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TRANSPYLORIC PROLAPSE OF GASTRIC MUCOSA

W. P. KLEITSCH, M. D. AND R. L. LAWTON, M. D., Omaha, Nebraska.

PROLAPSE OF gastric mucosa through the pyloric canal is the result of hypertrophic gastritis of the antral mucosa. The hypertrophied rugae are forced into the pylorus by gastric peristalsis and may produce varying degrees of obstruction. The clinical picture, therefore, is that of chronic gastritis upon which are superimposed the symptoms of an inconstant pyloric obstruction. This combination of symptoms is suggestive of peptic ulcer, and many of these patients are so considered and so treated.

The radiologic appearance of the lesion is inconstant. It varies with the amount of mucosa prolapsed and with the degree of obstruction produced. When a moderate amount of mucosa has prolapsed through a relatively normal pylorus, a filling defect is produced in the base of the duodenal cap. This has been described as the "toadstool" or "umbrella" sign and is considered to be pathognomonic when present (Fig. 1). It is possible, however, to have such a high degree of obstruction, due to complete filling of the pyloric canal with prolapsed mucosa, that the duodenal cap will not visualize. In this instance the pyloric antrum has a pinched off appearance which might be called a "funnel" sign (Fig. 2). A third radiographic picture is formed when compensatory dilatation of the pyloric canal occurs as a result of stretching of the Sphincter muscle. This results in the prolapse of relatively large amounts of antral mucosa into the duodenum. The roentgenogram in this instance shows a dilated pylorus with evidence of gastric rugal pattern extending into the base of the duodenal cap (Fig. 3). This type of picture is often seen in individuals who are asymptomatic.

The difficulty of establishing the correct diagnosis is a burden not only for the internist and radiologist. It is possible for the surgeon to overlook the condition even at laparotomy. Palpation of the pylorus is inconclusive; although the hypertrophied mucosal folds are palpable as a vaguely outlined thickening in the antrum, it is usually difficult to decide whether or not it really

From the Surgical Service of the Veterans Hospital and the Creighton University School of Medicine, Omaha, Nebraska.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are a result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Submitted, Sept. 4, 1952.

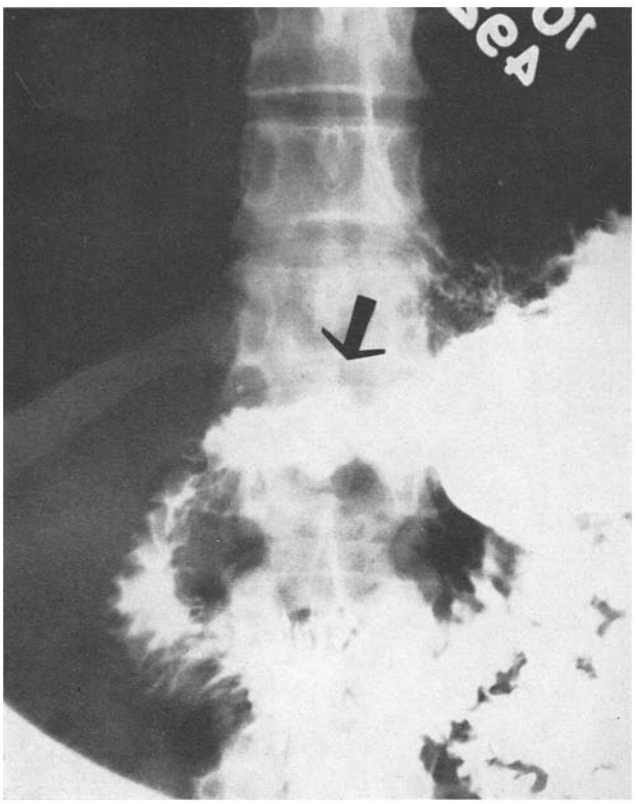

Fig. 1: A roentgenogram of the pylorus showing the typical "toadstool" or "umbrella" sign of prolapsed gastric mucosa (Case No. 1).

represents pathology. When a dilated pyloric muscle is present, it offers an excellent indication for gastrotomy. A dilated pylorus in the absence of duodenal obstruction is almost certainly due to prolapse of a gastric polyp or of antral mucosa. Slight thickening palpable in an otherwise normal appearing antrum and in the absence of other pathology is sufficient evidence to do a gastrotomy for confirmation of the diagnosis.

