

REPORT OF A CASE OF PERFORATION IN TYPHOID FEVER CLOSED BY AN ADHERENT TAG OF OMENTUM, FOLLOWED BY A RELAPSE, SECOND PERFORATION AND DEATH.¹

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MAGGIE M., eleven years old, was admitted to hospital on February 7, 1899, the eighth day of typhoid fever. The disease ran rather a severe course, with high temperatures (104°), moderate tympany and diarrhea (four or five stools daily), and, on the twenty-third day, four hemorrhages from the bowels, the patient losing, during twenty-four hours, about as many ounces of blood. These were accompanied by increased tympany, complaint, on the patient's part, of abdominal pain, general tenderness on deep pressure and slight rigidity of the recti muscles. These symptoms soon abated, but not entirely, continuing in a lessened degree until the morning of the twenty-sixth day (three days after the hemorrhage), when the patient complained much of abdominal pain, which, she said, had become worse again during the night. The abdomen was moderately distended and slightly rigid; there was considerable pain, not localized, on deep pressure; there was no alteration in liver dulness; vomiting had not occurred, nor collapse, although the skin was cool and the expression anxious. The temperature (10 A. M.) was $99\frac{1}{2}^{\circ}$; pulse, 102; respiration, 36, following a plunge given at 7 A. M., when the temperature was $103\frac{1}{2}^{\circ}$, a fall of $3\frac{1}{2}^{\circ}$ in three hours. But as depressions of temperature of almost equal extent frequently follow plunges, its significance was not appreciated. Dr. G. G. Davis saw the case with me at 1 P. M., when the temperature had risen to $101\frac{1}{2}^{\circ}$; pulse, 126, respiration, 36. Dr. Davis did not think the condition serious, or the symptoms definite, enough to warrant operation. The next day (February 25th, twenty-seventh day of disease) the tympany was quite marked. The lower border of liver dulness was $1\frac{1}{2}$ inches above the costal border, right nipple line. The complaint of pain was continuous and insistent, and there was great abdominal tenderness on the slightest pressure, general in distribution, but greatest about the umbilicus; there was considerable muscular rigidity. No movable dulness could be detected, and there had been no vomiting. Temperature, $102\frac{1}{2}^{\circ}$; pulse, 140, respiration, 32. Dr. Davis again saw the patient, and it was decided to operate should the condition not speedily improve. The following morning the child was in every way better. The temperature was $99\frac{3}{4}^{\circ}$, pulse, 95; the distention and tenderness were much diminished, and there was no pain. After this the patient rapidly improved, the temperature falling almost to normal, and the child evidently entering upon a favorable convalescence. On the thirty-fifth day, however, what proved to be a genuine relapse began, as shown by gradual temperature rise, fresh rose spots and re-enlargement of the spleen. This second attack was more severe than the first. The temperature was constantly above 104° , and on the sixth day (thirty-ninth day of disease) seven ounces of blood were lost by intestinal hemorrhage. Seven days later (forty-sixth of illness, thirteenth of relapse) abdominal pain was again complained of, dull in character and continuous. At 1 P. M. the temperature was $103\frac{3}{4}^{\circ}$, pulse, 128, respiration, 26. There had been no marked fall,

although at 4 A. M. the temperature had sunk to $101\frac{1}{2}^{\circ}$ from $103\frac{1}{2}^{\circ}$ at 1 A. M., a fall of $2\frac{1}{2}^{\circ}$, but as similar depressions had been frequent during the attack, and as it had followed a sponge, no significance was attached to it. The next day the tympany had somewhat increased, but the liver dulness was unchanged. Tenderness on pressure was very marked, the most tender point being on the left side, just below and close to the umbilicus. Decided muscular rigidity was present. Shifting dulness could not be elicited. Vomiting had occurred once, an hour before my visit (1 P. M.). Temperature, 103° , pulse, 134, respiration, 56. There had been no fall. Dr. Davis again saw the case, and while not positive that a perforation existed, considered that such an accident was imminent. At 4 P. M. (three hours later) the temperature fell to $99\frac{1}{2}^{\circ}$ (from 103° at 1 P. M.), but rose rapidly to $103\frac{3}{4}^{\circ}$ at 7 P. M. Symptoms of perforative peritonitis — collapse, vomiting, great tympany and rigidity rapidly developed, death occurring the next day at 1 P. M.

