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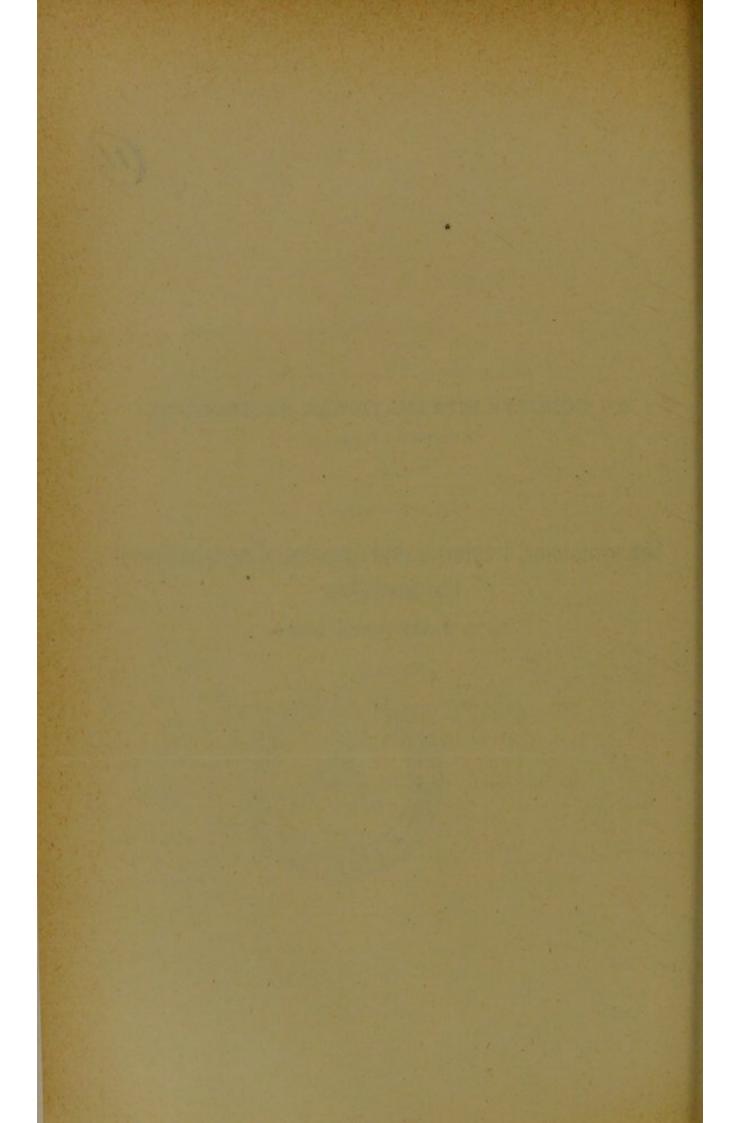
SECTION IN - CHIRCRGIE

The symptoms, treatment and sequelae of non-malignant duodenal ulcer

Par M. D'ARCY POWER, Londres

(SEPARATA)







The symptoms, treatment and sequelae of non-malignant duodenal ulcer

Par M. D'ARCY POWER, Londres.

The symptoms, treatment and sequelae of non-malignant ulcer of the duodenum have hardly received the attention they deserve owing to their frequency and severity. Until about the year 1892 duodenal ulcers were of purely academic interest. They were recognised post mortem and their occurrence was occasionally suspected during life, but no attempt was made to treat them surgically or to differentiate them from gastric ulcer. Increasing familiarity with the surgery of the stomach led naturally to a better acquaintance with abnormal conditions in the duodenum. Surgeons dealt first with duodenal ulcers which had perforated acutely: they were then led to interfere in cases where hæmorrhage was the chief symptom: more recently they have concerned themselves with the results of duodenal constriction due to cicatrisation of ulcers in this part of the alimentary canal.

My position as a hospital surgeon has given me numerous opportunities of seeing and treating various cases of duodenal ulcer and I have thought this a fitting opportunity of communicating what I have learnt about them and the opinions at which I have arrived.

