PERFORATION IN UTERO OF A GASTRIC ULCER*

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Gastric ulcer in children before the tenth year is unusual and warrants recording. The literature of the last thirty-five years contains a number of references to gastric ulcer in patients within the first decade of life. A complete summary of those which we have been able to find is as follows: Lockwood 125 cases, Covey 1 case, Skill 5 cases, Fenwick 18 cases, Parkinson 1 case, Welsh 1 case, making a total of 160 cases. During the first year of life there are eight cases recorded, one each by Rillet, Billard, Goodhart, Phelip and Fey, Cade, Lockwood and Rotch.

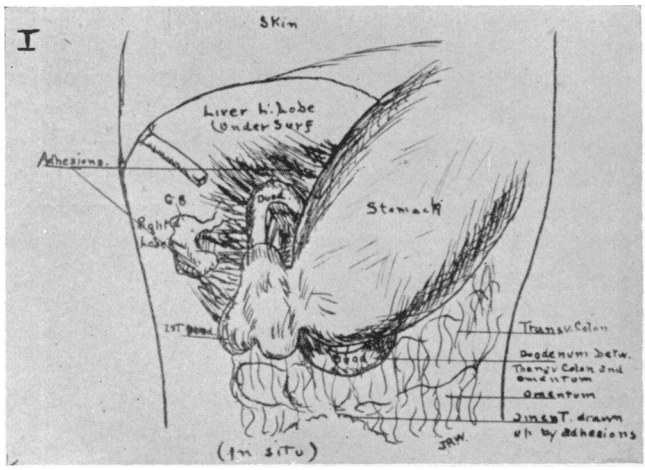

Fig. 1.—Showing organs adjacent to pyloris, bound together by adhesions.

In the group of cases of perforation in the newborn, we can find but two cases aside from the one to be reported. One of Bisset, in an infant forty-eight hours old, and one of Nuzum, who was twenty-four hours old. We are inclined to believe that these two cases do not fall into the class in which we have placed the present one, namely perforation *in utero,* because the two cases alluded to were apparently normal at birth and remained so until there were symptoms of an acute abdominal catastrophe, followed by melena neonatorum or vomitus or both. These symptoms were acute and death rapidly followed in about the time of sequence that the rupture of a hollow viscus usually takes.

Case Report.—The patient, a colored male infant, was born September 8, 1922, and admitted to Children's Hospital when ten days old. Though his twin brother had been perfectly normal (and has had no illnesses up to the time of reporting this case, January, 1923) our patient had exhibited symptoms of pyloric obstructions from birth. The mother had previously borne a normal boy and a

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girl, who are now living, and has had one miscarriage. The birth of the twins presented no unusual difficulties.

From the very first the patient vomited immediately after taking fluid. This vomiting was at times projectile but usually regurgitant. The vomitus itself was "foamy" and of a greenish color. The parents reported several bowel movements consisting of yellow and green fecal matter. Urination had been normal. No history could be obtained of convulsions or any abdominal catastrophe as might be evidenced by acute abdominal pain and distention, nor could any history be obtained of bloody vomitus or melena.

Physical examination showed a small, fairly well nourished male negro baby ten days old. The skin was copper colored, dry and wrinkled as if from the loss of weight or fluid. The child presented the appearance of dehydration. The head, chest, genitalia and extremities were apparently normal. The abdomen showed folds of skin about the groins and had the appearance of dehydration. There was a small umbilical hernia. Liver and spleon not palpable. No rigidity or tenderness. It was not possible to demonstrate peristaltic gastric waves or palpate any mass in the right hypochondrium. Impression, pyloric stenosis or high duodenal obstruction.

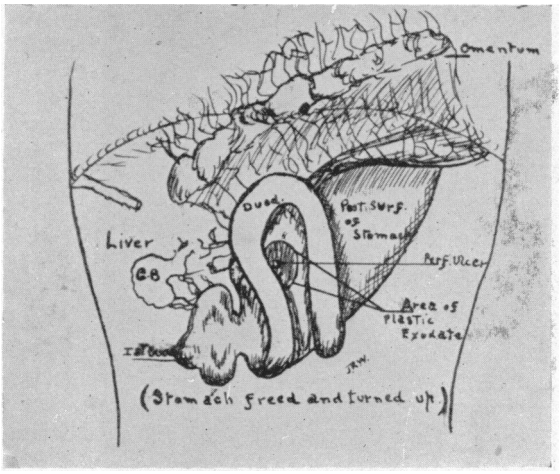

FIG. 2.—Showing opening in wall of stomach, brought into view by separating adherent omentum.

