing of the ulcer is prevented by the irritation of the bile; but in this connexion—in opposition to this theory—it is important to remember that simple ulceration of the duodenum is far more frequent in the upper than in the lower end of the duodenum.

In my experience, perforating ulceration of the duodenum far more frequently causes fatal haemorrhage than perforating ulceration of the stomach. This is a most important practical point which I wish particularly to emphasize. In simple perforating ulcer of the stomach profuse and recurring haemorrhages are extremely common, but fatal haemorrhage is rare. I have seen and recorded many cases in which very large bleedings occurred from the stomach: in some of these cases the patients were completely blanched by the bleeding; in some of them repeated large bleedings occurred; in all of them, almost without exception, recovery took place. It is extremely rare for a young woman to die from haematemesis of this kind. This fact should, I think, make one hesitate in too hastily advising operative interference for gastric haemorrhage in young women.* It is a very different state of matters when you have to deal with haemorrhage due to simple ulceration of the duodenum; then the haemorrhage is very frequently fatal. It so happens that during the last two months I have seen 4 cases of simple ulceration of the duodenum, 3 of which have died from haemorrhage. That is a remarkable and lamentable experience which has profoundly impressed me. Let me briefly describe the leading features of these cases:

Case I.—The first patient was a man, 46 years of age. Until two and a half years previously he had enjoyed good health. He then began to suffer from dyspeptic symptoms—pain in the stomach, flatulence, water-brash, constipation, loss of weight. The pain usually occurred two hours or more after eating and was not attended with vomiting; the pain usually lasted for several hours and was generally relieved by the introduction of food into the stomach. Now these facts were suggestive of ulceration of the duodenum.

In cases of gastric ulcer, as I have already told you, the patients are usually young women; they are often anaemic; the pain generally comes on immediately or soon after the introduction of food into the stomach—the food irritates the raw surface and causes the pain; there is almost always vomiting; the vomiting, like the pain, generally comes on soon after the introduction of food—vomiting is Nature's effort to expel the irritating material. In cases of ulceration of the duodenum, the patients are usually men from 30 to 45 years of age; the pain usually occurs at a considerable interval after eating, that is, when the food passes from the stomach into the duodenum. The pain is often relieved by the introduction of food into the stomach—this is a point, I think, of great diagnostic importance; there is usually no vomiting.

Well, this man suffered for six months from these symptoms—pain in the stomach, flatulence, constipation, loss of weight. The pain was often extremely severe. Then one day while he was at work he vomited a large

quantity of blood, fainted, and became blanched; during the next two days he passed a large quantity of blood by the bowel—frequent large tarry stools. For a week he was so ill that he could not be removed to the hospital. Then he was brought to Ward 28. On examining him we found that he was very anaemic and very weak; no hardness or tumour could be detected in the abdomen, which was easily examined because it was collapsed; there was slight tenderness to the right of the middle line in the epigastric and hypochondriac regions.

Two days after his admission to hospital he suddenly complained of feeling faint and became more blanched; there had obviously been another haemorrhage; he did not vomit blood on this occasion, but soon after the attack he passed several large tarry stools. Now we had no difficulty in diagnosing that the case was one of duodenal ulcer—the time at which the pain occurred after taking food, the fact that the pain was relieved by the introduction of food into the stomach, the very severe character of the pain, the absence of any vomiting except on the one occasion when he vomited blood, the age and sex of the patient, and the fact that during the second bleeding the blood was all passed into the intestine and not into the stomach—all pointed to ulceration of the duodenum. After the second bleeding uninterrupted recovery took place, and in the course of six or seven weeks the patient was discharged from the hospital apparently quite well.

After his discharge the patient returned to work—he was the keeper of a public weighing machine—and continued to work for more than a year without any discomfort or abdominal symptoms. Then in June, 1905, the symptoms returned, and he again passed blood by the bowel. From June until October he continued to have attacks of severe abdominal pain, on more than one occasion passed tarry stools, and lost a stone and a half in weight.

On October 18th, 1905, he came to the hospital as an out-patient; he looked thin, pale, and ill; there was some tenderness on pressure in the right side of the epigastrium, but no hardness or tumour. The diagnosis was a recurrence of the duodenal ulceration. I gave him strict injunctions with regard to diet and regulation of the bowels, and prescribed Hewlett's preparation of bismuth and pepsin (it contains bismuth, pepsin, and a little opium). Three weeks later he again came to the infirmary, saying he felt very much better; he had gained 3 lb. in weight, there had been no fresh bleeding, he still occasionally felt pain, but it was much less frequent and severe. He was told to go on as he was doing.