Frequency. Duodenal ulcer occurs in a much smaller proportion of persons than gastric ulcer. Sir E. C. Perry and Dr. L. E. Shaw (1) investigated the records of 17 652 post mortem examina-

t's Guy's Hospital Reports, 187, p. 171.

tions made at Guy's Hospital, and amongst these were seventy cases of ulcer of the duodenum either open or healed. It appears, therefore, that duodenal ulcer occurs in 0,4 per cent of persons dying from all causes as against gastric ulcer which is observed either open or cicatrised in about five per cent of all autopsies. It is further worthy of remark that ulcer of the duodenum is three times as frequent in males as in females, whereas gastric ulcer is twice as common in females as in males. It is interesting, too, to note that of the seventy cases of duodenal ulcer found post mortem, nine were fatal by hæmorrhage, eight by perforation and three as the result of cicatrisation either of the bowel or common bileduct. Hæmorrhage or perforation is equally likely to cause death in those affected with duodenal ulceration, but only in about thirteen per cent of the cases, whilst cicatrisation and constriction to a dangerous extent takes place in about eleven per cent of the cured cases.

Situation. Sir Edwin Perry and Dr. Shaw (4) state that in 149 cases of duodenal ulcer collected from various sources 123 were situated in the first part of the duodenum, 16 were in the second part and only two were in the third part. The ulcers were multiple and were scattered throughout the whole length of the duodenum in eight cases. Duodenal ulcers are often associated with chronic gastric ulcers, and in many cases the ulcer is situated so near the pylorus as to make it appear during an operation that the perforation has taken place in the stomach.

Causes. Nothing is known with certainty as to the causes of ulcer of the duodenum. It is often associated with ulcer of the stomach and the causes which produce gastric ulcer probably play a part in causing ulcer of the duodenum. The duodenum, too, is liable to the same inflammatory conditions as other parts of the alimentary canal. Tuberculous and typhoid ulceration are therefore not uncommon. There is no doubt that general septic and pyæmic conditions may be associated with duodenal ulcer and to these causes, perhaps is to be assigned the duodenal ulceration found in a certain proportion of patients who have been burnt especially when there has been much suppuration. The experiments made by Dr. William Hunter (2) point in the same direction, for he showed that the subcutaneous injection of toluene-diamine into dogs was followed

⁽¹⁾ Loc. cit.

^(*) Pathological Society's Transactions, vol. 41, p. 205.

by marked duodenal ulceration, the drug being destructive to the red blood corpuscles and being eliminated by the liver. There seems to be an ill-defined relation between duodenal ulceration and some cases of interstitial and tubal nephritis, whilst the stress of anthrax infection may fall upon this part of the alimentary canal.

Symptoms. I do not want to weary you with lengthy and uninteresting details of cases of duodenal ulcer so that I will detail the symptoms by a composite picture, premising that every case of duodenal ulcer which has come under my notice with acute symptoms of perforation or hæmorrhage has been in a man. Women suffer in a smaller proportion of cases and in them I have repeatedly performed a gastro-jejunostomy on account of adhesions and constriction owing to the cicatrisation of an ulcer.

(A) Perforating duodenal ulcer

Perforation. The subject of a perforating duodenal ulcer is usually a man in the prime of life who assures you that he has either never suffered from any dyspeptic symptoms up to the moment of his sudden illness or has had so little indigestion that he has taken no account of it. Without warning he is suddenly seized with a stomach-ache of such severity that he becomes collapsed at once and sends immediately for assistance. He may vomit, but from the onset he passes neither flatus nor faeces. Examination within an hour or two of the attack shows him lying on his back, afraid to move, his breathing shallow and rapid, his pulse small, regular and quick, but not nearly so much accelerated as his respirations. He looks pinched and haggard but his temperature is normal. The patient cannot localise the pain but complains that it is worse along the upper half and down the right side of the abdomen. The abdomen is not distended and is not motionless, though it moves less freely during respiration than it should do. At first it is held rigid and the muscles on the right side are somewhat more tonically contracted than those on the left. It is everywhere tender and tympanitic. The area of liver dulness may not be altered and there is sometimes a point of maximum tenderness in the right hypocondrium. If no operation be performed, the pain becomes less acute and more generalised than it was at first. The patient rallies from the initial shock and may fall asleep: he loses his pinched appearance, and though the abdomen is still rigid it moves slightly during respiration. If unfortun

ately the patient is still left untreated, either because an incorrect diagnosis has been made or because he does not apply for relief, the pain again becomes urgent, but is now felt in the right iliac fossa which becomes especially tender, full and motionless. The patient shows the ordinary signs of peritonitis, he again becomes collapsed, his pulse quickens, his temperature rises and he dies usually with a diagnosis of appendicitis.