Abstract of clinical notes previous to operation. While in the hospital the temperature range was erratic, from 96 to 100.4. The pulse varied from 100 to 144. Respirations from 28 to 52. There was apparently no relation between these variations. There were two bowel movements during the first four days in the hospital, both following enteroclysis. They were partly formed and dark green in color. On the day of operation there was a small yellow-green stool. He vomited from one to five times during each twenty-four hours before operation.

Patient was admitted to the medical service and was fed with mixtures composed of skim milk, farina and wine whey in various combinations. He lost weight steadily, falling from 4 pounds 9 ounces, to 4 pounds 4 ounces on the day of operation. Urine normal except for a faint trace of albumen and a few hyaline casts. Vomitus was acid in reaction and contained no bile or blood. The stomach contents after lavage contained bile.

Röntgenological report.—On September 21, 1922, he was given a barium meal. Six hours later no barium had passed into duodenum and after twenty-two hours only a very small portion had passed through the pylorus. Diagnosis.—A very definite obstruction at the pylorus.

Operation.—Under ether anæsthesia the abdomen was opened through a right upper rectus incision 6 cm. long. The pylorus was found lying very deep in the hepatic fossa and it was exposed with difficulty. Adherent to the pylorus and to the greater curvature of the stomach there was a section of small intestine,

which upon separation of the adhesions, was found to be the distal portion of a very long duodenum. When these two long limbs of the U-shaped duodenum were finally separated the flexure or knee, corresponding to the second portion of the duodenum, was found to be adherent to the under surface of the liver and gall-bladder. These adhesions were strong, well organized and separated with difficulty owing to their density. They were practically avascular. Following the release of this duodenal obstruction the distal portion was seen to fill and distend with contents expressed from the stomach. The stomach was then more carefully examined and a circular constriction was found about 2 cm. from the pylorus which presented the appearance of an hour-glass contracture. The wound was closed by layer sutures and the child's condition was very satisfactory when he left the operating room. He continued to do well post-operatively except for shallow breathing and lack of vitality. There was one stool subsequent to operation which contained yellow-green feces. There was no vomiting after operation until probably just before death, during the twelfth hour, when the nurse found the child moribund.

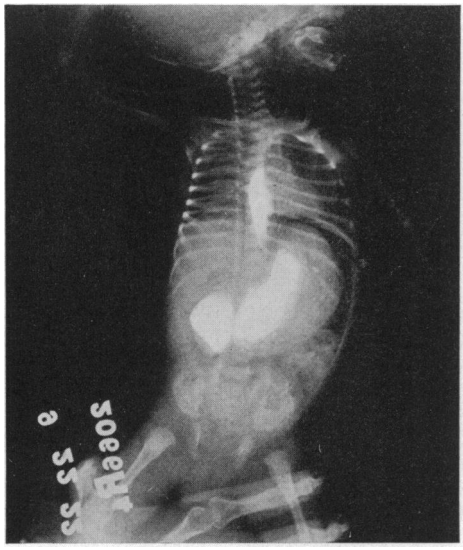

FIG. 3.—Showing distinct hour-glass contracture and position of dense band of connective tissue.

Autopsy.—Performed by enlarging the operative incision. Intra-abdominal palpation failed to reveal any abnormality except in the right upper quadrant. The entire region surrounding the pylorus was a mass of adhesions in which all the organs seemed bound together. These adhesions were very strong, dense and fibrous. There were no so-called fresh adhesions. Before cutting any of these fibrous bands a survey revealed the pylorus and the hour-glass contraction pulled down, back and beneath the inferior surface of the right lobe of the liver. A small portion of the duodenum was seen protruding below the stomach in this region. Pressure upon the stomach showed that the operative mobilization had been complete in relieving the obstruction, for air could be freely forced from the stomach into the lower gut. Upon raising the left lobe of the liver the stomach and first portion of the duodenum were exposed more freely. Dense adhesions were found between the gall-bladder, the under surface of the liver, the duodenum and the stomach, and the posterior wall of the stomach was distinctly involved in these adhesions. Upon separating these and cutting several of the fibrous bands the posterior surface of the stomach was exposed.

