HEMORRHAGIC JEJUNAL DIVERTICULITIS*

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MECKEL'S DIVERTICULUM is a rather common and well-accepted source of gastro-intestinal hemorrhage; however, recognition of non-meckelian diverticula of the small intestine as a cause of this distressing form of blood loss is indeed rare, as judged by the number of reported cases in the medical literature. It is our belief that this situation, while admittedly quite rare, occurs more frequently than it is recognized, and should thus be considered in any differential diagnosis of overt or occult gastro-intestinal hemorrhage or of any hypochromic anemia which cannot otherwise be explained.

The report of a representative case which stimulated our interest in this problem follows.

Case 1.—A 58-year-old white male linotype operator was first seen on May 9, 1950, with a chief complaint of "weakness and anemia" of about 6 months' duration. He was apparently well until December, 1949, when persistent coryza developed, with generalized aches and pains for which he received injections of penicillin from his local physician. Shortly thereafter he noted the onset of weakness, and of aching and stiffness in his neck. During January, 1950, he was hospitalized, elsewhere, because of increased weakness, and about this time had some vomiting but no hematemesis. Extensive roentgenologic and laboratory studies were carried out. Then he was told that he was anemic and had "arthritis and heart disease"; he was started on treatment with iron by mouth and a "heart tablet" (digitalis?).

After he left the hospital, continued weakness compelled him to spend the next 5 weeks in bed at home, but from March 6 to May 3, 1950, he was able to carry on at his regular job, though he never felt well. On the latter date, vomiting recurred and weakness again became marked, necessitating hospitalization; in the hospital the concentration of hemoglobin was found to be 31 per cent. He underwent four transfusions of blood and was referred to the Mayo Clinic. Stools had been normal in color until he began taking iron in January, but he was told that tests for blood gave positive results. Blood had never been noted in the vomitus, and there had been no abdominal pain or change in bowel habits. Appetite and digestion remained good, but he had lost 20 pounds during his illness.

A review by systems was noncontributory. The past history was essentially negative. There had been no previous surgical operations, and the family history contained no evidence of chronic or familial disease.

Physical examination revealed a well-developed, well-nourished white man who was extremely pale and appeared chronically ill but who was not in acute distress. There was a blowing systolic murmur over the precordium. The blood pressure was 160 mm. of mercury systolic and 88 mm. diastolic, and the pulse was regular and of good volume, with a rate of 84 beats per minute. The edge of the liver was palpable 2 to 3 cm. below the costal margin, and was smooth and nontender. The spleen was not palpable, and no tenderness or abdominal mass or fluid was detectable. The prostate was slightly enlarged, and black stool was noted on the examining finger. There was no palpable lymphadenopathy or tenderness of bone.

The value for hemoglobin was 7.6 Gm. per 100 cc. of blood, and the erythrocyte count was 2,400,000 per cubic millimeter. The leukocytes numbered 6600 per cubic millimeter with an

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essentially normal differential count. Basophilic stippling of the erythrocytes was present, with microcystosis and hypochromasia. The sedimentation rate (Westergren) was 126 mm. in 1 hour (normal less than 20). The fragility of the erythrocytes was within normal limits. The platelet count was 259,000 per cubic millimeter (normal 100,000 to 250,000), and the prothrombin time was 22 seconds (normal 19 seconds). The value for serum bilirubin was 0 with the direct reaction and 0.5 mg. per 100 cc. with the indirect reaction. The Kline reaction was negative, the blood group was O, and the blood was Rh positive. With the sulfobromophthalein (bromsulphalein) test 14 per cent of the dye was retained. The thymol turbidity test gave a value of 0 units, and the cephalin-cholesterol flocculation reaction was negative. The concentration of blood urea was 78 mg. per 100 cc., but with hydration of the patient it dropped to 40 mg. in 3 days. The value for total serum proteins was 6.6 Gm. per 100 cc. with an essentially normal albumin-globulin ratio of 4.2:2.4.

The urine had an acid reaction and a specific gravity of 1.010, and there was albuminuria, grade 4. A few granular casts were present, but there was absence of sugar, erythrocytes, pus cells, and Bence Jones protein. A 24-hour specimen contained a total of 0.02 mg. of lead (a normal value). These findings were all explained on the basis of low-grade chronic glomerulonephritis.