The errors in diagnosis are quite excusable in those who have not already had to treat a case of perforated duodenal ulcer and in those who are only called in to see a patient in the later stages. The initial shock caused by the sudden perforation is generally sufficient to indicate that some grave catastrophe has occurred and has overwhelmed the peritoneum. The symptoms are not quite like those of a perforated gastric ulcer, because the duodenal contents are more digested and less acid than those of the stomach and there is consequently less irritation. The symptoms subside therefore for a time and the fluid poured out by the duodenum slowly accumulates in the iliac fossae and more in the right than in the left. The onset of peritonitis demands an examination of the abdomen and as the patient now locates his symptoms to the lower part of his abdomen it is easy to treat the case as one of appendicitis. Here are two cases illustrating these mistakes: -

A porter aged 41 was admitted into St. Bartholomew's Hospital saying that he was at work and quite well until 11 a. m. when he was suddenly attacked with pain in his epigastrium. The pain continued and he vomited several times before coming to the hospital. He had passed no flatus since the pain began; his bowels had been well open on the previous day. He was a temperate man and was sure that he had never suffered from indigestion. At 2 p. m. the patient was reported to be a well-nourished man in obvious pain. His tongue was clean and moist: his respirations were very shallow and 60 in the minute: his pulse was of fair volume and tension, regular and 100 a minute. In the chest the percussion note was impaired at the right base and the entry of air at the base of the right lung was weaker than at the left. No additional sounds were heard by auscultation. The abdominal pain was not localised but the patient complained of it chiefly over the upper half and down the right side. The abdomen was not distended but moved very little during respiration. The movement, however, was equal all over, though the abdomen was held somewhat rigid. It was tender and tympanitic everywhere except that the liver dulness was present. Nothing abnormal could be felt. It was impossible at this time to make any definite diagnosis and the surgeon left directions that the patient should be carefully watched on the assumption that he was suffer ring from pulmonary rather than abdominal trouble and most likely from pneumonia. There was no definit change at 6 p. nz, an enema saponis had been retained, but in spite of fomentations the pain was unrelieved. The temperature was 99°,8

F., the pulse was 120, the respirations 60. At 12 midnight there was still no material change in the condition of the patient, whose temperature was 99°,8 F., pulse 120, respirations 60-70. A little liquid had been vomited. At 1 a. m. the abdomen was distended and the patient was slightly collapsed with a pulse of 144 almost running. At 2 a. m. the patient was still more collapsed. I then saw him for the first time and at once determined to open his abdomen. Liquid escaped as soon as the peritoneal cavity was opened and on drawing the stomach into the wound a hole was found in the duodenum large enough to admit a full sized probe; there was a considerable deposit of lymph all round the margins of the aperture An attempt was made to close the opening with Lembert's sutures, but the operation was very difficult, owing partly to the awkward position of the ulcer and partly to the rotten state of the tissues in the neighbourhood. Four sutures were passed and it seemed as though the opening had been successfully closed. During the suturing large quantities of a thin liquid kept welling up from the perforation until the last suture had been inserted. The peritoneal cavity was then cleansed and afterwards closed The patient bore the operation badly and his pulse at the end was hardly perceptible He died at 7, 30 a. m.

A post mortem examination at three p. m. on the same day showed that the peritoneum was acutely inflamed, its endothelial aspect being covered with a layer of fibrino-purulent lymph. There were collections of purulent matter at the bottom of the pouch of Douglas, in the lumbar region and in the right subphrenic space which was almost completely shut off from the rest of the peritoneal cavity. The beginning of the duodenum at the upper and posterior part was the seat of a conical ulcer which measured half an inch in diameter. It had sharply cut edges but there was no infiltration at the margin The floor of the ulcer had perforated but the sutures were not accurately applied as water and intestinal contents easily passed through.

The particular interest of this case lay in the fact that the localised peritonitis must have lasted a much longer time than, the sudden onset of the symptoms would have led one to suppose, whilst the symptoms when they appeared were so obscure as to make it seem that the patient was suffering from pneumonia rather than from peritonitis. It is farther interesting because it is a record of the course taken by a case of perforated duodenal ulcer which though carefully watched was practically untreated.

