Reflux Gastritis Following Gastric Surgery

THEODORE DRAPANAS, M.D., MORRISON BETHEA, M.D.

From the Department of Surgery, Tulane University School of Medicine and the Tulane Division, Charity Hospital of Louisiana, New Orleans, Louisiana

Reflux gastritis is a distinct clinical entity produced by the reflux of duodenal and proximal intestinal content into the stomach following operations which create a stoma between the stomach and proximal intestine or following pyloroplasty. In 24 patients this clinical pattern was documented and these findings were supported by gastroscopic demonstration of reflux and the microscopic pattern of reflux gasritis. Diversion of proximal intestinal content from the stomach in such patients uniformly led to relief of symptoms with disappearance of gastritis and postoperative weight gain. The possible role of bile, pancreatic juice and acid as an explanation for the mechanism of this entity is presented.

With the increasing utilization of fibroptic gastroscopy and with improved methods for determination of gastric mucosal function, there has occurred within recent years an increased awareness of reflux gastritis. For many years it has been recognized that gastritis to a varying degree may occur in any procedure which bypasses the pylorus or in which the pylorus is rendered incompetent. Nevertheless, a number of investigators have begun documenting during the past two decades the possible deleterious effects of reflux of bile and/or pancreatic juice into the stomach following such procedures.[1,8,13–15,17,20,21,23,27,29,31,33–35]

In reviewing a large series of patients who had undergone gastric procedures at Charity Hospital, we became impressed with a small but significant group of patients who demonstrated a distressing symptom complex of weight loss, vague, diffuse epigastric pain, and nausea with emesis which was occasionally mixed with bile. Most of them had multiple hospital admissions. In almost every instance their gastric juice was achlorhydric in both the fasting sample and following histamine stimulation. The impressive pattern of diffuse gastritis present on gastroscopy made us re-examine carefully their problems and the present report represents an extensive study of the 24 patients in whom the diagnosis of reflux gastritis was made and surgical intervention was undertaken during the past five years (1968–1973).

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Mailing Address: Theodore Drapanas, M.D., Department of Surgery, Tulane University School of Medicine, 1430 Tulane Avenue, New Orleans, Louisiana.

Clinical Material

Of the 24 patients who have undergone surgical correction for reflux gastritis, 17 were males and 7 were females. They ranged in age between 33 and 70 years. All patients' initial surgery was for peptic ulcer disease and all had operations which either bypassed or created an incompetent pylorus. Five of these patients had as their original procedure a bilateral truncal vagotomy, antrectomy and gastroduodenostomy (Billroth I); eight had a bilateral truncal vagotomy, antrectomy and gastrojejunostomy (Billroth II); seven had a subtotal gastrectomy and gastrojejunostomy without vagotomy; two had a bilateral truncal vagotomy and pyloroplasty; and the remaining two had a simple gastrojejunostomy without vagotomy.

All of the patients except one presented with diffuse epigastric pain and associated nausea and vomiting (Table 1). Nineteen had bile present in the emesis which was not necessarily associated with meals. Nineteen patients also presented with chronic weight loss ranging from 5 to 60 pounds with a mean of 15 pounds. Nine patients had some evidence of upper gastrointestinal bleeding, two of whom had massive hematemesis requiring immediate transfusion and admission to the surgical service. Three patients in addition to reflux gastritis had dysphagia due to associated reflux esophagitis.

The interval between initial surgery for peptic ulcer disease and the onset of symptomatic reflux gastritis ranged from two months to almost 20 years. All patients demonstrated either a histamine fast achlorhydria or a hypochlorhydria on gastric analysis. Upper gastrointestinal x-rays were uniformly within normal limits except in one patient who demonstrated an ulcer on the gastric side of a gastrojejunostomy. This patient was also achlorhydric. Six patients had a microcytic, hypochromic anemia secondary to chronic gastric hemorrhage. All patients were gastroscoped preoperatively and free reflux of bile into the gastric remnant was demonstrated; this was associated with an extremely friable, atrophic gastric mucosa often covered with multiple small superficial ulcers.

All of these patients had been followed for a variable period and represented failures in medical management consisting of dietary controls, antacids and spasmodics. Four of these patients were also treated with Cholestyramine in an effort to remove bile salts from the stomach, but even these efforts were unsuccessful. Of further interest was the fact that three patients had additional gastric procedures for the symptoms following their initial procedure and these procedures were also unsuccessful. Our indications for surgery in this group of patients were threefold; failure to improve on strict medical management or gastric bleeding, gastroscopic documentation of bilious reflux through the anastomosis or pylorus and gross and microscopic evidence of diffuse gastritis.