The basal metabolic rate was +9 per cent, and the electrocardiogram was normal except for left axis deviation. The value for free hydrochloric acid in the stomach was 60 units and for total acids 80 units. Occult blood was found in the stool by both the benzidine and guaiac methods.

The roentgenogram of the chest was interpreted as disclosing no abnormality, and that of the neck revealed calcification in the ligamentum nuchae, with hypertrophic changes in the cervical portion of the spinal column. The esophagus, stomach, and duodenum were normal on fluoroscopic examination, but roentgenograms of the small intestine revealed several large diverticula in the upper part of the jejunum (Fig. 1). Repeated fluoroscopic examinations of the stomach failed to detect any evidence of duodenal ulcer, esophageal varices, diaphragmatic hernia, or other lesions which might be the source of bleeding. Diverticulosis of the colon was demonstrated by barium enema.

Marrow aspirated from the sternum was normal. There was active erythropoiesis.

With the diminished function of the liver, as indicated by the retention of sulfobromophthalein, a further investigation of the possibility of esophageal varices was deemed advisable. Consequently, on May 23, 1950, esophagoscopy was performed, and no evidence of any varicosities could be seen. The esophagus appeared normal.

With rather good evidence of gastro-intestinal bleeding of significant degree, surgical exploration seemed the wisest course to follow, especially since the patient was known to have at least one lesion which had been seen by the roentgenologist and which not only was capable of causing the hemorrhage but also was amenable to surgical treatment; namely, jejunal diverticulosis. Consequently, on May 25, 1950, with the patient under nitrous oxide, oxygen and ether anesthesia, exploratory laparotomy was performed through a primary transverse incision in the left upper quadrant. The liver was normal in color, somewhat soft, and had a sharp edge. Moderate diverticulosis of the colon was demonstrated, as had been found preoperatively. In the upper part of the jejunum, beginning about 50 cm. below the ligament of Treitz, 4 large diverticula were seen arising from the mesenteric margin and extending out into the mesentery; they measured 2 to 4 cm. in diameter. They occurred over a length of about 20 cm. of jejunum. The walls were thin and the structures were completely collapsed, making it very difficult to locate them in the mesenteric fat; in fact, except by distending the bowel with air, it was impossible to find them by palpation alone. The stomach and duodenum, gallbladder, spleen, and other abdominal organs were all normal to inspection and palpation. A segment of jejunum measuring approximately 25 cm. in length and containing the diverticula was removed (Fig. 2) and an end-to-end jejunojejunostomy performed in the usual manner, with an inner row of running catgut and an outer row of interrupted cotton sutures. One thousand cubic centimeters of whole blood were administered intravenously during the operation because of the severe anemia present preoperatively. The abdomen was closed in layers with No. 1 chromic catgut.

The pathologist reported that the walls of the diverticula consisted of mucosa and submucosa covered by peritoneum, with a few strands of muscle. No ulceration of the mucosa, ulcer scars, or heterotopic gastric mucosa or pancreatic glands could be demonstrated. Special stains for the detection of amyloidosis failed to show any evidence of such a condition; this was done because of the presence of low-grade chronic nephritis along with the evidence of impairment of hepatic function.

The patient's course following operation was uneventful, and he was dismissed from the hospital on June 2, 1950, the ninth postoperative day, and allowed to leave Rochester and return to his home 3 days later. On November 30, 1950, the patient wrote that he felt well and was working

daily, and that the value for hemoglobin was found by his local physician to be 13 Gm. per 100 cc. of blood, with an erythrocyte count of 3,900,000 per cubic millimeter.

In September, 1951, 16 months after operation, the patient returned to Rochester for a follow-up examination. He felt well and had no complaints of any kind. He stated that he had missed no time from work and that he had even played a

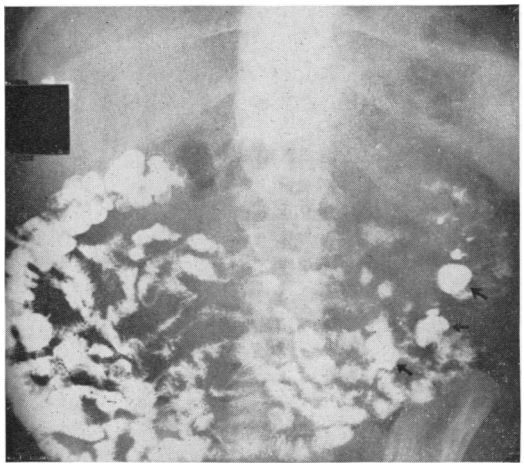

FIG. 1.—(Case 1). Roentgenograms of the small intestine showing three of the four diverticula containing barium.