The treatment of the condition should be kept as simple as possible. A trial of medical management is in order. If this does not afford relief, or if symptoms progress, laparotomy should be resorted to. The usual course of events is for the patient to undergo medical treatment for vague epigastric distress until an upper G. I. series is done. The findings of the radiologist are frequently misinterpreted; a malignant lesion is often suspected and laparotomy is recommended. In the face of such a story the surgeon is well advised to palpate the pyloric antrum most carefully—especially if he finds it to be normal in appearance. A gastric resection is not indicated for prolapse of gastric mucosa and should be avoided. When doubt exists, the best procedure to do is a 2 cm. gastrotomy in the anterior surface of the pyloric antrum immediately orad to the pylorus. If the symptoms are due to transpyloric prolapse of the gastric mucosa, the offending, hypertrophic rugal folds will be easily demonstrable. With the diagnosis established the gastrotomy incision can then be lengthened caudad to transect the pyloric muscle, and the defect can be closed transversely (Heineke-Mikulicz pyloroplasty). This is a relatively minor surgical procedure which relieves the obstructive symptoms with a minimum derangement of gastrointestinal physiology.

CASE REPORTS

Case No. 1, H. E. R.—A 39-year-old white male patient was admitted to the hospital for the treatment of headache, chills and fever accompanied by vomiting and epigastric discomfort. Although his fever subsided, the nausea and vomiting persisted, and at time of admission he was unable to retain either liquids or solids. He said that he had been treated for duodenal ulcer for the past seven years. His symptoms were recurrent, and he had been hospitalized several times for treatment.

Physical examination revealed a well-nourished and well-developed adult male of the stated age who was apparently suffering with severe abdominal pain, lying in bed with his knees flexed on his abdomen. He was vomiting at the time of examination, and there was marked tenderness in the mid-epigastrium. The liver was tender on percussion, and its edge was palpable at the costal margin on deep inspiration. There was no muscular rigidity and no distention. The laboratory data revealed a leucocytosis of 13,700; 4.67 million red cells and 16.1 grams of hemoglobin. The urinalysis and serology were negative. The serum amylase was 81 mg. percent, and a gastric analysis revealed 59 units total acidity. A flat plate of the abdomen revealed no pathology, and a routine chest plate was normal. A G. I. series did not suggest any specific pathology to the radiologist. The umbrella sign, however, could be demonstrated in retrospect (Fig. 1).

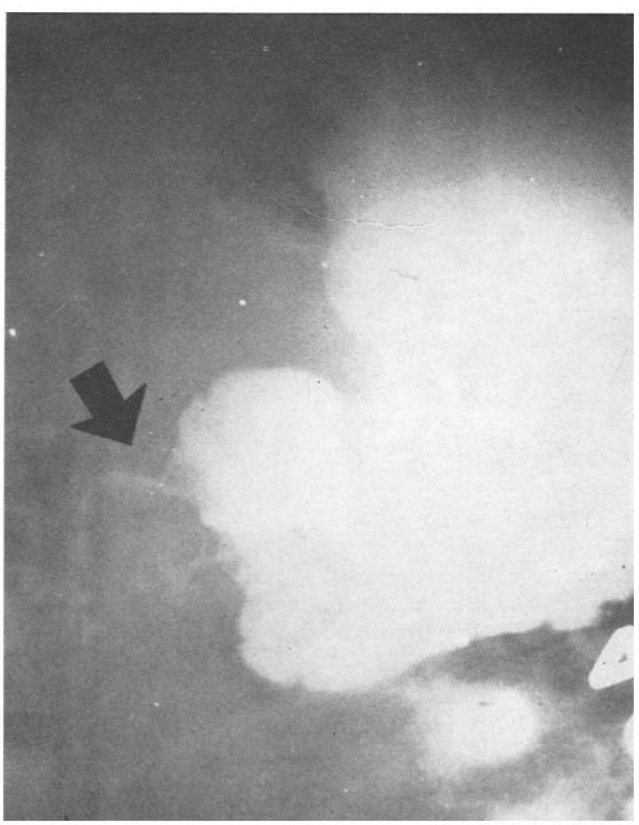

Fig. 2: Almost complete pyloric obstruction, indicated by the "funnel" sign, due to prolapse of gastric mucosa (Case No. 2).