TABLE 1. *Reflux Gastritis Symptoms*

	No. Patients
Epigastric pain	23
Nausea and vomiting	23
Bilious vomiting	19
Weight loss	19*
Bleeding	9
Dysphagia	3

* 5–60 lbs. (mean = 15 lbs.)

Gastroesophagoscopy

All patients had at least one and often repeated gastroscopic studies in order to determine the effects of medical management and to document the course of their disease. A striking pattern appeared to evolve from these studies. First, was the almost continuous regurgitation reflux into the stomach of a mixture of bile stained duodenal content. Secondly, there was evidence of extreme friability of the gastric mucosa with multiple punctate ulcerations and a granular, atrophic appearance. The mucosa appeared to bleed easily upon contact with the fibroptic gastroscope. In all cases, recurrent or marginal ulcer was excluded. Biopsies were taken from numerous sites whenever possible and these were correlated with the gastroscopic pattern.

The typical changes are shown in Fig. 1 from a gastro-

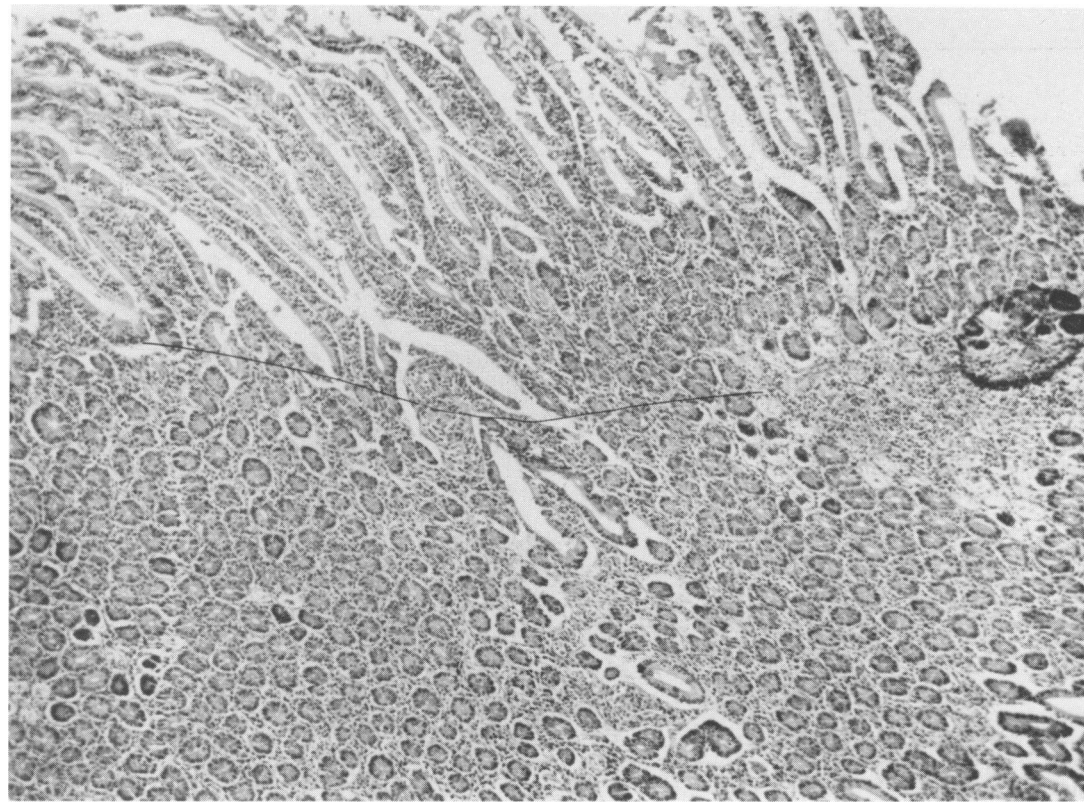

FIG. 1. Typical changes in mild reflux gastritis. Gastroscopic biopsy obtained in this patient revealed chronic inflammation, edema and mild ulceration of the mucosal glands. There is elongation and "corkscrewing" of the gastric glands extending downwards towards the lamina muscularis mucosa. (45×)

scopic biopsy in a patient with long standing symptoms of epigastric pain, nausea and vomiting and weight loss six years following previous gastric surgery. We have interpreted the pattern in this figure as "mild", but elongation of the gastric glands can be clearly seen along with inflammatory changes present in the mucosa. Only rarely did the extent of gastritis penetrate the lamina muscularis mucosa. Small superficial ulcerations of the tips of the gastric glands are also evident.

A more advanced form of reflux gastritis is shown in Figs. 2a and b, taken from different areas of the stomach in the same patient. Denudation of the gastirc mucosa is clearly shown along with marked elongation of the gastric glands extending to the muscularis mucosa and a severe inflammatory response and congestion.

