
**THE PATHOLOGY, DIAGNOSIS AND TREATMENT OF
PERFORATED GASTRIC ULCER.**

BY

GEORGE E. ARMSTRONG M.D.,

Associate Professor of Clinical Surgery, McGill University ; Surgeon to the
Montreal General Hospital, Montreal.

Reprinted from the Montreal Medical Journal, August, 1900.





THE PATHOLOGY, DIAGNOSIS AND TREATMENT OF PERFORATED GASTRIC ULCER.

BY

GEORGE E. ARMSTRONG. M.D.,

Associate Professor of Clinical Surgery, McGill University; Surgeon to the
Montreal General Hospital, Montreal.

The last quarter of the nineteenth century has seen marvellous advances in the different departments of what may be called intraperitoneal surgery. The most obvious and frequently present lesions, naturally, were the first to be recognized, studied, and systematically dealt with. Thus, appendicitis, cholelithiasis, the pathological lesions of the uterus and its adnexa, and the surgery of the small and large intestine, have now a well determined standing, and they are recognized and treated by symptoms and rules evolved from an increased knowledge of their pathology, and a large clinical experience. Most encouraging features of this work are its gradually extending sphere of usefulness and its lessening death rate.

The surgical treatment of gastric ulcer is of comparatively recent date. Interest in this field of work has been gradually increasing until, at the present time, the various problems arising out of a consideration of the causes and such complications as hæmorrhage, perforation of the stomach wall, subphrenic abscess, pyloric stenosis and adhesions, are being more carefully studied than almost any other. This statement receives confirmation in the fact that the time of the last meeting of the American Surgical Association in Washington was devoted almost exclusively to the reading of papers on the surgery of the stomach, and their discussion. This interest in gastric ulcer is fully justified by its frequency and mortality. Between four and five per cent. of the whole population of Germany suffer at one time or another, from gastric ulcer, writes Ewald, and he estimates the mortality to be from 1.23 to 13 per cent. According to Welch, the mortality is 15 per cent. of all cases, 6.6 per cent. dying from the results of perforation (Tinker), Leube treated 1000 cases in ten years (Tinker). "In the extensive post mortem records collected by Welch, ulcer cicatrized or open, was present in about 5 per cent. of persons dying from all causes. The scars are more frequent than open ulcers" (Osler).

It is my intention in the present papers to confine my remarks strictly to perforations of the stomach wall by simple, non-malignant ulcer. We

have now the reports of a sufficient number of cases to group together and from which to draw valuable lessons.

The first operation for perforated gastric ulcer was performed in 1880. Mikulicz was the operator. The patient died. The first successful closure of a gastric ulcer was in 1892, and Kriege the operator. From 1880 to 1896, a period of sixteen years, there were reported 78 cases. These were collected together by Weir and Foote from English, German and French sources. Keen and Tucker collected 78 cases operated upon during the following two years, 1896 to 1898. An analysis of both groups may be found in the Cartwright lectures delivered by Keen in 1898. Since then Tinker has collected and published a detailed report of 57 additional cases in his dissertation "Ueber die perforierenden Magengeschwüre und ihre chirurgische Behandlung," read in Berlin on 1st July, 1900. A translation of this with the reports of yet 16 cases more may be found in the *Philadelphia Medical Journal*, February 5, 1900. I am able to add 8 more cases, so that we have now before us, for study and analysis, 240 cases. The lessening of the death rate among the later series is very great. It has been reduced from 71.51 per cent. in the first series collected by Weir and Foote, to about 40 per cent. Mikulicz reported a series of 103 cases with 33 deaths, a mortality of about 32½ per cent. This represents the total mortality. If now we go further into the details of these cases we shall find some really startling figures.

And, first, as to the results obtained in early operations, say, within twelve hours of perforation, as compared with operations performed more than twelve hours afterwards.