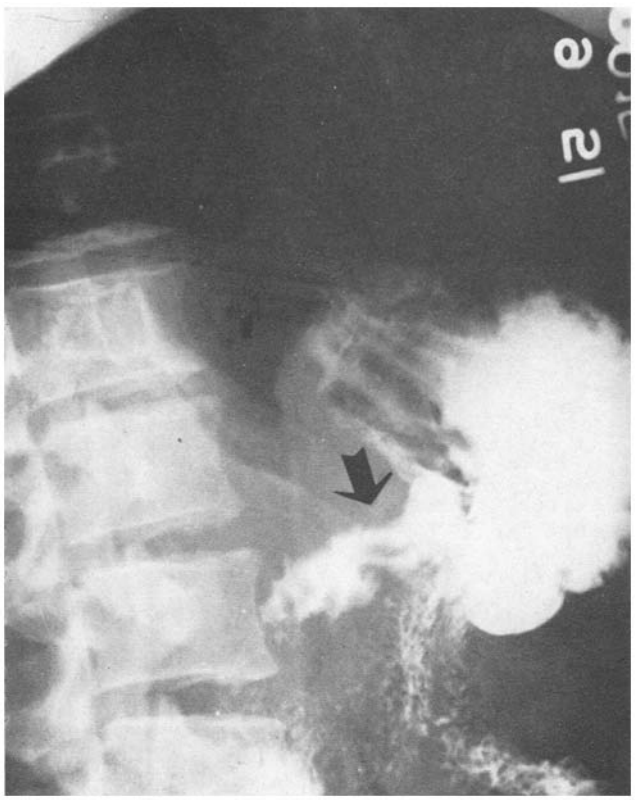

Fig. 3: Transpyloric prolapse of massive amounts of gastric mucosa carrying the gastric rugal pattern into the pyloric canal. Note the longitudinal striations in the pyloric canal (Case No. 3). The gastric rugal pattern is accentuated throughout the stomach.

Because of the pyloric obstruction and the history of chronic duodenal ulcer he was subjected to a laparotomy. At operation the abdominal exploration was negative with the exception of a questionable amount of thickening in the region of the pylorus. A gastrotomy was felt to be indicated, and a longitudinal incision was made through the pylorus extending for approximately 1 cm. into the duodenum and 1 cm. into the antrum. When the incision was opened, it became quite obvious that the obstruction was due to hypertrophic gastric mucosa which could be demonstrated hanging through the pylorus. A Heineke-Mikulicz pyloroplasty was done. The postoperative course was completely uneventful, and symptomatic relief was complete.

Case No. 2, J. C. T.—A 57-year-old white male admitted to the hospital for treatment of an undiagnosed intestinal condition. He gave a rather incoherent story of abdominal difficulties of two years duration which began with an emotional upset occasioned by the death of his wife, brother and father within a period of eight months. His chief complaint was recurrent epigastric pain occurring most commonly toward evening and usually right after a meal. The pain was described as a dull ache localized to the mid-epigastric region. Occasionally he had obtained relief from the pain by vomiting. There had been a weight loss of 45 pounds in the past two years.

Physical examination revealed a well-developed, under-nourished white male of the stated age who was ambulant and appeared chronically ill. Physical examination was not particularly helpful. The abdomen was scaphoid, and high in the mid-epigastrium was an area of tenderness. The laboratory data revealed a normal urinalysis and serology; a leucocytosis of 14,000; a red count of 5.04 million, and a hemoglobin of 14.6 gms. A gastric analysis revealed a total acidity of 96° and 82° of free acid. A routine x-ray of the chest revealed no significant disease. The gallbladder visualized and emptied normally. A G. I. series was reported as discovering an ulcerated lesion of the antrum with a question of carcinoma. These x-rays were repeated and reported as, "Spasm and deformity about the pyloric antrum. . . . a definite conclusion regarding the question of neoplasm cannot be made at this time. I believe there is definite ulceration and that it is on the gastric side of the pylorus. I believe that there is still a strong possibility of malignancy. . . ."