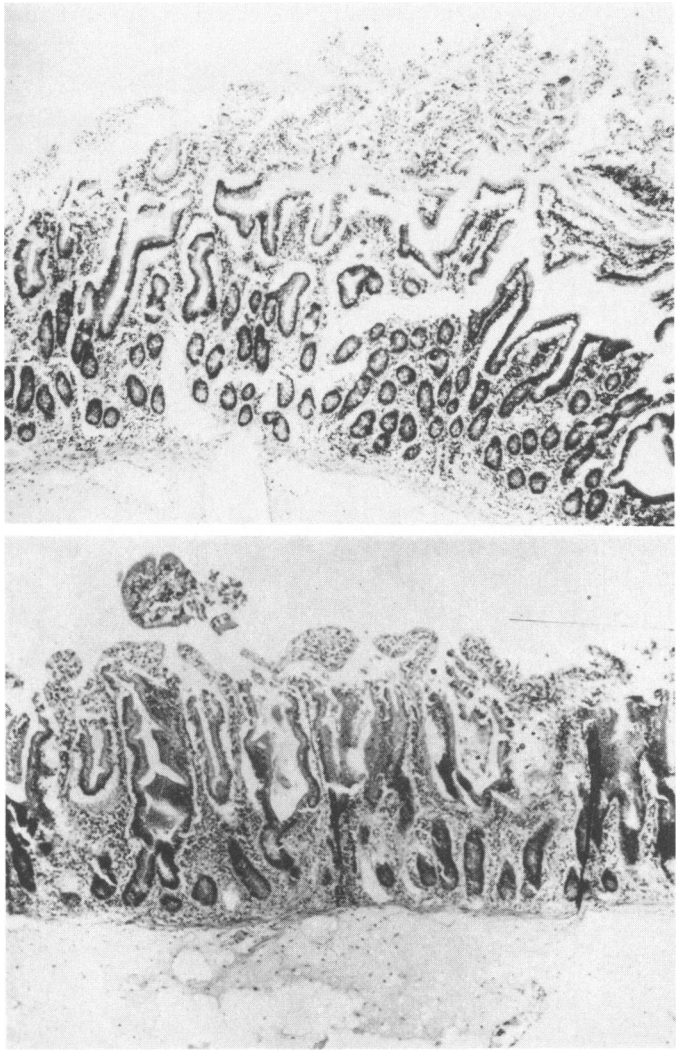

FIG. 2a, b. A more advanced form of reflux gastritis is depicted in these gastroscopic biopsies from another patient with bilious vomiting, epigastric pain and weight loss who had histamine fast achlorhydria. 2a (above) shows denudation of the gastric mucosa with an inflammatory exudate on the surface. 2b (below) taken from another area of the stomach shows marked atrophic changes in the mucosa with diminution in the numbers of chief and parietal cell. (45×)

The degree to which reflux may produce severe alterations in the gastric mucosa is shown in Fig. 3. There can be seen shallow ulcerations with sloughing of the tips of the gastric glands, a diffuse fibrinous exudate and numerous inflammatory cells. This patient had disabling symptoms and had lost 40 pounds of weight over the previous five years.

Surgical Procedures

In these 24 patients who were deemed surgical candidates according to the previously mentioned criteria, a variety of procedures were performed, all aimed to prevent reflux of duodenal juice, including bile, into the stomach or gastric remnant (Figs. 4–7). Fifteen patients had a Roux En-Y anastomosis (Fig. 4) in which at least a 25 cm interposed jejunal limb was utilized. Of these fifteen patients, five had had a previous vagotomy and antrectomy with gastroduodenostomy, four had a previous vagotomy, antrectomy with gastrojejunostomy, four had a subtotal gastrectomy (Billroth II) with no vagotomy, one had a previous vagotomy and pyloroplasty and one patient had a simple diverting gastrojejunostomy.

In five patients a Tanner 19 procedure was performed in which the afferent limb of the gastrojejunostomy was transected and reimplanted into the efferent limb (Fig. 5). This defunctionalized limb of jejunum also measured 25 cm. Advantages of the Tanner 19 procedure included the ease with which it could be performed by obviating the need to dissect the entire anastomosis which in some instances was extremely high, particularly in patients with more radical gastric resections. All five of these patients had had a previous gastrojejunostomy, four with vagotomy and antrectomy and one with a high subtotal gastrectomy alone.

A 10 cm isoperistaltic loop of jejunum (Henley loop) interposed between the gastric remnant and the duodenum was utilized in three patients (Fig. 6). Two of these patients had a previous subtotal gastrectomy and one patient a previous vagotomy and pyloroplasty. Finally, in the remaining patient who had had a gastrojejunostomy performed fifteen years previously for an obstructing duodenal ulcer which had healed, the anastomosis was taken down and normal intestinal continuity restored (Fig. 7). All patients except the last one mentioned had a vagotomy added to the diversionary procedure.