The two limbs of the duodenal loop were firmly adherent to the posterior surface of the pyloric antrum. Between these two limbs and well down towards the greater curvature of the stomach, partially covered by the one nearest to the pylorus, was a dense patch of flat, yellowish oval shaped tissue measuring 2 x 2.5 cm. By accident the edge of this patch was raised and finding that it stripped easily from the stomach it was removed and a small perfectly rounded hole in the stomach wall was revealed. The peritoneal edges of this hole were inverted. There appeared to be no inflammatory reaction in the lesser peritoneal cavity

in this region. Pressure on the stomach caused several bubbles to emerge from this perforation. A probe carefully inserted through the hole proved the lesion to be a complete perforation of the stomach wall, it measured several millimeters in diameter. A section of the stomach wall containing the perforation was removed and again the absence of active inflammation about this area was especially noted. The interior of the stomach failed to reveal any other lesions except a very dense mass of indurated tissue in the stomach wall at the site of the circular contraction of the pyloric antrum, but there was no ulceration seen in this area. The described perforation was approximately 4 cm. from the pylorus and was situated upon the posterior wall of the stomach near the greater curvature. There

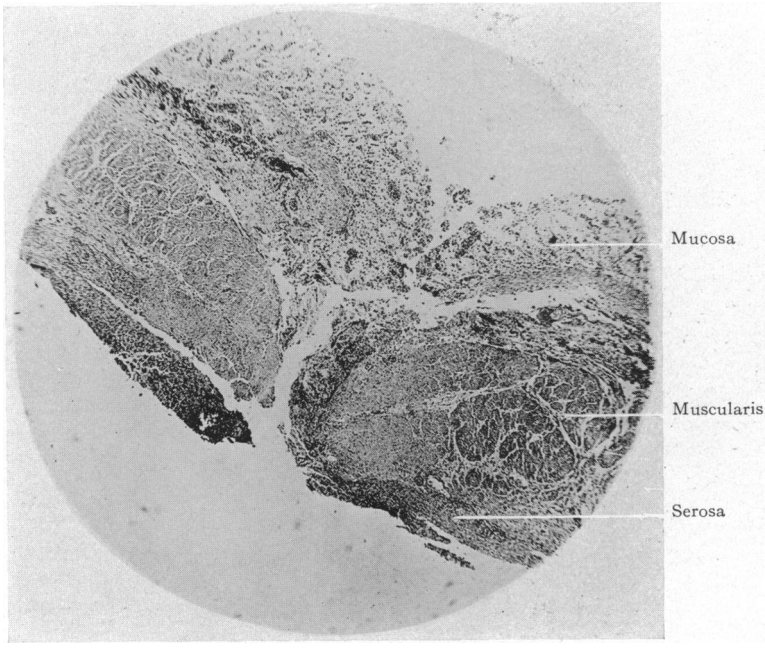

FIG. 4.—Low-power view of perforation.

were no evidences of peritonitis in the lesser or greater peritoneal cavities. There were no evidences of free blood in the stomach or intestines.

Microscopical Examination.—Section shows a strip of muscular and mucous membrane covered on one side by a split-up fibro-cellular zone upon which is a recent fibrino-purulent exudate. The muscular band is fairly well preserved although somewhat split by œdema. The submucous zone is distinctly hemorrhagic. Mucous membrane is hemorrhagic, lacking in fully formed tubules, while many epithelial cells are degenerated and absent from near the surface. The whole strand shows a defect or solution of continuity running from mucous surface to the exudate mentioned above as lying on the outer side. This defect is partly bridged over by a fibrinous exudate and by a piece of necrotic muscle; remnants of glands may be seen near it as if displaced from the mucous to the muscular layer. The edges of the mucosa proper at the point of defect are turned in somewhat but do not show distinct ulcerative-tissue reaction or marked round-cell infiltration. Diagnosis.—Rupture of a part of the intestinal tract, probably stomach, with fibro-purulent peritonitis.

Discussion.—Our case was apparently suffering from gastric or intestinal obstruction at the time of birth which became gradually worse until the tenth day of life. At the time of admission to the hospital all the symptoms of pyloric obstruction were present. Contrary to Holt's broad statement that the only symptom of gastric ulcer in children is hemorrhage, our case gave no symptoms of melena or bloody vomitus. In fact, there were no symptoms of gastric ulcer or perforation before operation nor was the diagnosis made at operation, so perfectly had nature taken care of the situation.