Weir and Foote—

	Total.	Died.	Recovered.	Percentage of Mortality.
Under 12 hours	23	9	14	39.13
12 to 24 hours	17	13	4	76.47
24 to 48 hours	18	16	2	88.88
Over 48 hours	14	12	2	85.71
Not stated	6	5	1	

Keen and Tinker—

Under 12 hours	26	5	21	19.23
12 to 24 hours	16	8	8	50.
24 to 48 hours	9	5	4	55.55
Over 48 hours	19	5	14	25.31
Not stated	8	5	3	

It is thus seen the percentage of mortality is only half as great in the second as in the first series, and that in both series it is twice as large in the second as in the first 12 hours' interval after perforation. And the results are still improving. In the last series of cases collected by Tinker, 19 cases came to operation within 12 hours of perforation, and of these 3 died, a mortality of 15.79 per cent.

The eight cases which I add to above series are as follows:—

Atherton	1	recovered.	
Kirkpatrick	1	recovered.	Personal knowledge.
Eve	1	recovered.	<i>Lancet</i> , 1900, Vol. I., p. 155.
Waitz	1	recovered.	<i>Deutsche Zeitschrift für Chirurgie</i> , Bd. LIV., 589.

and four cases which I have operated upon myself, of which one died and three recovered. These eight cases give a mortality of only 12.5 per cent., the fatal case being operated on 32 hours after perforation.

During the past five years there have been operated on in the Montreal General Hospital six cases of perforated gastric ulcer. All the cases operated on during the first 24 hours after perforation recovered. The late Dr. Kirkpatrick operated on two of these cases successfully. I operated on four, three of these successfully. The fourth case was that of a young girl who refused operation until 32 hours after perforation. The opening was found and closed, but the patient succumbed to the toxæmia of a general peritonitis. This is a very good result to have obtained, considering the nature of the cases that we had to deal with, as will appear later on. I have no doubt that other Canadian hospitals can show equally good results. Our success in the Montreal General Hospital is largely due and the credit should be largely given to the physicians who made the early diagnosis and promptly sent their people to the hospital. In no other way can one account for the lack of success in such an hospital as the Massachusetts General, where only one case has been saved out of ten. (Richardson, *Philadelphia Medical Journal*, Feb. 3, 1900.) Finney reports that they had but one case in the Johns Hopkins Hospital, and that that case died on the table (*Brit. Med. Jour. Epitome*, June 2, 1900). The mortality without operation is estimated at about 95 per cent.

Now, as to the site of perforation in operated cases; considering all the cases together, I find that the perforation has been on the

Anterior wall in	125 cases.
Posterior wall in	32 cases.
Near the lesser curvature in	61 cases
Near the cardia in	74 cases
Near the pylorus in	40 cases

These figures are in striking contrast to the figures of Welch, who found that of 793 post-mortem cases of gastric ulcer they were on the

Anterior wall in	69 cases.
Posterior wall in	235 cases.
Lesser curvature in	288 cases.
Near the pylorus in	95 cases.
Near the cardia in	50 cases.
Greater curvature in	27 cases.
At the fundus in:	29 cases.

(Osler, *Practice of Medicine*, p. 479.)

A possible explanation of the greater frequency of perforation on the anterior wall and at the cardia, may be the occurrence less frequently in these regions of protective adhesions. In fact, the adhesions between the base of the ulcer and neighbouring tissues play an important rôle in the history of peptic ulcer.

“In rare instances adhesions and a gastrocutaneous fistula form, usually in the umbilical region. Fistulous communication with the colon may also occur or a gastroduodenal fistula. The pericardium may be perforated, and even the left ventricle. Perforation into the pleura may also occur. It is to be noted that general emphysema of the cutaneous tissues occasionally follows perforation of a gastric ulcer.” (Osler, p. 479.) No doubt, in many cases, adhesions result practically in a sort of cure by preventing the escape of stomach contents.

Again, several cases have been reported of more than one perforation and death has resulted after operation, from the second perforation not having been recognized, or a second ulcer, existing at the time of operation, has perforated later on during convalescence.