Because of a possible gastric malignancy the patient was prepared for operation. At laparotomy, exploration revealed a fibrous thickening of the pancreas; careful examination of the gland revealed no tumor nodules. The pylorus of the stomach was found to be somewhat thickened, and there was scarring of the first portion of the duodenum indicating an old healed ulcer. The thickening in the region of the pyloric antrum could not be definitely interpreted by palpation, but because of the evidence of duodenal ulceration a gastric resection was decided to be indicated. An anterior isoperistaltic Polya type of anastomosis was done. Examination of the gross specimen of stomach revealed the pathology to be a ring of gastric mucosa which was prolapsed through the pylorus. The prolapsed mucosa was found to be hyperemic, thickened and formed a pseudovalve approximately 1 cm. long (Fig. 4). The histopathology showed atrophy and thinning of the mucosa with a heavy infiltration of lymphocytes (Fig. 5).

Case No. 3, H. C. H.—A 57-year-old white male was admitted to the hospital for treatment of a peptic ulcer. He complained of epigastric distress of three years duration. This usually came on one or two hours after eating and especially after the noon meal. It was described as usually being a feeling of hunger

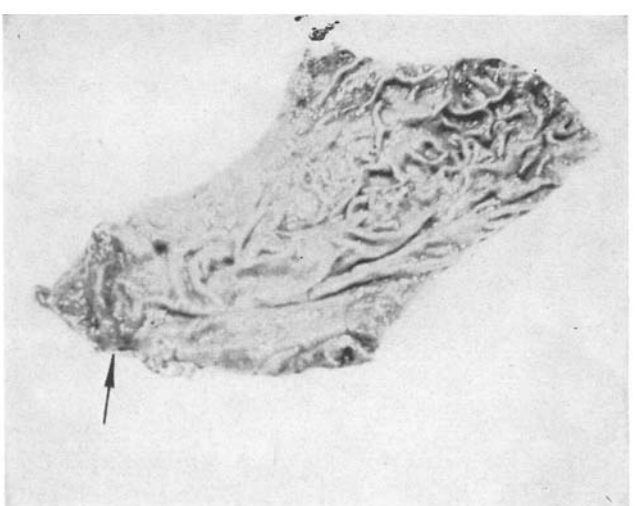

Fig. 4: The gross appearance of prolapsed gastric mucosa in a section of stomach resected for duodenal ulcer (Case No. 2). The arrow indicates the hyperemic fold of mucosa which was obstructing the pylorus.