few games of volleyball during the summer. The concentration of hemoglobin was 12.6 Gm. per 100 cc., and the erythrocyte count was 4,500,000 per cubic millimeter, with an essentially normal blood smear. The sedimentation rate was still elevated (72 mm. in one hour), but the retention of sulfobromophthalein had dropped to 8 per cent. The urinary findings were essentially unchanged, except for grade 1 glycosuria, and further investigation revealed mild diabetes, which was controlled by diet alone.

HISTORICAL DATA

While Chomel in 1710 described what apparently was a non-meckelian diverticulum of the duodenum, the first mention of such lesions in the jejunum is generally credited to Sir Astley Cooper in 1844, although Walker has stated that Cooper died in 1841, and that the reference* is contained in his book published in 1807. Only scattered references to this medical curiosity are found in the literature between that time and 1920, when Case reported five cases of jejunal diverticulosis diagnosed roentgenographically, two of which seem to be the first published examples of surgically treated lesions of this kind which had been diagnosed preoperatively. Up until that time, nearly all references concerned necropsy material. Case's paper may be arbitrarily taken as the beginning of the modern era of diagnosis and treatment of this condition, and since then many other publications on this subject have made their appearance. Brief mention is warranted of the contributions of Rankin and Martin, and of Benson, Dixon, and Waugh who surveyed the literature and outlined the experience at the Mayo Clinic before 1943.

CLASSIFICATION AND ETIOLOGY

The generally accepted classification of diverticula of the small intestines provides for two categories: (1) true or congenital diverticula and (2) false or acquired diverticula. It would seem desirable to drop the terms "true" and "false" in this connection, as they really have little meaning, and reserve the term "false" for those diverticula of the first portion of the duodenum which are occasionally found in association with duodenal ulcer, the "ulcer diverticulum" of Edwards, and which have been explained as resulting from cicatricial contraction of the opposite wall of the viscus. The saccculations accepted as congenital contain all three component layers of the bowel wall, while in the acquired type they have either a highly attenuated or absent muscularis and represent what is probably a true herniation of mucous membrane. Various authors have championed either the congenital or acquired origin as explaining all of these diverticula. Probably closer to the truth would be the fact that some fall under each heading, although it does seem that

* An excellent engraving from Cooper's book published in 1807 is reproduced by Walker, leaving no doubt that Cooper was talking about this condition.

most of those in the jejunum are acquired, with the congenital group being represented by those rare instances of partial reduplication of the gut.

The etiology of the acquired group has occasioned some controversy in the literature, but the mass of opinion leans toward the mechanical factor of intraluminal pressure producing herniation through a "locus minoris resistentiae," or site of decreased strength, around the entry point of blood vessels into the bowel wall.[6] This explanation would account for the mesenteric location of these structures, and for the fact that they are found in the older age group. It would also be in line with the etiologic basis for pulsion diverticula of other portions of the gastro-intestinal tract, that is, the esophagus and colon, as we understand it, and is compatible with their usual multiplicity in the jejunum. Various contributing factors include chronic constipation, chronic low-grade intestinal obstruction,[1] and an inherent weakness of the connective tissue elements of the bowel wall.

INCIDENCE

Figures indicating the incidence of jejunal diverticula vary with the author quoted.

