

IV. CLINICAL SIMILARITY OF INFECTION WITH INFLUENZA BACILLI AND WITH OTHER ORGANISMS.

It was hoped that by dividing the respiratory infections into groups, according to the predominance or exclusive presence of one variety of organisms, that some clinical difference in the course and termination of the different cases might be obtained.

The pneumococcus infections may be selected for comparison with the infections with influenza bacilli.

These eight cases of infection with pneumococci gave a history of cough for from one to five weeks before entrance.

In their onset they presented no difference from the cases of acute infection with influenza bacilli, beginning with catarrhal symptoms, with or without fever and prostration. The general symptoms were as severe as in the infections with influenza bacilli.

Examination was negative in three cases. Of the remaining five there were signs of diffuse bronchitis in one, and localized bronchitis in four: In one at the right apex, in a second at the left apex and throughout the left back, in the third at the right apex and the right axilla and in the fourth at the right axilla.

I was able to follow five of the cases to their termination. In three the physical examination was negative. In one the bronchitis was diffuse and in the last case, the râles were confined to the left back and the left apex. No tubercle bacilli could be found in their sputum.

They were well and without cough in from six to twelve weeks (four within eight weeks).

I was unable to follow the five cases of micrococcus catarrhalis infection to their termination. Of 132 cases investigated by Ghon, Pfeiffer and Sederl⁴ this organism was identified in 81 (59%). In only one case was it found alone. In five other cases, though associated with other bacteria, it was in such numbers that it was regarded as the cause of the process.

In the one case in which the organism was in pure culture it was isolated from the bronchial pus after death from diffuse bronchitis. In the remaining five cases, the acute onset of the disease dated from a few weeks to one day before entrance. The respiratory symptoms began with the usual disturbances accompanying an acute infection.

Examination showed diffuse or local bronchitis and bronchopneumonia. In two cases the resolution of the pulmonary solidification took place slowly, not being complete in one until more than a month had elapsed.

The sputum showed enormous numbers of micrococcus catarrhalis with a few pneumococci and pyogenic cocci; influenza bacilli were present in two cases. No tubercle bacilli were found.

In one case the cough lasted for over four months, the remaining patients were discharged, well, within six weeks.

⁴ Ghon, Pfeiffer and Sederl: *Der Mikroococcus Catarrhalis* (R. Pfeiffer) als Krankheitserreger. *Zeit. f. klin. med.* 44, 1902, p. 277.

Concerning the clinical picture, Sederl concludes that it has nothing characteristic. "It is most like infection with influenza bacilli and pneumococci and might, not infrequently, be confused with them, especially the former, since only an exact bacteriological investigation will permit of their differentiation."

(To be continued.)

PERFORATED DUODENAL AND GASTRIC ULCERS: A REPORT OF TWO CASES: OPERATION: RECOVERY.

BY CHARLES L. SCUDDER, M.D., BOSTON.

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CASE I. *Mr. J. McE. Dyspepsia for years. Sudden severe epigastric pain. Operation, closure of a perforated gastric ulcer. Recovery. Subsequent posterior gastro-enterostomy. Recovery.*

A patient of Dr. C. C. Day and Dr. Healey of Newburyport. Thirty-four years old. Single. For some years the patient had had attacks of indigestion and dyspepsia, and two years ago he was in bed with an attack of what was called gastritis. To-day, July 1, 1904, while at work in an iron foundry, he was suddenly, at five o'clock in the afternoon, seized with abdominal pain, referred to the pit of the stomach. He was seen by Dr. Day within one-half hour of this attack of pain, and taken immediately to the hospital. The man had a temperature of 97°, pulse 64, and respiration 32. His facies was not particularly peritonitic. His tongue was moist, his abdomen was hard and board-like, the muscles were rigidly contracted. He was most tender over the seat of the initial pain in the epigastrium, and all the pain was referred to the epigastrium, although he spoke of being sore in the lower half of the abdomen. He had not vomited.

Operation: Abdominal section through the right rectus abdominis muscle. A yellowish, thin, pea-soup material was present in the abdomen, limited largely to the right side. The pylorus presented, with a perforation upon the gastric side on the anterior surface, the size of the lead of a lead pencil. There was no gas in the abdominal cavity. Gas came from the perforation as it was manipulated. The perforation had a punched-out look. There was no fat necrosis visible. The gall bladder was normal in appearance. There was an indurated margin about the perforation half an inch in width. The perforation was closed with three Pagenstecher mattress sutures. A bit of omentum was folded over the line of suture. (See Fig. 1.) A suprapubic incision was made. The abdomen was washed thoroughly with saline solution. Two tubes and a wick were placed in the pelvis through the suprapubic opening and a wick through the laparotomy opening to the line of suture in the stomach. Through-and-through silkworm-gut sutures partially closed both incisions. The patient was in good condition at the end of the operation. He made an uninterrupted and satisfactory recovery. All wicks were removed on about the fifth day.