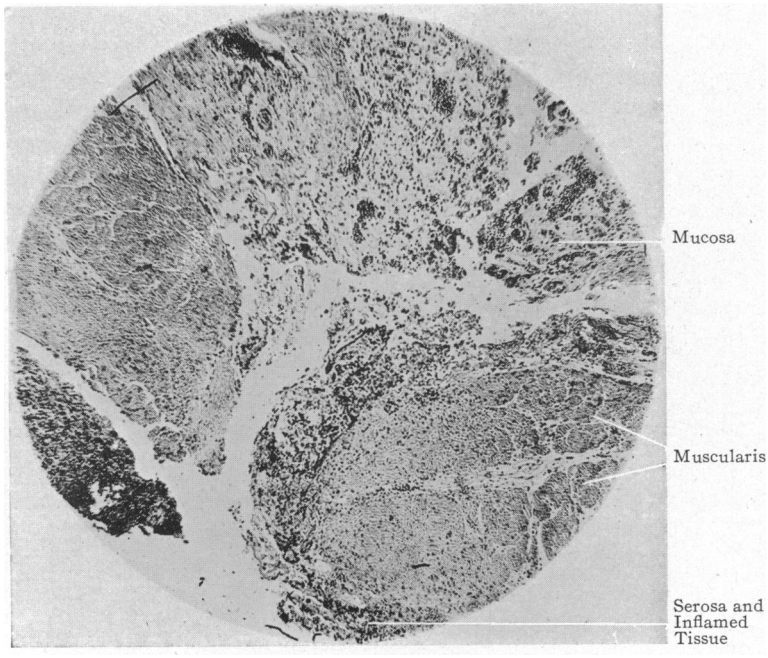

FIG. 5.—High-power of Fig. 4.

We submit the following reasons for our diagnosis of perforation *in utero*. (*a*) Unless an acute gastric ulcer occupies but a matter of a few hours from its start to perforation, we may safely assume that the two cases of Bisset and Nuzum developed their gastric ulcers *in utero*. Both of these cases were apparently normal for a number of hours after birth, when symptoms of an acute abdominal catastrophe were followed by peritonitis. These conditions were entirely absent in our case.

(*b*) Though intra-abdominal adhesions may form in a few hours under these conditions, they are friable, vascular and of the consistency of cobwebs. In our case the adhesions showed a degree of firmness and density and a vascularity that is seen in a stage far later than that of acute inflammation. The age of these bands was certainly several weeks antedating the child's birth. The cause of these adhesions, the perforated ulcer, antedated the adhesions.

(*c*) When we consider that after birth the stomach and intestines are distended for the first time with food, is it not reasonable to think that if this perforation had occurred after birth such gastric contents escaping into the peritoneal cavity would have given acute signs of a general or at least a localized peritonitis? As our patient exhibited no signs even remotely suggestive of an acute inflammatory abdominal condition, nor any pathology of such a process, we feel that the effective plugging of the perforation by a mild chemical reaction of the gastric secretion took place before the stomach contents were infected. The stomach before birth is naturally sterile and contains slightly acid secretion and this could well have set up an acid irritation on the serous surfaces of the lesser peritoneal cavity sufficient to cause adhesions without having a bacterial infection.

(*d*) Many authorities on children's diseases believe that hemorrhage is a constant symptom of gastric ulcer in children. The control of bleeding from such an ulcer is, of course, by the process of thrombosis of the vessels. A close study of the microphotograph will show that several small vessels, surrounding this perforation and in the ulcer area, are filled with plugs of tissue which are probably connective tissue. Some time was required to reach this stage of organization. Again the piece of tissue inverted from the serous surface into the canal is not that of an acutely eroded surface but resembles chronic tissue.

(*e*) The definite hour-glass deformity of the stomach, with infiltration of the stomach wall, would seem to indicate a chronicity of the process which was longer than fifteen days preceding the operation.

(*f*) The fact that the barium meal given on the thirteenth day showed no leakage through the perforation into the lesser peritoneal cavity is evidence that the perforation was closed at this time.

BIBLIOGRAPHY

Lockwood, C. D.: Trans. of West. Surg. Assn., 1913, p. 255.
Covey, G. W.: Neb. State Med. Jr., vol. v, No. 4, p. 104.
Still: Br. Med. Jr., May, 1899.
Fenwick: Book—Ulc. of Stomach and Duod., 1900.
Parkinson: Reports of Soc. for Dis. of Child., vol. i, No. 50. p. 143.
Rilliet, Fred.: Traite Clin. et Prat. des Mal. des Enfants, 1890.
Billard, Chas. M.: Treat. on Diseases of Infants (translation), 1890.
Goodhart, J. F.: Trans. Path. Soc., London, 1881.
Phelip and Fey: Arch. de Med. d'Enf., 1920.
Cade: Rev. Mens. d'Mal. de l'Enf., vol. xvi, p. 57, 1898.
Rotch, T. M.: Am. Jr. Med. Sc., 1899, No. 118.
Bisset, A. G.: Lancet, London, 1905, vol. ii.
Nuzum, T. W.: Wis. Med. Jr., 1916–1917.
Holt: Book—Dis. of Inf. and Child., 1911, p. 336.
Cohnheim, P.: Book—Col. of Phys. Oh-130 a. 3.
Gundermann, W.: Arch. f. klin. Chir., Berl., 1913, vol. ci, pp. 546–59.
LaRoque, G. P.: Am. Jr. Med. Sc., 1913, vol. cxlvi, pp. 566–86.
Rosenow, E. C.: J. A. M. A., 1913, vol. lxi, pp. 1947–50.