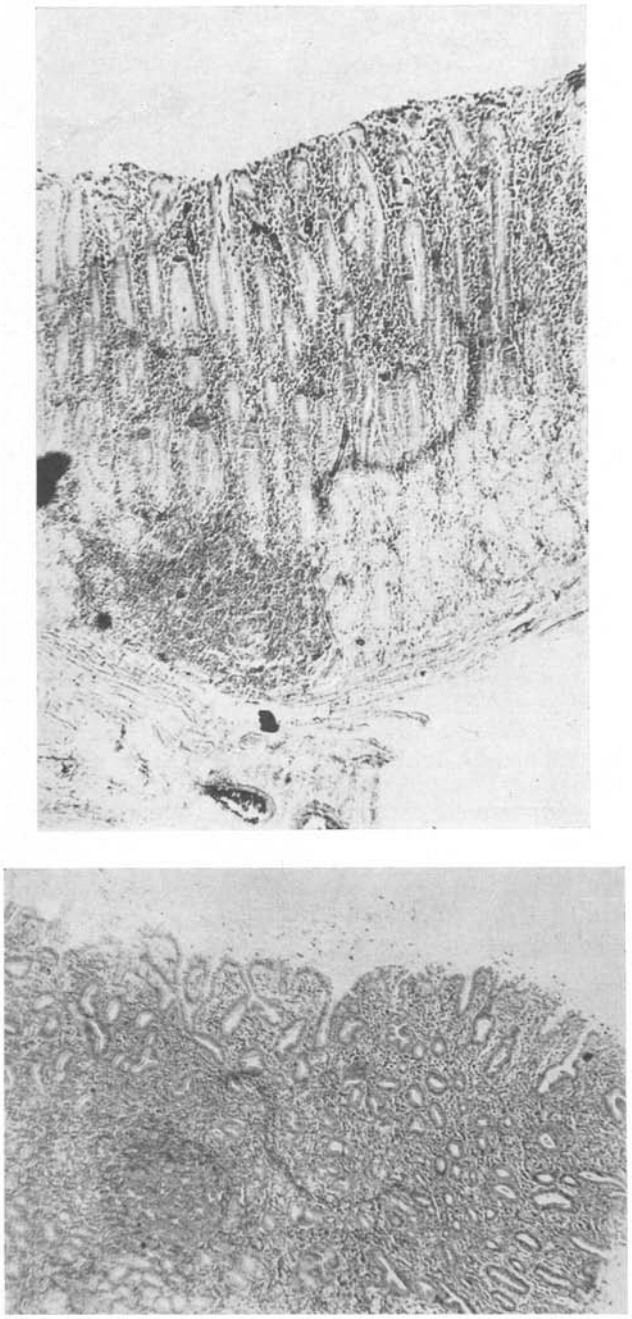

Fig. 5: Photomicrographs of the prolapsed mucosa of Cases 2 and 3. There is considerable polymorphonuclear infiltration but no ulceration. Lymphocytes are abundant also and are forming germinal centers.

but occasionally as a sharp pain. Milk and antacids always seemed to relieve his distress.

On physical examination a well-developed, well-nourished, tall and rather thin patient was described. There were no unusual findings elicited; the abdomen was flat and non-tender. The laboratory examinations were within normal limits with the exception of a G. I. series which showed coarsening of the rugal pattern of the stomach. A gastroscopic examination confirmed the presence of hypertrophic gastritis. He was placed on a Sippy regimen, became symptom free and was discharged with advice to continue his ulcer treatment.

He was readmitted about a month later, having had a recurrence of the pain. Another G. I. series suggested the presence of a gastric ulcer which was reported as ". . . questionable ulcer middle third, lesser curvature of stomach. Marked hypertrophy of the gastric mucosa."

Because of the persistence of a gastric ulcer the patient was subjected to laparotomy. Exploration of the abdomen was completely negative with the exception of some polyps in the sigmoid colon and a dilated pylorus. Careful examination of the duodenum failed to reveal an obstruction or other cause for dilatation of the pyloric ring. A soft, vaguely outlined mass was palpable in the pyloric antrum which could be prolapsed through the dilated pyloric ring by manipulation. It was thought that this might be a polyp, and therefore the pylorus was opened by means of a longitudinal incision. This revealed large, hypertrophic, gastric rugae which could be demonstrated prolapsing through the pyloric canal. A biopsy of the mucosa was taken, and the longitudinal incision was closed transversely (Heineke-Mikulicz pyloroplasty). Treatment of the sigmoid polyps was deferred because the bowel had not been prepared for resection.

The histopathology of the mucosal biopsy was as follows: "These sections show a covering of this tissue with a reasonably normal appearing gastric type mucosa. The muscularis mucosa is present in the central portion of the polypoid mass and is partially infiltrated with lymphocytes. There are focal areas of collections of lymphoid tissue into the germinal follicle pattern. Through the mucosa there is a scattered polymorphonuclear cell infiltration which in some areas is reasonably intent. The epithelium lining the glands is quite normal in appearance. (Fig. 5).

DISCUSSION

Transpyloric prolapse of gastric mucosa is regarded by some as a physiological variant of the normal. Most clinicians, however, feel that at least in some cases symptoms are produced by prolapsing gastric mucosa. In the cases here reported it would be difficult to say that the symptoms were not produced by the prolapsed mucosa since relief after operation was so positive.

There is undoubtedly a pronounced personal variation in the interpretation of the radiological examination. In our experience the diagnosis is rarely if ever made by the radiologist. Confusion of the radiologic findings with those of cancer brought our cases to laparotomy. The diagnosis was made at gastrotomy, and the roentgenograms were correctly interpreted in retrospect. From this one could deduce that a radiologic diagnosis alone, of prolapse of the gastric mucosa, is insufficient evidence for surgical exploration. If exploration is indicated because of symptoms, the surgical procedure should be as simple as possible. Mutilating operations are to be avoided. A procedure such as the Heineke-Mikulicz pyloroplasty is adequate. By enlarging the pyloric canal and dividing the muscle it relieves the obstruction and prevents strangulation. The treatment of the accompanying gastritis is medical.

SUMMARY

The importance of transpyloric prolapse of gastric mucosa as a source of symptoms is reviewed. The difficulties encountered in arriving at this diagnosis are de-

scribed. Mutilating operations are not felt to be indicated. Three cases are reported.