November, 1904: Operation posterior gastro-enterostomy without a loop. Complete recovery.

May, 1905. The patient at times complains of a burning sensation in the epigastrium which disappears after taking sodium bicarbonate in small doses.

CASE II. *Mr. F. P. D. Indigestion for three years. Sudden epigastric pain. Operation, suture of a perforated duodenal ulcer. After five weeks second operation, posterior gastrojejunostomy. Complete relief of symptoms.*

PERFORATED DUODENAL AND GASTRIC ULCERS — SCUDDER

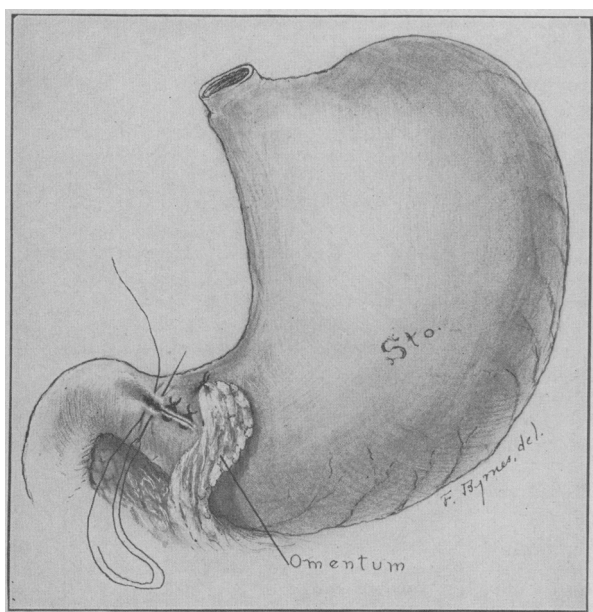


FIG. 1. Case I. Perforating gastric ulcer. Ulcer situated anteriorly at the middle of suture line. Note two mattress sutures placed and tied, introduction of third suture. Note a bit of omentum caught up by one suture. This omental graft was, after tying the third suture, turned over the suture line and held by two stitches.

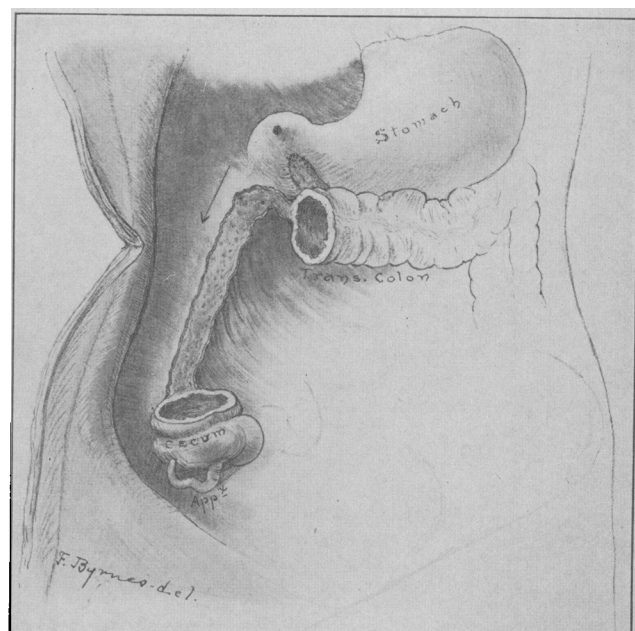


FIG. 2. Case II. Perforating duodenal ulcer. General peritonitis. Operation. Recovery. Gastrojejunostomy subsequently. Recovery. Note seat of perforation indicated by black spot in duodenum. Note that the attachment of the ascending mesocolon facilitates the descent of extravasated duodenal contents toward the appendix as indicated by the arrow.

Twenty-four years old. Massachusetts General Hospital, No. 137,582. This man had had indigestion for three years. Two days ago he was seized suddenly with acute abdominal pain in the epigastrium. Later this pain became general. The pain was at no time localized in the region of the appendix, although he was somewhat sensitive at that spot, more sensitive there than anywhere else in the abdomen. The bowels were constipated. The abdomen was slightly distended and the abdominal muscles were rigid. There was distinct spasm of both recti. There was no mass to be felt and no dullness upon percussion. The temperature was 100.2°, the pulse 80, the white blood count 23,000.