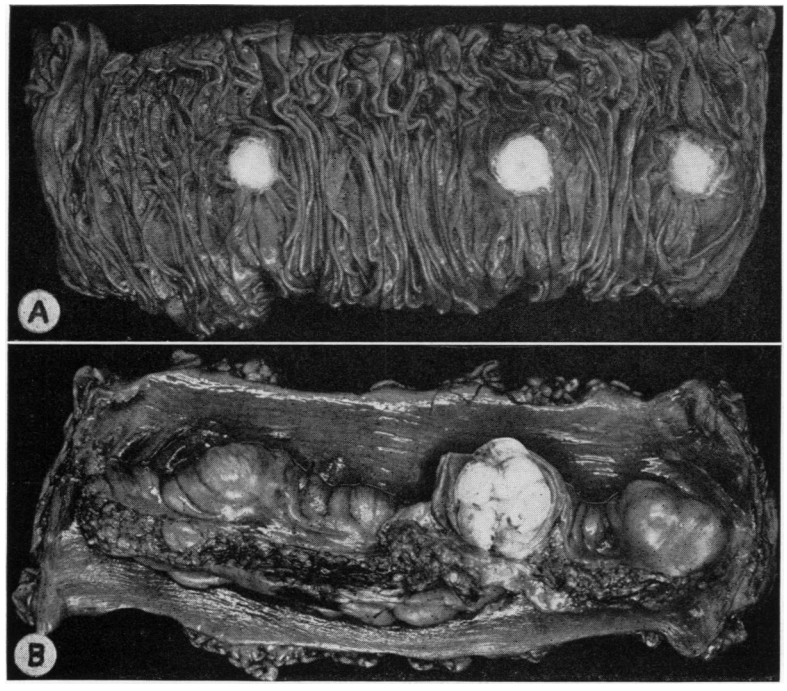

FIG. 2.—(Case 1). Mucosal surface (A) and serosal surface (B) of the opened segment of resected jejunum showing the location of three of the four diverticula, which have been filled with absorbent cotton. In "B" one of the pouches has been opened.

Benson, Dixon, and Waugh found records of 122 cases in the files of the Mayo Clinic for the period 1909 to 1942 inclusive; of these cases, in 85 the condition was found at necropsy, all but one incidentally, in 21 it was seen during the course of an abdominal operation, and in 16 it was detected on roentgen examination. In 6847 persons who underwent roentgenologic examination of the gastro-intestinal tract, Case found 85 diverticula in the duodenum and only five in the jejunum (in one of these the ileum in addition was involved). Rankin and Martin reported three instances of jejunal diverticulosis disclosed in a series of 956 exam-

inations of the small intestines at the Mayo Clinic. Most authors seem to agree that the actual incidence is greater than that reported, and this idea seems logical enough in view of the great difficulty one frequently encounters in locating the diverticula, both at necropsy and at operation. Thus, Rosedale found only three cases recorded in 5000 necropsies, but when special effort was made, including inflation of the bowel, four cases were found in the next 300 postmortem examinations.[17] The occurrence of hemorrhage from such an unusual lesion again is exceedingly rare, and the cases in which it has been reported, along with pertinent data, are represented in Table I. These are the cases usually cited as representing hemorrhage from a jejunal diverticulum, but as a glance at this table will show, several of these are included on rather slim evidence.

In an effort to shed further light on the frequency of occurrence of hemorrhagic-jejunal diverticulitis, the records of the Mayo Clinic for the ten years 1940 to 1949, inclusive, were studied, and all cases of non-meckelian diverticula of the small intestine in which an operation was performed for one reason or another were enumerated; among these, we were able to find 21 cases in which there were such lesions in the jejunum. These did not include a number of cases in which a diagnosis was made by roentgen rays only. Of these 21, only two presented any evidence of hemorrhage or anemia. In both cases surgical exploration was carried out; in one case, the involvement of jejunum was not considered significant, as no gross reaction could be seen at the time of operation, while in the other, the surgeon found it impossible to locate the sacs, which had previously been seen in

TABLE I.—*Cases Usually Cited as Those in Which Hemorrhage From Jejunal Diverticula Occurred.*

Author	Age, Sex	History	Jejunal Lesion	How Found	Hemoglobin, Per Cent	Red Cells, Millions	Pathologic Report	Follow-up
Braithwaite (1923)	59 M	Vomited 2 pt. blood	Multiple diverticula	Operation (resection)	?	?	None	Well (2 mo.)
Harrtung* (1926)	66 F	Melena for 3 da.	Single diverticulum	Operation (resection)	"Secondary anemia"		No mention of ulcer	Well (2 mo.)
Somerford (1930)	7 mo. M	Melena for 2 mo.	Reduplication	Necropsy	20	1.75	No ulcer or bleeding point seen	
Tengwall (1931)	49 F	Weakness, dyspnea, for 6 mo.	Multiple diverticula	Operation (resection)	35	2.3	No mention of ulcer or bleeding point	Well (7 mo.)
Jones and Crile (1936)	40 F	"Red blood in stools 3 or 4 times"	Multiple diverticula	Operation (excised larger ones)	"Normal"		No mention of ulcer	Improved (6 mo.)
Guthrie and Hughes (1937)	54 M	"Several large hemorrhages from bowel"	Multiple diverticula	Operation (resection)	?	3.7	None	Well (4 mo.)
Normark (1938)	2 M	Melena for 22 mo.	Reduplication (?)	Operation (resection)	30	3.97	Ulcer in bowel near diverticulum	Well (15 mo.)
Klidjian (1946)	73 M	Large rectal hemorrhage (shock)	Multiple diverticula	Operation (resection)	52	?	One diverticulum discolored, contained sharp concretion. No ulcer seen	Well (6 mo.)
Kozoll *et al.* (1950)	64 M	Melena (passed 16,600 cc. blood)	Multiple diverticula	Operation (resection)	9 Gm.	2.65	Definite ulcer	Well (15 mo.)
Orr and Russell (1951) (case 8)	71 F	Hematemesis and melena	Multiple diverticula	Operation (resection)	?	?	No ulcer seen	Well (time?)
Orr and Russell (1951) (case 10)	54 M	Hematemesis	Multiple diverticula	Operation (resection)	?	?	No ulcer seen	Well (time?)