Tinker (*Phil. Med. Jour.*, Feb. 5, 1900, pp. 258) states, after examining 232 cases, that “more than five-sixths of the cases occur among women. About three-fifths of the cases among women occur before the twenty-fifth year, and mostly among the servant-girl class. On the other hand, only about one-twelfth of the cases among men occur at this age, while more than one-half of the patients among men were more than forty years old. The youngest patient was nine, the oldest seventy-one.”

Into the subject of etiology and morbid anatomy of gastric ulcer I will not enter. The pathology of peptic ulcer is still involved in more or less obscurity. One very interesting point, however, I would like to draw attention to. I well remember the first perforated gastric ulcer I ever saw. The patient was a nurse in the Western Hospital, and had been going about performing her duties in a satisfactory manner until within forty-eight hours of her death. She had, however, complained

somewhat of dyspepsia and was anæmic. No operation was performed, but at the autopsy there was found a large round hole in the anterior wall of the stomach as large as a silver half-dollar. The edges were very little thickened, smooth and regular. The first case that I operated on presented a very large hole in the anterior stomach wall. When I opened the abdomen, there escaped a quantity of gas, serum, and stomach contents. On bringing the stomach forward, a small opening was seen, through which gas escaped. After adhesions to the left lobe of the liver had been separated, the opening appeared large enough to admit the end of the finger, and the walls of the opening were at least an inch in thickness. This thick wall was found to be made up almost altogether of lymph. It would not hold sutures, and I found it necessary to separate it from the stomach wall. When this was accomplished and the edges of the opening into the stomach were laid parallel with each other for suturing, the opening measured just three and a-half inches. The only explanation of this appears to be that adhesions had formed between the stomach wall and the left lobe of the liver before perforation became an accomplished fact, and that conservative adhesions had gone on forming at the same time that the ulcerative loss of substance had continued in the stomach wall; and the symptoms of perforation only occurred when the inflammatory process failed to effect a closure. It is in this way, I think, that the large openings sometimes reported can be accounted for.

Diagnosis.—Nearly all patients giving evidence of perforated gastric ulcer give a history of indigestion, gastric distress, or hæmatemesis, extending over a longer or shorter period. There are exceptions to this general rule, however, which one case, which I will report presently, illustrates very well; so that the absence of a history of stomach trouble should not be allowed to have too great weight.

The first symptom to indicate the occurrence of a perforation is pain. Pain may be called the danger signal, and should always receive most careful attention from the attending physician. The mere fact of there being pain in the abdomen is not diagnostic of anything. Pain in the abdomen, as elsewhere, derives its importance from its associations. Tenderness on pressure is the next symptom of importance. These two, if properly studied, will generally give a pretty good indication of the locus of the disturbing element. If the pain is found to be of maximum intensity in the gastric region, to be referred to the left along the left costal border or through to a point in the back just behind the stomach, and if associated with marked tenderness over this region, the physician's suspicions should be thoroughly aroused. If now there be found to be nausea or vomiting, and if these three symptoms, pain and tenderness

in the epigastrium and vomiting, be found associated with disturbance of temperature and pulse, the diagnosis of a perforated stomach may be made with a very considerable degree of certainty, more especially if these symptoms occur in a patient giving a previous history of stomach derangement.

The real difficulty, however, is not so much a question of fine, exact, differential diagnosis, in which the most experienced and astute practitioner may err, but in carelessly and without much consideration assuming that the pain is due to indiscretion in diet or to colic, in the loosest sense in which that loose word may be used. A dose of opium or a hypodermic, and locally heat or cold, are quickly ordered, and soon the condition of the patient is so altered and masked that only time can make it clear.

Lastly, a few words as to treatment. As already stated, the mortality from perforated gastric ulcer, not treated surgically, is estimated at 95 per cent. Let us look for a moment at the causes of death in the reported operated cases.