Thinking that this was probably a case of appendicitis with beginning general peritonitis, an incision was made over the appendix. Free gastric contents were found. The appendix was normal. There were spots of necrosis in the omentum up toward the stomach. The bowel was injected only slightly, but considerably distended. Upon following the lead suggested by the necrosis of the omentum and the fibrin, which increased in amount upward toward the stomach, a second incision was made in the median line in the epigastrium and a perforating ulcer in the first part of the duodenum discovered. Gas escaped from the perforation, which was surrounded by fibrin and inflammatory thickening. The perforation was closed by three quilted sutures and the sutures were covered by an omental graft. The abdomen was washed clean with normal salt solution. Gauze wicks were placed at the bottom of the pelvis through the appendix incision. One wick was placed to the right loin through the appendix incision. Another wick was placed through the median incision to the omental graft. The wounds were closed in part with silk-worm-gut sutures.

The patient made an uninterrupted recovery. He was nourished at first by nutrient enemata of peptonized milk, beef juice, egg and salt solution. The average temperature was 99° Fahr., the pulse about 80, the respiration at first 30, later 16 to 20.

About five weeks following this first operation a posterior gastrojejunostomy without a loop was done. This operation was undertaken in order to give the ulcers in the duodenum and stomach, should there be any other than the one which perforated, the best opportunity to heal. Short circuiting the ulcer-bearing area theoretically was indicated. The technique followed was that with clamp and suture. There was no rise of temperature attending the convalescence from this operation. It was interesting to note the thickened condition of the peritoneum and the many adhesions of the bowel to the parietes. Several of these adhesions were divided and the bowel freed.

From this operation the patient made an uninterrupted recovery. Ten months after the first operation the patient reports himself in good health.

I have here recorded two cases of ulcer of the gastro-intestinal canal. One case was an ulcer of the stomach. The other case was an ulcer of the duodenum. Both ulcers perforated, causing acute peritonitis. The first patient was operated upon four hours after the initial sign of perforation. The diagnosis of a perforated gastric ulcer was confirmed. The second patient was operated upon forty-eight hours after perforation. It was thought previous to operation that the patient had an appendicitis and a general peritonitis.

Both the ulcers were closed by sutures. The

peritoneal cavity in each instance was quickly cleansed by salt flushing and wiping. Cigarette and tube drains were used for a few days only in each case. Both patients recovered. In each case at a subsequent time a posterior gastrojejunostomy was done. The anastomosis was made close to the ligament of Treitz. Both cases recovered from the second operation and are well to-day.

In the case of gastric ulcer the diagnosis was based upon these facts: The man was a young adult with a history of indigestion and dyspepsia for several years. He had had one previous attack of what had been called acute gastritis. The present sudden seizure of acute epigastric and abdominal prostrating pain, the tenderness localized most noticeably in the epigastric region, the board-like rigidity of the abdominal muscles following upon the above story of chronic indigestion, made the diagnosis reasonably certain.

In the case of duodenal ulcer it was impossible from the meagre history to make a definite diagnosis when the man was presented for operation. This man was likewise a young adult and had had indigestion for some years. He was attacked suddenly with violent epigastric pain. The pain became general and when he was presented for operation it was found that the most tender spot was over the appendix. The abdomen was distended and the abdominal muscles were rigid. The leucocyte count was 23,000. He had been ill forty-eight hours. A diffuse peritonitis was present. The cause of the peritonitis was thought to be an appendicitis. Operation proved that this diagnosis was wrong.

In considering these two cases of acute perforation of a gastric and a duodenal ulcer certain interesting questions arise. Obviously, it may be possible to distinguish between an acutely perforating duodenal and gastric ulcer if the individual is seen very soon after the initial symptom of peritoneal involvement. The first case reported was evidently gastric because the pain was at the epigastrium at the outset. Had it been duodenal the pain would have been referred further to the right. The duodenal case was forty-eight hours old when seen, and this lapse of time precluded the possibility of distinguishing it from a peritonitis of other origin. Moreover, the history of the gastric case was more definite and clear than was the history of the duodenal case. After peritoneal symptoms become general it is impossible to distinguish between a perforated duodenal and a gastric ulcer in the absence of a clear history.

Before perforation has occurred it is possible to diagnose a duodenal ulcer from a gastric ulcer. Up to within a short time this has not been true, but recently Moynihan¹ of Leeds and Graham² of Rochester have reported relatively large groups of duodenal ulcers. These groups afford valuable data. It is evident that an ulcer of the duodenum is not very uncommon and that it may be

¹ Transactions of the Clinical Society of London, 1902. *Lancet*, London, March 1904.

² Paper read before the Minnesota State Medical Association, June 2, 1904; and the Minnesota Valley Medical Society Dec. 2, 1902.

associated with a gastric ulcer in the same patient. The duodenal ulcer is most commonly found in the first portion of the duodenum. The symptoms are determined by a careful study of the history of the case. That the diagnosis of duodenal ulcer may at times be blind and uncertain is true, especially if it be complicated by gastric pyloric ulcer or some disease of the biliary passages.