The following case illustrates how easy it is to mistake a perforated duodenal ulcer for an acute attack of appendicitis:

A bookstall keeper, aged 26, was admitted into St. Bartholomew's Hospital under my care suffering from abdominal pain and sickness. He said that he had suffered from other attacks of similar pain, but this was much more severe, and had begun suddenly at 7 p. m. on the previous day. The abdomen when I saw him at 10 a. m. the next morning was hard, tense and painful. The temperature was 97°,2 F. and the pulse 112. He localised his pain over the right iliac region which was tender and full, the rest of the abdomen moving during respiration. I thought that he was suffering from acute appendicitis which had ended in perforation and

I therefore opened the abdomen in the right iliac region and found the appendix normal. Gas issued from the abdominal cavity as soon as the peritoneum was incised and there was gush of alkaline fluid which did not smell but was clearly bilestained. The end of the ileum was inflamed in patches which seemed to correspond with Pever's patches. The clear alkaline fluid which escaped from the peritonnal cavity told me that the perforation had occurred much higher up the alimentary canal. I therefore plugged the iliac wound and opened the abdomen in the middle line above the umbilicus. The stomach was found to be normal, but a perforation was discovered in the duodenum and on its anterior surface, measuring about an eight of an inch across. The hole was closed with two layers of Lembert's sutures. Drainage tubes were inserted at the upper and lower ends of the median incision as well as in the iliac wound. The patient bore the operation well, making a steady and uneventful recovery. He left the hospital on the 48th day after the operation. It is now three years since he was under my care. I have seen him at intervals of a year and he always says that he is a healthy man with unimpaired digestion and able to follow his original occupation.

Hæmorrhage. The hæmorrhage in some cases of duodenal ulcer is a characteristic symptom. It is sudden, painless and very considerable in quantity. It seems to be due to erosion of the large arterial trunks which lie outside the duodenum — the superior pancreatico-duodenal artery being the most commonly affected, though the gastro-duodenal, the pyloric, the gastric and the pancreatica magna as well as the superior pancreatico-duodenal vein have been found ulcerated. The hæmorrhage may be so severe as to be fatal at once, more often the patient is blanched or he may become faint without knowing the cause. This may happen on more than one occasion and as the hæmorrhage is concealed the cause is only recognised by the subsequent passage of large tarry motions. In other cases the patient may have hæmatemesis instead of melæna or both may be present.

Here is an example of such a case:

A man aged 31, a printer's labourer, was admitted into St. Bartholomew's Hospital under my care on May 18th. 1904, and was discharged on June 17th. The patient had been treated in Luke ward by my colleague dr. Herringham since April 22nd, for pain in the lower part of his abdomen and vomiting. The pain had first been felt six months previously and had gradually become worse until on getting out of bed one morning nine weeks since he had felt faint and vomited «a quart of dark blood all in lumps.» He was at home for five weeks after this and was then in the infirmary for three weeks. On April-19th, he retched after food and on April 20th, he vomited «a pint of blood» after food. There was no hystory of melæna and the patient said that he had never had any illness of importance. Two years ago he was in Australia and Canada. He drinks one or two pints of beer a day and smokes a little. He has never had venereal disease: is married and has two children living: the last two were stillborn. His father died of acute alcoholism at the age of 48, his mother in childbed.

The patient is a pale man with a temperature of 97° F.: pulse 92, soft: and respirations 28. His abdomen moves evenly during respiration and there is a point of maximum tenderness in each iliac fossa. There is also a tender spot one inch to the right of the ambilious, but this point varies somewhat in position.

So long as the patient was in the hospital he had neither pain, vomiting nor melena; but in view of his history, of the large quantity of blood he had brought up and the situation of the pain I had no difficulty in making a diagnosis of duodenal ulcer. I performed a posterior gastro jejunostomy upon him, with the concurrence of Dr. Herringham, on 19th. May. The patient made an absolutely uneventful recovery; the wound was healed and the stitches were removed on May 28th, and when he was discharged to the Convalescent Home at Swanley on June 17th, he had suffered neither indigestion, stomach ache or pain since the day after the operation.