* This name has been repeatedly misspelled "Haltung" throughout the literature for many years.

the roentgenograms. In this connection also, the paper by Benson and co-workers contained no mention of a hemorrhagic lesion in the 122 cases they reported from the Mayo Clinic for the period prior to 1943. Reports of the two cases just mentioned follow.

Case 2.—A 74-year-old white man was first seen on August 7, 1949, with a history of having been well until 4 days previously, when he passed "3 or 4 quarts" of bright red blood by rectum while straining at stool. He had been transfused with 6 pints of whole blood by his local physician and had had no further rectal bleeding until the night before admission, when another "large hemorrhage" had occurred. Complete gastro-intestinal roentgenologic studies gave negative results except for a single diverticulum in the jejunum, 3 cm. in diameter. On surgical exploration no diverticulum could be located in spite of careful palpation of the entire small bowel three times.

This illustrates the difficulty of locating these lesions by palpation alone, and points up the importance of distending them as carried out in Case 1.

Case 3.—A 61-year-old white man was first seen on December 21, 1948, with a complaint of "weakness and black stools" for 2 weeks. The value for hemoglobin was 5.6 Gm. per 100 cc. of blood, and the erythrocytes numbered 2,340,000 per cubic millimeter. The relevant positive findings were hypochromic anemia, a small esophageal hiatal hernia, and roentgenologic evidence of several jejunal diverticula. The gastro-intestinal tract was otherwise roentgenologically normal. Esophagoscopy and gastroscopy revealed no evidence of upper gastro-intestinal ulceration or varices. The stools were positive for occult blood by both benzidine and guaiac tests. Consequently, after adequate transfusion, surgical exploration was carried out to locate the source of the blood loss. Examination of the abdominal contents disclosed no abnormalities except for "30 or 40 small jejunal diverticula." As there was no evidence of inflammatory or other reaction around them, the surgeon felt that they could not be a source of bleeding and thus left them undisturbed.

From our study of this subject it would now seem that under similar circumstances it might be wise to resect the involved bowel, in the absence of other obvious source of blood loss.

COMPLICATIONS

The commonly recognized complications of diverticulosis of the small intestine are perforation, diverticulitis, obstruction (from inflammation, distention of the sac, adhesions, or volvulus), and hemorrhage. Benson and co-workers added two less common ones in their thorough discussion: foreign bodies (bones, parasites, and so forth), and neoplastic change.

It might be expedient here to discuss the relationship of the complications to the symptomatology in this condition. In the numerous publications on this subject all shades of opinion are represented as to the role this lesion plays in the production of symptoms. Many references are found to the "flatulent dyspepsia" which has led to the discovery of the diverticula. However, several factors cast considerable doubt on the premise that diverticulosis *per se* causes any real symptoms in the absence of complications. Among these are the similarity of this "gas and bloating" to the "irritable bowel syndrome" which is ordinarily unrelated to organic disease, and the failure of this syndrome to respond favorably to the surgical extirpation of the involved bowel.

The symptoms resulting from the complications are nonspecific, since the symptoms would be the same regardless of the origin of the complicating conditions. This is true of the complication with which we are now primarily concerned (hemorrhage), so that if the diverticula are not visualized roentgenologically, or if roentgen examination is not considered advisable, the patient will come for an exploratory operation with a diagnosis of gastro-intestinal hemorrhage or hypochromic anemia and the source of the bleeding will be found only when the abdomen is opened and the bowel thoroughly examined by an alert surgeon. The diverticula may well be missed by a casual examination of the entire length of the small intestine, as is well

illustrated by two of the cases reported herein.