The first cause of failure in saving the patient has been failure in finding the perforation. In the first series of 78 cases reported by Weir and Foote, the perforation was not found in sixteen. In the second series of 78 cases of Keen and Tinker, the perforation was not found in nine; and in Tinker's series of 76 cases, it was not found in four. This defect in the treatment thus is gradually disappearing.

Another cause of failure is the non-recognition of other perforations or ulcers likely to perforate later on.

Hæmorrhage has occasionally been the cause of death. This may perhaps be avoided by not attempting to resect the edges of the ulcer, by having due regard for neighbouring vessels, and by carefully removing the layers of lymph found around the margins of the opening, so that sound tissue may receive the sutures.

Pneumonia and pleurisy have caused death in a few cases. Peritonitis and toxæmia have been the cause of failure in many cases. This is best prevented by early operation.

As soon as the diagnosis is made with a fair degree of certainty, an incision should be made in the median line below the ensiform cartilage, the stomach brought forward, and search made for the opening. Adhesions should be freely separated. Failing to find an opening in the anterior wall, the gastrocolic omentum should be divided and the posterior wall examined.

When the opening is found, if it is sufficiently large, it is well to pack around carefully with gauze pads and wash out the stomach with steri-

lised, normal, salt solution. This washing secures to the operator a clean, dry field, and to the patient a considerable lessening, if not total relief, from the after nausea and vomiting. The hole in the stomach wall should then be closed by two or three rows of sutures. If the opening cannot be closed, a tube may be inserted and packed around with iodoform gauze. It has been recommended to stitch a layer of omentum over the suture line. I have never found this necessary.

The opening closed and the stomach replaced, the operator should most carefully cleanse, by washing with normal, saline solution, the space above the transverse colon. A small opening should then be made just above the symphysis and any fluid accumulated in the pelvis removed by washing, and by a drain inserted and retained as long as seems advisable. If general peritonitis is present at the time of operation, the general peritoneal cavity should be carefully washed out.

I have found it advantageous to give a little peptonised milk twenty-four hours after operation, if it is well retained, and to gradually increase it. At the same time I give by rectum, every six hours, a nutrient enema composed of two ounces of peptonised milk, one ounce of raw beef juice, one egg, and half an ounce of brandy. It is generally well retained, if not a few drops of laudanum are added. I try to move the bowels once each day by enema, and find none so effectual as a pint of soap and water containing twenty grains of bisulphate of quinine in solution.

The eight cases that I have collected are as follows:—

Atherton—One case; no details; recovered. This I believe was the first case in Canada.

Waitz—Female, æt. 25; operation 21 hours after perforation, which was found at the lesser curvature near the cardia. (*Deut. Zeitschr. f. Chirurgie*, Bd. LIV., p. 589.)

Eve—Female, æt. 38; recovery. Operation probably about nine hours after perforation. The opening was found on the posterior wall near the lesser curvature. (*London Lancet*, Vol. I., 1900, p. 155.)

Kirkpatrick's second case (one already reported)—Female, æt. 20; recovery; ulcer about middle of anterior wall.

Armstrong—Female, æt. 20; operation 22 hours after perforation; large opening in anterior wall of stomach; recovery.

F. D., female, aged 27. This patient was admitted to the Montreal General Hospital six hours after perforation. A diagnosis of perforated gastric ulcer was made, but she positively refused operation until 32 hours after perforation. This is the only fatal case out of the six that we have had in the Montreal General Hospital. She died of peritonitis. The opening was on the anterior wall, two inches from the pylorus.

My third case, J. B., male, aged 40, has a rather interesting clinical history. I saw him with his family physician, Dr. LeBlanc, on the morning of Oct. 28, 1899. He was then suffering severely from abdominal pain. I was told that in November, 1898, he had suffered from a similar attack, accompanied by yellow discolouration of the skin. His family physician had seen him during the night before my arrival and had given him hypodermically one-half grain of morphia, and applied hot fomentations over the abdomen. He had not vomited. His family history was negative.