The attacks of pain and vomiting recommenced about five weeks after the patient left the hospital and a fortnight after he had been at home and had returned to his ordinary diet. When he reported himself to me four months later, they were often as severe as they used to be before the operation. The attacks bear no relation to the taking of food which he says he fimits to fish, stews milk and a very little meat, though his wife affirms that the denies himself nothing and she is sure he is the worse for it.

The patient was readmitted to the Hospital on January 6th. 1905 on account of sickness and pain after food. He vomited from time to time, vomit containing clots of blood, but the patient said that he did not feel ill in himself. He was placed on low diet, no solids, and was given a little essence. On January 10th, he suddenly became collapsed in the morning as he was lying in bed. He felt faint and became very cold. A few minutes later he vomited a little dark blood. His bowels were open involuntarily whilst he was collapsed and the motion seemed to contain a little blood. At 3.30 on the same day he vomited twenty ounces of dark coloured blood. At 4 a. m. on January 12th, he again became collapsed without any warning and at 8, 30 a. m. on the same day he vomited twenty-five ounces of blood in clots, but he had no melæna on this or any subsequent day. He was collapsed for a third time on January 24th, the attack being quite sudden and his pulse becoming imperceptible, but he had neither hæmatemesis or melæna afterwards and was without further bad symptoms during the remainder of his stay in the Hospital. He was discharged on March 8th, 1905 and has not since been heard of.

Diagnosis. The diagnosis of a perforated duodenal ulcer should be easy but in practice it is often a matter of great difficulty as is shown by the cases I have just quoted. The patient is suffering acute abdominal pain and the attack began suddenly. The history of previous good health, the fact that the abdomen is moving during respiration, the absence of any great amount of rigidity in the abdominal muscles or of any point of maximum tenderness, are all misleading and make it difficult for the surgeon to believe that the patient is suffering from such a dangerous condition as a perforated ulcer of the duodenum. He may think of a perforating or leaking gastric ulcer, but the symptoms are much

less characteristic than he is accustomed to find in this condition and it is not until he has seen one or two similar cases that the occurrence of a perforated duodenal ulcer is called to mind. The diagnosis, therefore, is too often left in abeyance in the hope that a few hours' delay will render the signs and symptoms more definite. Such advice is likely to prove fatal for instead of making the diagnosis clearer time only renders it more obscure. The slight clues which could be picked up shortly after the onset when the peritoneum was only affected at the point of perforation are soon masked by the general peritonitis produced by the diffusion of the duodenal contents throughout the abdominal cavity. Delay thus allows the extravasated fluid to gain access to the innermost recesses of the peritoneal folds so that a subphrenic, pelvic or iliac abscess may still further complicate a condition which is well-nigh desperate from the beginning.

When I am called to a patient who has been seized suddenly with intense abdominal pain, without much history of previous indigestion, I ask that he shall not be given morphia and that his pulse shall be counted carefully and accurately recorded by the same person every half hour. If on my arrival the pulse-rate has increased although the patient has been kept at rest and free from disturbing influences, I have no hesitation in advising an immediate exploratory operation, even though the objective signs be very slight.

The escape of gas and liquid as soon as the abdomen is opened prove that a perforation has occurred and the first thought of a surgeon will then be that he is dealing with a ruptured gastric ulcer. The character of the fluid often gives the first indication as to the seat of the perforation. When it comes from the duodenum it is limpid or bile-stained, free from smell, alkaline and thus wholly different from the extravasated contents of the stomach which often contain undigested portions of food. The fluid from the duodenum is the succus entericus and is, I suppose, the secretion of Brunner's glands. It is very abundant and comes welling up from the perforated intestine in quite a characteristic manner. The perforation is usually small, close to the pylorus and often rather at the back of the duodenum so that is very awkwardly placed for suture. It is however possible to close it completely as in the case which I have already quoted.

If the patient be left without operation, the diagnosis, as I have said, becomes still more obscure and my experience tells me that

a perforated duodenal ulcer may be mistaken for pneumonia, for appendicitis or for suppurative peritonitis from any other cause.