PATHOLOGY

The pathology of the diverticula themselves has been well covered elsewhere.[6] We would naturally expect ulceration to be the lesion associated most often with blood loss from these structures, but oddly enough this has been true in only two reported cases,[11, 13] and many characteristics of Normark's case make us wonder if it may not have been a case of aberrant Meckel's diverticulum. Careful search by the pathologist in our Case 1 did not reveal any gross or microscopic ulceration. It would be logical to expect to find gross lesions, when present, associated with rapid massive hemorrhage rather than with slow loss of blood. We have been able to find only one reported instance[13] in which heterotopic tissue, such as pancreatic cells or gastric mucosa, was present in a case in which there was clinically significant hemorrhage, although Benson and co-workers had one case in which pancreatic tissue was present in a jejunal diverticulum. It is our opinion that these are cases of high Meckel's diverticulum. Thus, the immediate cause and the pathogenesis of the bleeding are so far only speculative.

Tearing of blood vessels by distention of the sac with bowel contents has been mentioned as a cause of bleeding from jejunal diverticula. Trauma that produces submucosal hemorrhage, which in turn might cause mucosal necrosis and then ulceration, was postulated by Kozoll and co-workers, in whose case there was a very suggestive relationship to a humeral fracture following a fall. The bleeding in Klidjian's patient seemed most likely to be due to a stone-hard, sharp-pointed concretion contained in the pouch. If a well-defined pathogenetic basis existed, perhaps this complication of hemorrhage would occur more commonly; its very rarity is further evidence of the more or less accidental causation, which is suggested by the variety of factors in the reported cases. Rapid healing and interval surgery may explain the lack of pathologic evidence of a bleeding site.

FIG. 3 FIG. 4 FIG. 5

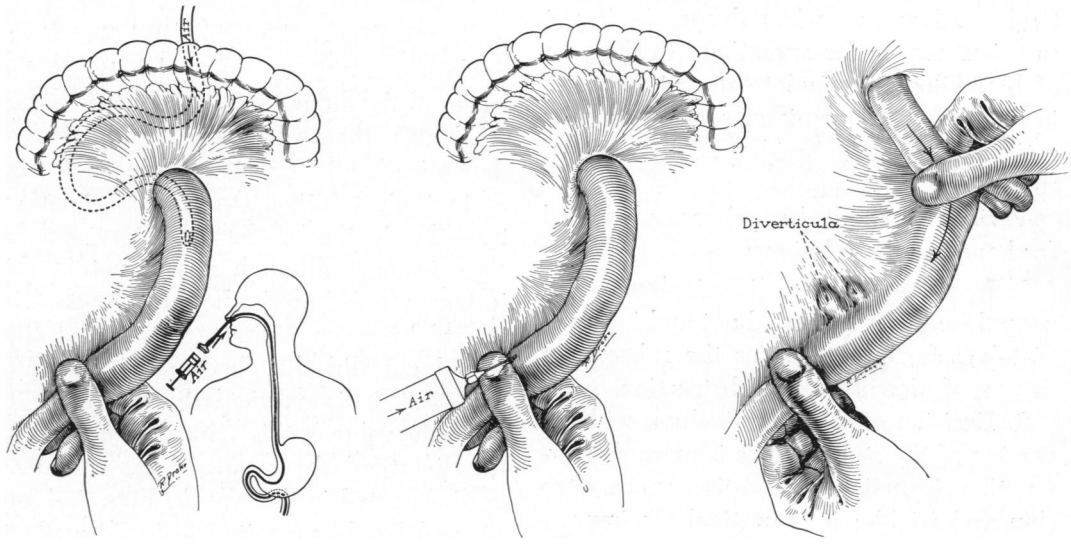

FIG. 3.—Intestinal tube with tip at the ligament of Treitz and extending just into the jejunum.
FIG. 4.—In absence of a tube, air can be injected into the jejunum by syringe and needle.
FIG. 5.—The jejunum is stripped, with closure at a distal point, in order to distend diverticula with air.