The symptoms characteristic of duodenal ulcer are: *Epigastric pain*, relieved by eating and again in evidence some two or three hours later. Moynihan has called this the "hunger pain." This "hunger pain" is relieved by vomiting an acid vomit and by the belching of gas. In duodenal ulcer gas is far more annoying than in cases of gastric ulcer. The type of duodenal ulcer pain is thought by Graham to simulate the pain of gallstone colic far more than does the pain of gastric ulcer. The pain in duodenal ulcer is far more intense than in gastric ulcer. *Vomiting* usually occurs some time after taking food, "delayed vomiting" (Graham). Little food is contained in the vomitus which is often very acid and is followed by temporary relief to the pain and distress. *Blood* may be present in the vomitus, but is more often in the stools (melæna) in duodenal ulcer. "When the duodenum is involved the pain, gas, acidity and vomiting are, as a rule, more intense than if only pyloric ulcer (gastric side) is present, and the type of pain is more apt to approach that of gallstones." (Graham.) From gallstones the distinction is made by the fact that in gallstones the pain is more sudden, radiates more widely, is almost entirely non-dependent upon food, acidity is rarely seen.

In the second reported case a diagnosis of appendicitis with peritonitis was made previous to operation. This is the most common mistake made in the diagnosis of a perforated duodenal ulcer. Moynihan³ has called attention to the reason for this mistake. It is occasioned by the anatomical arrangements of the parts about the duodenum. The transverse colon directs the escaping duodenal fluid to the right loin. The sulcus between the ascending colon and the parietes is so deep that the escaping gastric and duodenal fluids gravitate to the right iliac fossa and the appendix region. (See Fig. 2.) In the case reported the greatest tenderness was at the point where the acute inflammatory process was most recent. The securing of a most careful history from each patient will alone prevent this mistake from being made.

Duodenal ulcers perforate in about half of all cases. The ulcer may perforate slowly, subacutely or acutely. Duodenal ulcers are more apt than gastric ulcers to exist in latent form causing few characteristic symptoms. These facts which Moynihan and Graham emphasize are very evident from the cases analyzed by them. Ulcers perforating anteriorly are less likely to be walled off by adhesions than ulcers perforating posteriorly.

After the abdomen has been opened and the

acutely perforating duodenal ulcer discovered, the ulcer should be sutured. Excision of the ulcer should rarely be undertaken. The peritoneal cavity should be cleansed. This may best be done by thorough flushing of the abdominal cavity with normal salt solution. Personally, I believe that a more thorough cleansing of the abdominal cavity is possible by flushing if a counter-opening is made supra-pubically or in the left loin. After the flushing of the abdomen and the placing of a glass or rubber tube to the pelvis and to the seat of the sutured perforation, the patient should be placed in the semi-sitting position of Fowler.

The question arises in these cases of duodenal ulcer, whether a gastro-enterostomy should be done at the time of the primary operation. If the patient is very ill, if the perforation has occurred a number of hours previous to the operation, it would be unwise to do a gastro-enterostomy. If the patient is in good condition the prolongation of the operation for a few moments that the anastomosis may be made will be safe. In each of these cases of ulcer a gastrojejunostomy was done several weeks after the original operation. In neither case was the pylorus narrowed. There was no obstruction at the pylorus, and yet it seemed wise in view of clinical experience to use the gastro-enterostomy to help the ulcer to heal. In view of the recent experience of Cannon and Blake who find in cats that after a gastro-enterostomy with an open pylorus stomach contents do not tend to go through the new stoma, but through the pylorus as before operation, one may well question whether gastro-enterostomy in the human subject when the pylorus is not obstructed brings about its good result because of drainage or because of some other yet undiscovered reason. Gastro-enterostomy was done in these two cases with the hope that its performance would facilitate the healing of the ulcer-bearing area in stomach and duodenum. The subsequent story of these cases and of similar cases will determine the wisdom of the application of the procedure to the conditions existing.

SOME SUGGESTIONS IN REGARD TO THE DIAGNOSIS OF SEMINAL VESICULITIS.

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By the publication of Dr. Fuller's book in 1894, attention was drawn to the subject of chronic inflammatory lesions of the seminal vesicles and the relation of this condition to chronic posterior urethritis. While fully prepared to admit that Dr. Fuller did a distinct and valuable service to the profession in the publication of this work, it seems possible that the impetus thus given has carried the pendulum somewhat too far in this direction and that the diagnosis of seminal vesiculitis is frequently made upon insufficient grounds. With a view to determining

³ Lancet, Dec. 14, 1901, p. 1656.