Abstract of the autopsy, by Dr. W. E. Robertson²: Considerable gas and fluid were found in the abdominal cavity. There was much lymph and many recent adhesions. The great omentum was rolled up and displaced to the right side of the abdomen, except one strand, which had formed an attachment to the ileum at a point six inches from the ileo-cecal valve, serving to close an aperture, very small and slit-like, in an ulcerated Peyer's patch, which had evidently perforated some time before. Another perforation was found twelve inches from the cecum, about one-eighth inch in diameter; its edges were sharply defined, and it allowed a free escape of the intestinal contents. The mesenteric glands were enlarged. The appendix was normal. There were a few other ulcers in the colon. The spleen weighed seven and one-half ounces; liver forty-two ounces. There were no erosions in the gall-bladder. The other lesions were those usually found after death from typhoid fever.

This interesting history illustrates how a perforating typhoid ulcer may undergo healing with complete cessation of symptoms, and doubtless many cases presenting symptoms of perforation, which have resulted in recovery without operation, have been of this nature. A similar case is reported by Buhl,³ where, at the autopsy of a patient dying from hemorrhage on the twenty-third day after symptoms of perforation, a piece of the omentum was found adherent to, and completely closing, the opening. Or the same end may be attained by the formation of a local abscess, as in a case reported by the same author, an abscess formed at the site of perforation, and caused death by rupture at the expiration of five weeks. Adhesions may also form and close the ulcer, thus preventing extravasation into the general peritoneal cavity. Such a condition was found at operation in a patient of J. Bion Bogart's,⁴ a man aged thirty, who developed during a second attack of fever symptoms of perforation. At the operation the appendix was found adherent to the ileum, four inches from the cecum. On separating the appendix from the gut a minute perforation was exposed. The perforation was completely closed by the adhesion and surrounding inflammatory exudation. The patient recovered from the operation, but died later from obstruction at the hepatic flexure from an old adhesion. This case of Bogart's, as well

¹ Read before the Philadelphia County Medical Society, April 26, 1899.

² Specimens exhibited at the meeting.

³ Zeitschrift für rationelle Med., 1857, N. F., viii, S. 12.

⁴ Annals of Surgery, 1896, vol. 1, p. 696.

as others in the literature, shows how successful operative interference is if undertaken early, before general peritonitis has occurred, especially in those instances where adhesions (or local abscesses) have formed, or where symptoms arise indicating that a perforation is about to take place. That such a condition sometimes exists cannot be doubted.

Harvey W. Cushing,⁶ whose valuable paper first directed my attention to this matter, and whose conclusions are liberally made use of in these remarks, has emphasized the frequent presence of what he terms a "pre-perforative stage of ulceration." This condition he describes as "a localized inflammation of the serosa leading to a slight adhesive peritonitis, due to the near approach of an ulcer to the general peritoneal surface." The symptoms of this stage, as I have observed them, are gradual, rather than sudden, onset of pain, continuous in character, *which is complained of by the patient himself*, associated with more or less general or localized abdominal tenderness on palpation, with often, but not always, slight muscular rigidity. In addition, Cushing states that there may be a moderate leucocytosis. Vomiting is usually absent, as well as any marked change in temperature, and, if a change occurs, a rise is as often observed as a fall.

In my case there was almost continuous complaint of abdominal pain, with tenderness on deep pressure, and variable but slight muscular rigidity for three days preceding the first perforation, which occurred, probably, with the marked fall of temperature on the morning of the twenty-sixth day. At the second perforation, similar symptoms foreran by one and one-half days the decided fall in temperature, collapse and other symptoms of general septic peritonitis.