(B) Non-perforating ulcer

The second group of cases where there is a duodenal ulcer without perforation is even more interesting than the perforating variety which has just been considered. The diagnosis is more difficult and the sequelæ demanding surgical treatment are no less urgent though they may be more remote. As may be gathered from what has been said of perforated ulcers, the signs of a duodenal ulcer may be absent or wholly inconspicuous and in such cases no diagnosis is possible. In some cases the patient complains of a continued pain in the abdomen which he can neither localise nor account for. He keeps his bed for a few days and then feeling better goes about again saying that he has had a bad bilious attack. If he is nervous about himself or should the pain have been severe, he seeks medical advice and is treated for gallstones, renal colic or appendicitis. But the exact nature of his illness is probably not recognised unless he vomits a considerable quantity of blood or has a sharp attack of melæna. Even then the case is thought to be one of gastric ulcer, unless an abdominal section is performed and the stomach is found to be healthy.

I do not know how a diagnosis can be established in the present state of our knowledge, but it is of no practical importance, for in the majority of cases the patient recovers from the attack unaided by art or if the bleeding be sufficient to need an exploration the surgeon performs a gastro-jejunostomy, whether the ulcer be situated in the stomach or in the duodenum. I have tried to recognise the condition by the character of the bleeding, by the time of the occurrence of pain after taking food, by the character of the material vomited and in many other ways, but always without success, for what is true and seems a valuable sign in one patient is worthless in another. I believe, therefore, that there is no pathognomonic sign of a non-perforating ulcer of the duodenum whilst the ulceration is in progress.

Constriction of the duodenum.—But if a non-perforating duodenal ulcer offers very few signs by which its presence may be detected, it may have sequelæ of the gravest character. The ulcer is usually single, and is situated in the first part of the duodenum, but like a gastric ulcer it is very chronic and may cause

extensive inflammation in the submucous and muscular coats of the intestine. The inflammatory changes may extend to the serous coat and to the under surface of the liver. In process of time the ulcer heals and by the subsequent cicatrisation of the inflammatory products the duodenum is either narrowed or it is constricted by the surrounding adhesions. The adhesions may not only affect the duodenum but they may also involve the liver, the gall bladder, the pancreas and such large blood vessels as the abdominal aorta, the hepatic artery and the portal vein. Such a patient presents himself as a man between 40 and 60, looking older than his years. Thin and haggard he tells you that he is a martyr to indigestion and that for months past he has suffered atrocious pain in his stomach which is relieved by vomiting. He has dieted himself in every possible manner, he has made all kinds of local applications to his stomach, he has visited all sorts of watering places and he has gone in vain from one physician to another seeking a cure. Examination shows him to be a mere bag of bones, badly constipated with cold extremities and a listless, dejected aspect. His abdomen is loose, the subcutaneous veins may be enlarged and there is visible peristalsis from left to right in the epigastric region. Palpation and percussion tell of a greatly dilated stomach and a tumour may sometimes be felt in the neighbourhood of the pylorus. For a moment you think of cancer of the pylorus or gall-bladder and you question the patient a little more closely. He is sure that he has been suffering for years, for so long, in fact, that he hardly recollects the beginning of his trouble. A few well-directed enquiries may elicit that 25 or 30 years ago, when he was a young man, he once or twice brought up a large quantity of blood without serious pain or discomfort or that he had an illness which no one seemed to know much about. He was treated for gallstones or for appendicitis or simply for «liver.» The attack was abdominal, was painful and kept him in bed, but the exact details have long since passed from his mind and for some years he was as healthy a man as ever.

This is a case of duodenal obstruction resulting from cicatrisation of an old ulcer, the irritation of which caused inflammatory thickening of the surrounding parts or of the duodenal walls. How many patients have been allowed to die of such a condition in the belief that they had malignant disease of the stomach no one can tell, but every pathological Museum contains several specimens of simple duodenal constriction. Here is such a case in detail:

A man, aged 69, was admitted into the hospital on August 15th. 1902 with a history that he had suffered severely from dyspepsia for the past five years and that for the last two years he had pain after his food and vomiting. During the six or seven months preceding his admission to the hospital there had been occasional streaks of blood in the material vomited. The attacks of vomiting usually took place once in two days and he would then bring up as much as two pints at a time. For the last two months he had rapidly lost flesh, It was noted on admission that he was a thin and wasted man with a flaccid abdomen, which moved freely during respiration. The stomach was greatly dilated as the lesser curvature lay about three inches below the costal margin and the viscus occupied the greater part of the epigastric, all the umbilical, a great part of the hypogastric and some of the iliac regions of the abdomen. A succussion splash was very distinct and a hard rounded tumour about the size of a Tangerine orange could be felt in the middle line, sometimes above and sometimes below the umbilicus. The liver was not enlarged. A test meal showed the presence of free hydrochloric, lactic and butyric acids with albumoses."