TREATMENT

Once a diagnosis of diverticulosis of the jejunum is arrived at, a choice between medical and surgical therapy must be made. Medical measures, consisting of the use of mild laxatives, eating of soft foods, and postural drainage, must be viewed with some reservations as to their effectiveness. However, resort to surgical intervention should be delayed until the indications are clear cut, as the mere presence of the diverticula and more or less dyspepsia is not sufficient reason for operation. In the case of blood loss, surgical treatment becomes justifiable when anemia of some degree, or active bleeding, as evidenced by melena, supervenes.

Various methods have been advocated for handling these lesions:

1. Inversion of the diverticulum. This procedure is easily carried out and is immediately safe, but it carries the subsequent danger of ulceration and obstruction.

2. Excision of the diverticulum, with inversion of the stump. This is more dangerous than inversion, as the tissues are often thin and friable; it is particularly impracticable when the pouches are multiple, as is usually the case.

3. Short-circuiting the involved site by means of entero-enterostomy. This would be advisable only when for some reason resection is not possible.

4. Resection of the affected bowel with end-to-end jejunojejunostomy. This is the treatment of choice. The principal contraindication to this method of management is involvement of all or of the major portion of the small intestine by diverticula. In such cases the dangers of extensive resection, with the postoperative disability, must be weighed against the probable dangers of leaving the patient with the lesions which prompted the exploration. Under these circumstances, a compromise might be effected by removing only the worst portion of the bowel, that is, the part containing the largest diverticula, or, if examination discloses which of the pouches are causing trouble, removing the segment in which they lie.

In this connection it would be well to add a word of warning about the difficulty of finding these diverticula. They may be very prominent if the walls are thickened from past and present inflammation or if they are distended with bowel contents, and thus they may be hard to overlook. However, as in two of our cases, when the walls are thin and pliable and the diverticula are empty and located between the leaves of the mesentery, it is no easy matter to identify them. In Case 1, palpation of the entire small intestine twice gave no hint as to their location. In such a situation, transillumination of the mesentery may be of assistance, although it was not in this instance; only when the bowel was distended by compressing segments that contained gas did they become visible. We feel that it is particularly important to stress the value of this maneuver of inflating the diverticula by means of gas contained or air inserted in the bowel (Figs. 3, 4 and 5), and would urge its use in all explorations of the bowel for indeterminate gastrointestinal hemorrhage, with the belief that it will decrease the incidence of negative results on exploration for "idiopathic hemorrhage." While we feel that this maneuver may not be new with us, we were unable to find any mention of its use in a rather thorough search of the publications in the English language.

COMMENT

Hypochromic anemia remains as the single most common form of anemia in the United States, and indeed, as one of the most common problems encountered in the practice of medicine. To classify a case of hypochromic anemia as "idiopathic" and to rely entirely upon symptomatic treatment is entirely unjustifiable without careful and thorough study of the patient and, in

particular, thorough study of the gastro-intestinal tract. Recently, Moore urged that the term "idiopathic hypochromic anemia" be deleted from medical literature, substituting instead "hypochromic anemia of undetermined etiology," indicating that all iron-deficiency anemias eventually prove to be the result of some demonstrable organic lesion. This is one of the principal reasons which prompted us to report the cases included herein.

SUMMARY

1. Hypochromic anemia is one of the most common conditions that the practicing physician is called upon to treat. Diverticulitis of the jejunum is another significant source of gastro-intestinal hemorrhage that produces hypochromic anemia, but it is not generally recognized as such. Anemia from this cause is rare, but we believe that it occurs more commonly than it is diagnosed.

2. The presence of jejunal diverticula is often difficult to demonstrate at the time of operation. The procedure of distending the diverticula by compressing and milking segments of the bowel that contain, or into which is injected, gas or air, as carried out in Case 1 by us, offers a further means of identifying them, and no abdominal exploration for gastro-intestinal hemorrhage or hypochromic anemia should be considered as productive of negative results until this maneuver has ben employed throughout the length of the intestine.

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DISCUSSION.—DR. OWEN H. WANGENSTEEN, Minneapolis, Minn.: Diverticula in the jejunum may be multiple. I recall having operated on a man named T———, University Hospital No. 642065, who had a picture that is familiar to all of you—snowball retention of barium in diverticular pockets. In 1926, when I did a gastrojejunostomy for an obstructing duodenal ulcer for this man, several diverticula were noted in the mesentery of the jejunum. They were small and I did not believe