In another case under my care this winter, a boy of fourteen, an extremely nervous lad, there was constant complaint of abdominal pain, moderate tympany, tenderness on pressure and, at times, muscular rigidity, but no vomiting or change in temperature, for three days before signs of extravasation into the general peritoneal cavity occurred. In a third patient, also a boy, of about fifteen years, rigidity, tenderness and complaint of pain in the epigastric region, without vomiting, and with higher temperature, continued for a week before general peritonitis developed. This case was taken home before death, but, from the description given, general peritonitis probably determined the fatal issue.

If there is a definite recognizable stage, characterized by symptoms, sometimes present before actual perforation occurs, it is evident that operative measures undertaken during such a stage would be attended by a much greater measure of success than is usually seen after surgical interference in typhoid perforations. And even where operation is not performed, the recognition of this condition would, at least, put us upon the alert: by enjoining greater quiet, the cessation of baths, and constant watchfulness for the occurrence of more pronounced symptoms. My own experience leads me to believe that, had I appreciated the significance of these symptoms in some of my cases, perforation might not have occurred. Of course, "the earlier operations are performed the more will mistakes in diagnosis be seen."⁶ Dr. Davis and myself were guilty of this error in one of our cases: A man, age forty, in the fifth week of typhoid fever, was seized

with sudden, sharp pain in the umbilical region after getting out of the bath. When seen by us seven hours later there was very great tenderness on pressure, particularly over the transverse colon and in the epigastrium, complaint of pain by the patient, sufficient to require a small dose of morphine, rigidity, and slight movable dulness in the right flank, moderate distention, a fall of temperature of 2°, with a pulse of 140, but no vomiting. An exploratory incision was decided upon. At the operation *no perforation was discovered*. The appendix was normal, and the portion of the ileum exposed, about three feet, revealed only intense congestion of the serosa and many thickened Peyer's patches; there was no peritonitis or adhesions. A small amount of serous fluid was present. The gall-bladder was not examined. The peritoneum was flushed with saline solution and the wound closed. The patient made an excellent recovery, and seemed rather benefited than otherwise by the operation. Cushing⁷ reports a somewhat similar case in a girl of fifteen, and C. J. Symonds⁸ operated on a case where the history and mode of onset indicated a perforating gastric ulcer, but found nothing abnormal. Both patients made uneventful recoveries. Cushing and Symonds regarded their cases as hysterical. In mine we were inclined to believe that either a pin-hole perforation had been overlooked, or that the symptoms were due to a temporary obstruction of some sort, a twist of the gut, for instance, that relieved itself before or at the time of operation. The operation, evidently, if skillfully performed, is almost devoid of danger. In advising operation, however, the surgical means at hand should always be considered by the medical attendant, for, as Symonds⁹ well says: "Success can only be obtained in the hands of an operator whose training makes an error in technique almost impossible."

The diagnosis of intestinal perforation in typhoid fever is at times one of the most difficult problems that is presented to the physician for solution. At the same time, early operation, before collapse symptoms have developed, affords, in the majority of instances, the only chance the patient has of recovery. And if painstaking and thorough study of our cases will show that the actual perforation is sometimes preceded by well-defined symptoms — and the cases reported in this paper would seem to corroborate the observations of Cushing in this respect — their early recognition will undoubtedly be the means of saving some lives that are now lost by waiting for the signs of perforation as usually given in the text-books.

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COMPOUND FRACTURE OF THE SKULL, WITH ESCAPE OF BRAIN MATTER.¹

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PRIVATE H. S., age 22, Light Battery G, Sixth Artillery, while on guard on board a water boat alongside the United States transport *Newport*, in the Bay of Hilo, Philippine Islands, during the evening of

¹ Published by authority of the Surgeon-General.

⁶ Cushing: Johns Hopkins Hospital Bulletin, 1898, vol. ix, No. 82, p. 257.

⁸ Symonds: British Medical Journal, March 4, 1899, p. 519.

⁹ Symonds: loc. cit.

⁵ Johns Hopkins Hospital Bulletin, 1898, vol. ix, No. 82, p. 257.

⁶ C. J. Symonds, M.S.: British Medical Journal, March 4, 1899, p. 519.