The patient was kept under observation and was dieted carefully until August 19th, when I performed a gastro-jejunostomy upon him as he was getting worse instead of better and I felt sure that he had a cancer of the stomach. When the peritoneum was opened, the pylorus was found to be thickened but the swelling which had been felt through the abdominal walls was a mass of adhesions surrounding the duodenum and attaching the whole of the first part to the neighbouring tissues.

The patient slept badly after the operation and was repeatedly sick suffering much pain until he left the hospital on September 19th. From the hospital he went to a nursing home at Reading and afterwards to Devonshire where he is still living.

He wrote to me under the date October 28th, 1903 — fourteen months after the operation; «I hardly know where to begin, so presume I had better start from our first interview which was on July 23rd, 1902. Went into Bart's August Ist. Operation performed on me August 19th., left September 19th, at my own request; went direct to a home at Reading: left there November 1st. for Hfracombe direct: bore the journey well. Out in a bathchair November 5th, for an hour and so every day when fine until December 11th, when the chair was dismissed and I walked, I can eat and digest my food. I occasionally take a glass of mild ale - no spirits or wine. I retire to bed about 10 o'clock, previously eating an apple. I rise at 6, make myself a cup of cocoa - half milk - and after washing, smoke a pipe and read till 8.30; then breakfast, - my best meal: - lunch at 10 generally cold meat and soup; tea at 5.0. Dinner or supper at 7.30, seldom get meat then, usually bread and butter, cheese, salad and plenty of onion. Can walk five or six miles without feeling tired; I am out of doors as much as possible». To this satisfactory account of himself he adds that his weight went up gradually from 9 stones four lbs. to 12 stones 6 lbs. and he concludes his letter by saying: «I have not weighed so much for 13 or 14 years but remembering that I am 71 years old I cant expect to keep this weight up although I dont feel my age.

I saw this patient again in the early part of June 1904, twenty-two months after the operation. He was in good health, maintained his weight and was passing through London to visit some grandchildren in Canada. The trip was successful for he writes to me again from Devonshire in October 1904, "I have just returned

from a visit to the North West Territory where I had to rough it considerably. I have been travelling day and night since October 2nd. and do not feel the least ill-effects. I hear, 1906, that he is still in good health.

The general symptoms of duodenal obstruction may be gathered from what has already been said. There is usually pain of the nature of dyspepsia felt an hour or two after taking food. The pain depends more upon the quantity of food taken than upon the quality. It is relieved by vomiting and the severity of the vomiting increases with the length of time the symptoms have lasted. It is usually copious and often painful. It is not a mere emptying of the stomach, for, when the patient has vomited a large quantity, he will in an hour vomit as much again, although he has taken nothing in the interval. The attacks of vomiting are often worse at night than in the daytime. This seems to be particularly the case with business men, perhaps because they make a light breakfast, have but little lunch and make their chief meal in the evening. The attacks of vomiting therefore, like the pain, bear a definite relation to the quantity, rather than to the quality of the food taken. The quantity of fluid vomited and the small amount of food digested and absorbed lead to very obstinate constipation. It is the relief of this symptom which is hailed with the greatest joy by a patient who has been cured by a gastro-jejunostomy and the regularity of the bowels is one of the best proofs to the surgeon that his anastomosis is working successfully many months after the operation. The same loss of absorptive power leads to diminution in weight, to a harsh and dry skin, to a brittleness of the nails and to loss of the subcutaneous fat; in other words, the patient shows many of the signs of chronic starvation.