At the time of my visit he was still suffering a good deal, notwithstanding his morphia injection, $\frac{1}{2}$ grain in 5 hours. There was general abdominal tenderness of an extreme degree. I could not quite decide that the tenderness was greater in one part than in another, but thought that he was most sensitive over the region of the appendix vermiformis. He complained of the weight of the bedclothes. Muscular rigidity was most marked; nay, I may almost say that the abdomen was as hard as a board. Respiratory movements were entirely absent over the whole of the abdomen. Liver dulness was present and normal. No tumour mass could be made out. I had considerable difficulty in persuading him to come into the hospital for operation, but being ably seconded by Dr. LeBlanc, finally succeeded.

On his admission to the hospital his temperature was $96\frac{4}{5}^{\circ}$ F., pulse 104, respiration 26. I operated $11\frac{1}{2}$ hours after perforation. Thinking that the chances were that I had to deal with an appendicitis, I first opened the abdomen on the right side. Foul-smelling pus escaped. The border of the great omentum was covered here and there by loose layers of lymph. The appendix was found to be normal and removed. I then made an incision in the right semilunaris and exposed the gall-bladder, which was normal. No gall-stones could be felt. On examining the anterior wall of the stomach, I came upon a hole near the lesser curvature and about three inches from the pylorus. The edges of the opening were sharply defined. Closure was effected by three rows of sutures and the whole abdomen thoroughly washed out with sterilised, normal, saline solution. Drainage tubes were inserted above and below into the pelvis. Cultures made from the pus showed the presence of staphylococci. This man made a slow but perfect recovery and is now in good health.

My fourth and last case was a male aged 25. He was admitted to the Montreal General Hospital at 11 a.m., on the 30th December, 1899, complaining of severe abdominal pain. He stated that on the 25th of December he felt sick and went home from work early in the afternoon. He was at work again on the 26th, 27th, 28th and 29th, not feeling well

and suffering more or less from nausea. At 4 p.m. on the 29th, he was seized with severe pain in the epigastrium, accompanied by vomiting. He vomited three or four times, but no blood. He entered the hospital the following morning. Family history negative.

On admission his temperature was 98° F., pulse 95. He was anæmic. There was present a board-like rigidity over the whole abdomen, which was motionless, the breathing being entirely thoracic. There had been no movement of the bowels for four days; the bladder was empty. Tenderness was acute and general; his expression haggard and anxious. An hour after admission his temperature had risen to 101 $\frac{1}{5}$ ° F., and his pulse to 104.

He states positively that he never suffered from any stomach trouble before the onset of the present attack except that fifteen years ago he suffered similarly for two days.

An examination of the chest discovered dulness and faint blowing breathing over back of the right lung from the spine to angle of the scapula. Sputum mucopurulent, green, thick, scanty, and tenacious.

Operation, 22 hours after perforation. In this, as in the former case, I could not decide where the lesion was most likely to be found. An exploratory incision was made, first in the median line below the umbilicus. The extraperitoneal fat was of a peculiar, jelly-like consistency and the peritoneum greatly thickened. When the abdomen was opened a greenish-yellow fluid poured out carrying with it masses of a cream-coloured lymph, and large masses of this greenish, jelly-like material. Over a great part of the coils of intestine the same jelly-like substance was found, and it appeared to be covered by a layer of epithelium. Fine blood vessels entered these masses from the intestinal wall.

After the abdomen had been thoroughly cleansed with sterilised, normal, salt solution at a temperature of 110° F., an intussusception was found a short distance above the ileocecal valve. It was easily undone. The bowel above and below was normal. A second incision below the ensiform cartilage gave access to the anterior wall of the stomach, which was found perforated near to the lesser curvature and about 1 $\frac{1}{2}$ inches from the pylorus. This was closed in the usual way, and drainage provided for the upper and lower spaces.

This man made a good recovery but it was tedious. The pneumonia increased, and resolved very slowly. He left the hospital in good condition.

These last two cases afford a very good demonstration of the marked contrast in degree of virulence of infection from the stomach as compared with that from the small intestine or appendix vermiformis.