A few weeks after this his doctor telephoned me saying that he was suffering from agonizing pain which failed to yield to any of the remedies he had tried. I advised that the patient should be at once sent to the surgical side of the infirmary in order that an operation should be performed without delay. This was done, but before an opportunity of operating occurred he became suddenly blanched and died. On *post-mortem* examination it was found that the intestine was full of blood, and that there was a simple (perforating) ulcer of the duodenum.

Case II.—About two months ago I saw in consultation a young man, 29 years of age, who that morning had had a very profuse bleeding into the intestine. I learned that this was the second bleeding which had occurred. The patient had been an extremely healthy, active, athletic young man until some six months before the haemorrhage occurred. He then began to suffer from pain in the stomach; the pain appeared some hours after taking food; it was not attended with vomiting; there was no flatulence. A week before I saw him he was suddenly seized while at work in his office with faintness; after getting home he vomited a large quantity of blood and subsequently passed a large quantity of blood by the bowel. He was treated by rectal feeding (for several days), adrenalin chloride by the mouth, and the application of ice to the pit of the epigastrium. Two days before I saw him, small quantities of peptonized milk were given by the mouth. On the morning of the day on which I saw him (a week after the first bleeding), he suddenly became collapsed and passed several large tarry motions. When I saw him he was recovering from this attack; his pulse was much better than it had been a few hours previously. I advised that he should go on with the same treatment (rectal feeding, adrenalin chloride by the mouth, ice to the

* In my *Clinical Studies*, vol. i, pp. 278, 359, 358, and vol. ii, p. 3, I have discussed this question in detail.

epigastrium). His medical attendant agreed with me in thinking that if any further bleeding occurred an operation was advisable. He continued to improve for a week, then he had another attack of collapse, evidently another intestinal haemorrhage. The surgeon who was called in found him too ill to bear an operation; he died soon afterwards from this, the third, bleeding. I have no doubt that in this case the haemorrhage was due to an ulcer of the duodenum.

Case III.—A short time ago I saw in consultation a gentleman, aged 35, who for fifteen years had suffered from recurring attacks of dyspepsia; pain in the stomach, coming on again at a considerable interval after taking food, and lasting some hours, relieved by the introduction of food into the stomach; no vomiting; no flatulence. On several occasions these attacks were attended with tarry motions, and on one occasion with vomiting of blood. These attacks only occurred in the winter, and were apparently due to exposure to cold; the patient's occupation exposed him to cold and wet. He always was better, and free from the attacks during summer and warm weather. I saw him just as he was recovering from a severe attack. There was nothing to be made out on examination. I agreed with his medical attendant in thinking that the case was one of ulcer of the duodenum. We advised that as soon as he was able to travel he should go to Egypt, in order to get into the sun; as I have already said, he always got better in warm weather. Unfortunately that prescription could not be carried out; he went on well for a week, and then suddenly died from very acute haemorrhage.

Now these three cases of duodenal ulcer, in all of which the patients died from haemorrhage, have made a very profound impression on my mind; they show that in cases of duodenal ulcer the risk of death from haemorrhage is very great; they seem to me to suggest that one should advise an operation as soon as one makes a definite diagnosis of duodenal ulcer, provided, of course, that the patient is in a condition to be operated upon. I am convinced that the risk of fatal haemorrhage is very much greater in cases of duodenal ulcer than in cases of gastric ulcer, at all events, gastric ulcer in young chlorotic women. In cases of duodenal ulcer there is also the risk of perforation and fatal peritonitis. I have seen several cases of that sort.

Case IV.—The fourth patient, a gentleman, aged 37, I saw in consultation about a month ago. For four years he had suffered from the same group of symptoms—recurring attacks of severe pain in the epigastric region, the pain occurring a considerable time after eating, and relieved by taking food; no vomiting; no flatulence. On several occasions he had had melaena, and on one or two occasions very profuse vomiting of blood. I diagnosed a duodenal ulcer, and advised that he should be operated on without delay. There was no ulcer in the anterior wall of the duodenum; one cannot get to the posterior surface. A communication was established between the stomach and intestine (gastro-enterostomy). The patient has done well.