A systematic physical examination of the abdomen should be made in every case where there is reason to think that the patient is suffering from duodenal obstruction. The patient should lie flat on his back on a couch, with the knees bent and the shoulders slightly raised by a pillow. A good light should be allowed to fall upon the abdomen and the surgeon should at first merely watch the abdominal movements without touching the patient. In thin people and in a case of marked duodenal obstruction the outline of the dilated stomach can often be seen with slow waves of peristalsis passing across the upper part of the abdominal wall from left to right. These waves in themselves are characteristic of some obstruction to the outflow of the gastric contents and the

more healthy and active the person the more marked are the waves. The surgeon now places his hand flat upon the abdominal wall and his practised touch readily discovers the outlines of the enlarged stomach, even if there be no tumour or nodule perceptible in it. The position of the pylorus is often marked in these cases by a thickened nodule, which is easily mistaken for cancer, especially if, as in the case related above, the patient vomits a little blood from time to time. But the actual position of the pylorus varies greatly when the stomach is dilated and its exact position may not be detected at the first examination. The absence of a tumour does not preclude the diagnosis of duodenal constriction. Percussion is not of any great value in determining the size of the stomach, because a dilated stomach is often associated with a distended colon, and it is difficult to distinguish the note yielded by one from that of the other. Auscultation, too, is of no great service except to elicit the succussion splash. A dilated stomach can be easily distended by pumping in air, a proceeding which, though painless, is unpleasant to the patient, or by the cruder method of administering separately the constituents of a Seidlitz powder. With these guides, there is really no excuse for overlooking a dilated stomach, or for being doubtful as to the existence of such a condition.

The teeth and gums should always be examined, as a matter of routine, to eliminate the possibility of the dyspeptic symptoms and constipation being due to chronic lead poisoning. It is better not to give a definite diagnosis until the bowels have been satisfactorily emptied by means of an enema, repeated if necessary, and it is wise to examine the abdomen more than once, and at different times after meals.

A test meal should be given, and the presence or absence of free hydrochloric acid should be noted. The usual test meal consists of a thick slice of bread and butter with a breakfastcupful of weak tea sweetened, but without milk. The meal is given at breakfast time, and is withdrawn from the stomach an hour later by means of a stomach tube. The material withdrawn is filtered, and the residue is examined microscopically to see how far digestion has proceeded. The filtrate is tested with Günsberg's reagent for free hydrochloric acid. A few drops of the filtrate are mixed in a porcelain dish with an equal quantity of a solution consisting of phloroglucin 2 parts, vanillin 1 part, and absolute alcohol 30 parts. The mixture is evaporated to dryness at a gentle heat,

and, if free hydrochloric acid is present, red crystals will form. As a broad rule, free hydrochloric acid is present in simple duodenal constriction, it is deficient or absent in cancer of the stomach.

Microscopically too the Oppler-Boas bacillus should be absent in cases of non-malignant constriction of the duodenum.

Palliative treatment in cases of duodenal constriction is chiefly directed to relieve the vomiting. This is done by rectal feeding and washing out the stomach, since the indications are clearly to keep the stomach empty, and prevent putrefactive changes. The stomach is washed out with a warm solution of bicarbonate of soda (20 grains to the pint). A soft tube is passed into the stomach and its contents are evacuated, half a pint of the solution is then poured into the tube and is allowed to run out again, and this is repeated several times. In the early stages of duodenal constriction, the stomach needs only be washed out every other day, and this is done most conveniently before breakfast; but in the later stages, when the vomiting is most distressing at night, the stomach should be washed out every evening just before the patient goes to bed, and about four hours after the last meal.

It is obvious that this treatment is only palliative, and it follows, from what has been said already, that patients, who suffer from painful vomiting, with loss of flesh and constipation, should always be examined carefully to find out whether there is any dilatation of the stomach. When the stomach is dilated, and diet, combined with simple remedies, fails to bring about a cure, there should be no hesitation in recommending an exploratory incision with a view to the performance of a posterior gastro-jejunostomy by direct suture.

The operation should not be postponed until the patient has exhausted himself, because early operation is attended with comparatively little risk, and has a twofold advantage. If the patient is found to be suffering from cancer of the stomach, much more radical measures can be pursued in the early stages of the disease, than if delay has allowed the growth to infiltrate all the surrounding tissues. But even if the constriction proves to be due to malignant disease, and removal is impossible, gastro-enterostomy serves excellently as a temporary expedient. It has, too, the advantage over colotomy for the relief of an analogous condition in the rectum, that it leaves no open wound. The patient, therefore, is able to go about his ordinary work, until the progress of

the disease renders him incapable of further effort. But if, on the other hand, the patient is suffering from duodenal constriction, which is not due to cancer, the surgeon can promise him a speedy relief from all the disagreeable symptoms, and a prolongation of his active life for many years in comfort.