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AN UNDERSTANDING OF PROLAPSED GASTRIC MUCOSA

FRANCIS W. WILSON, LT. COL., USAF (MC)*, San Antonio, Texas.

FEW DISEASES of the gastrointestinal tract have occasioned more controversy than has the relatively recently described condition of prolapsed gastric mucosa into the duodenum. Autopsy (1) and surgical (2, 3, 4) findings, in addition to the characteristic x-ray findings (5, 6), leave no doubt as to the actual anatomical entity; but the clinical significance of such herniated folds poses quite a problem.

THE NATURE OF THE DISEASE

The first factor in the understanding of this disease is the realization of just what the disease represents. For practical purposes the prolapsed gastric folds represent nothing more than herniated gastric folds into the duodenal bulb. These may vary from single to multiple folds or even to a prolapse of the circumference of the stomach in the antral area. Regardless of the etiology, and the causes are undoubtedly multiple, the condition results when any mass, whether it be an enlarged or displaced rugal fold or even a neoplasm (15), is presented at the pyloric canal and passes through it. The gastric hyperperistalsis consistently associated with prolapsed gastric mucosa may be primary or secondary. The primary type occurs in those individuals whose psychogenic gastrointestinal disturbances result in their having gastrointestinal hypermotility in general. In these individuals, the gastric hypermotility probably precedes the prolapse, and is a definite aggravating factor in the production of the disease. In the secondary type the hypermotility probably was not present before the prolapse was established, and in these individuals it is probably a compensatory mechanism to propel the gastric contents past the variable degree of pyloric canal obstruction resulting from the presence of the gastric folds in the canal.

As is true of any hernia, this herniation of gastric folds may be reducible or non-reducible. Complete or relative reduction of the herniation probably can result from external pressure (6, 7), from reduction in edema of the prolapsing folds (1) and from reduction in the degree of congestion or inflammation of the folds (7, 8). Aggravating factors include edema (1) of the gastric mucosa, gastritis (8), the occasionally reduced lumen of the pyloric canal (2) and gastric hyperperistalsis (5, 6, 14); and amelioration of one or more of these tends toward spontaneous reduction of the herniation. On the other hand, little or no reduction in the degree of prolapse can be anticipated if the basic etiological factor or factors are not materially corrected or if the volume of the herniated folds be great, as was case No. 5 reported by Patterson and his associates (9).

To consider such a condition a physiological variant of normal (10) is the equivalent of considering an inguinal hernia to be physiological.

SIGNS AND SYMPTOMS

The signs and symptoms of prolapsed gastric mucosa have been carefully observed and reported by numerous authors (5, 6, 11, 14), but a brief discussion of the cause and significance of such is in order. Vomiting is a rather frequent finding in patients presenting prolapse of the gastric mucosa. Fluoroscopic findings in these patients tend to rule out reversed gastric gradients (12) as a cause. The most probable cause is a relative blockage of the pyloric canal by the herniated folds, possibly aggravated by some degree of pylorospasm. Reduction in the "swelling" of the folds, reduction in the degree of pylorospasm (by anti-spasmodics, sedation, psychotherapy, etc.) or the regular ingestion of soft foods which pass such a block easier tend to reduce the degree of obstruction and thus reduce or stop vomiting.

If there is a break in the mucosa of the prolapsed folds, the action of hydrochloric acid on the defect will give pain the same as it would were the fold in its normal position in the stomach. Should the break in the continuity of the mucosa be of such location anatomically as to involve blood vessels, bleeding can result readily; and no doubt bleeding is encouraged by the relative interference with venous return to the base of the fold as the result of pressure imposed by the pyloric canal.

Most workers analyzing the symptoms associated with prolapsed gastric mucosa have been impressed by the vagueness and the inconstant pattern of the symptoms. This is not unduly remarkable when one considers that diseases of the gastrointestinal tract in general are notorious for their vague and indefinite symptoms. The stomach, in which even carcinoma may be silent until it is far advanced or in which peptic ulceration tends to be more vague symptomatically than when it is present in the duodenum, is the site of origin of the prolapsed folds and so is the source of innervation. Thus it is not surprising that the symptoms of prolapse tend to be vague and of no definite

Submitted Aug. 26, 1952.

*Chief of Medical Service, U. S. Air Force Hospital, Lackland Air Force Base, San Antonio, Texas.