In conclusion, let me say that in cases of acute peritonitis due to the perforation of a gastric or duodenal ulcer immediate operation is imperative; every hour that passes, that is put off, diminishes the patient's chances of recovery. I am strongly convinced from personal experience that in cases of this kind the patient's chance of recovery largely depends upon two things in particular—one is early operation and the other is quick operation; every hour that elapses between the occurrence of the perforation and the operation diminishes the risk of recovery, and the quicker the operation (other things being, of course, equal) the greater the chances of recovery. If I were to develop peritonitis in consequence of the rupture of a gastric or duodenal ulcer, I would have an operation at the earliest possible moment, and I would ask the surgeon to perform the operation as quickly as possible.

UNDER the will of the late Mr. Vyell Edward Walker, formerly a famous amateur cricketer, who died on January 3rd, the London Hospital and the Cancer Hospital each receive £2,000, and the Middlesex Hospital, the City of London Hospital for Diseases of the Chest, Victoria Park, the Royal Hospital for Incurables, Putney, the Poplar Hospital for Accidents, the Idiot Asylum, Earlswood, the Hospital for Consumption, Brompton, and the Victoria Hospital for Sick Children, sums of £1,000 each.

A Clinical Lecture ON THE SURGICAL TREATMENT OF GASTROPTOSIS,

WITH AN ACCOUNT OF FIVE CASES.*

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GASTROPTOSIS, or falling of the stomach, may be associated with Glenard's disease, or falling of all the abdominal viscera, with mobility of the right kidney, or it may exist alone. The condition depends mainly on stretching of the suspensory ligaments of the stomach—namely, the gastro-phrenic ligament and gastro-hepatic omentum. It may be further exaggerated by displacement downwards of the liver, to which the latter structure is attached.

Gastroptosis is by no means uncommon, Glenard himself having observed it 400 times among 1,300 patients. The causation is obscure, but its chief basis is probably anatomical. The subjects are usually women the lower orifice of whose thorax is narrow, the dome of the diaphragm low, and the costal margins diverging less obliquely than usual. Tight-lacing, by producing this condition, is undoubtedly an important factor. The muscular parietes of the abdomen are as a rule lax, the muscles ill-developed, and sometimes there is separation of the recti. This condition may have been increased by pregnancy. Loss of fat and consequent diminution of intra-abdominal pressure is a determining cause. In one reported case the omentum was firmly adherent in the pelvis, and in one of my own cases there were adhesions between the anterior wall of the stomach and the parietes. The right kidney is usually movable. In two cases at present under my care this peculiarity exists.

Neurasthenia, often in a high degree, is almost invariably present, and materially adds to the difficulties of treatment. It is not my intention to discuss the relation of neurasthenia to the physical condition; but I have little doubt that in many cases the neurosis is secondary and the result of malnutrition following on the disturbance of the functions of the alimentary canal.

SYMPTOMS.

Dr. Beyea well sums up the symptoms when he says: "The picture is that of chronic severe atonic dyspepsia, with neurasthenia and increasing emaciation."

The patient complains of severe chronic dyspepsia of long standing, with eructations, sense of fullness and weight at the epigastrium and pain after food. In the more severe cases she is unable to take meat or even solid food; there is vomiting, either slight or only of mucus, in attacks at infrequent intervals, and, in the worst cases where considerable dilatation of the stomach has occurred, the vomiting is frequent and copious. Severe lumbar pains may be complained of. In two of the cases quoted below there was a tender spot on pressure over the epigastrium, and in two a history of haematemesis. In my first case the functions of the stomach were so much altered that free hydrochloric acid was absent from a test meal.

Physical examination shows displacement downwards of the stomach, the lesser curvature being situated in the neighbourhood of the umbilicus, just above it, or even well below it. The pancreas can be felt above the lesser curvature of the stomach, a condition on which Bertrand Dawson¹ has laid stress. The stomach is often dilated as well as displaced, and splashing may be obtained. Lavage in one of my cases proved distinct gastrectasis to exist.

The coincidence of mobility of the kidney may raise a question as to the part respectively played by the stomach and kidney in the production of the symptoms. The seat of the pain and the character of the symptoms will usually define which organ is mainly responsible. Reported cases, however, show that nephropexy has been first performed without relief to symptoms; on the other hand, Dr. F. J. Smith informs me that in two cases under his care, believed to be suffering from gastroptosis, the

* Part of a clinical lecture delivered at the London Hospital.