Results

The results of surgery in the entire group of 24 patients are shown in Table 2. Thirteen of the fifteen patients with a Roux-Y loop are classified as an excellent result and remain asymptomatic for periods ranging from three

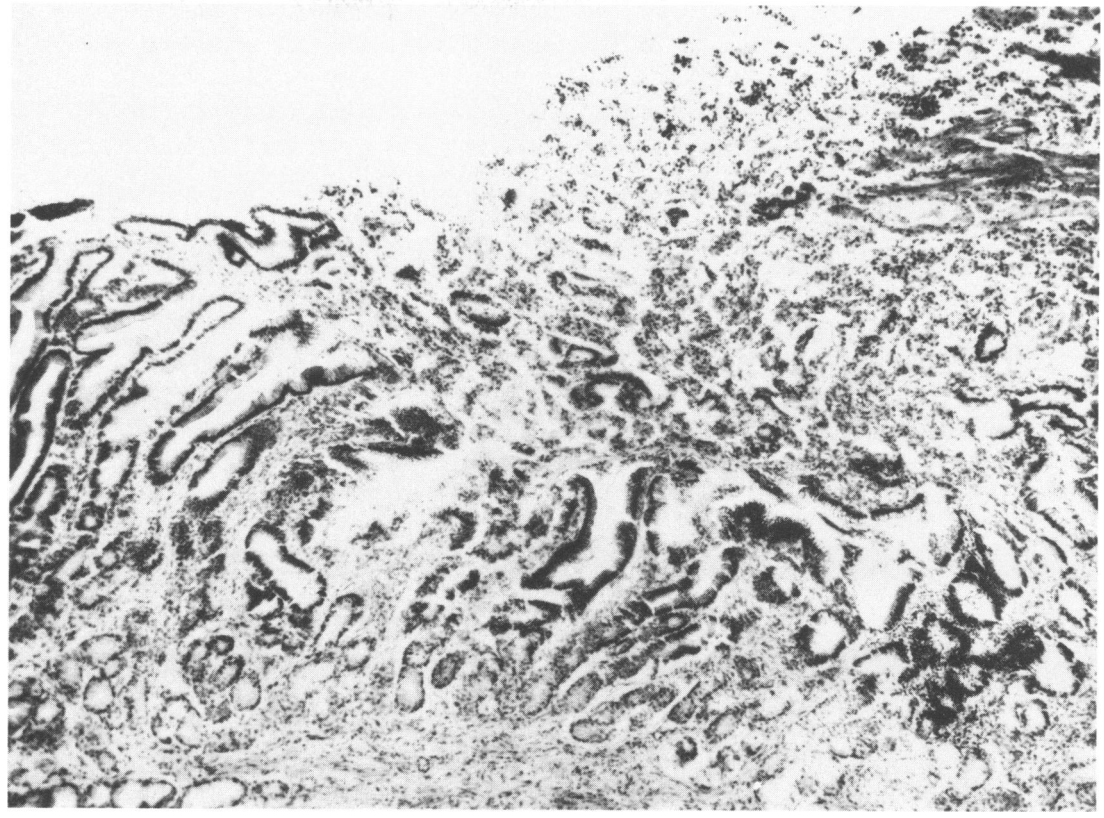

FIG. 3. A severe form of reflux gastritis is shown in this specimen obtained from a patient who had disabling symptoms. The gastroscopic biopsy revealed ulceration of the gastric mucosa, elongation of the gastric glands, severe inflammatory response and areas of hemorrhage. The disease is superficial to the muscularis mucosa. (45×)

months to four years following the definitive procedure. One additional patient is classified as a good result, for despite the fact that his preoperative symptoms of abdominal pain and vomiting had ceased, he continued to have mild dumping symptoms which existed preoperatively. Another patient, a 52-year-old male, was doing well in the postoperative period until the 10th postoperative day when he developed a sudden massive pulmonary embolism and expired.

Of the 5 patients offered a Tanner 19 procedure, all remain asymptomatic with excellent results. Similarly, the three patients with a Henley loop also remain asymptomatic and are classified as excellent; the patient with takedown of the gastrojejunostomy also remains asymptomatic.

The relief of symptoms following these surgical procedures was indeed striking. Abdominal pain rapidly disappeared, in most instances during the immediate postoperative period, vomiting was no longer a problem and all patients gained weight. The clinical response was also accompanied by a marked improvement in the gastroscopic apearance of the stomach in all of these patients. A typical patient is presented in the following case report:

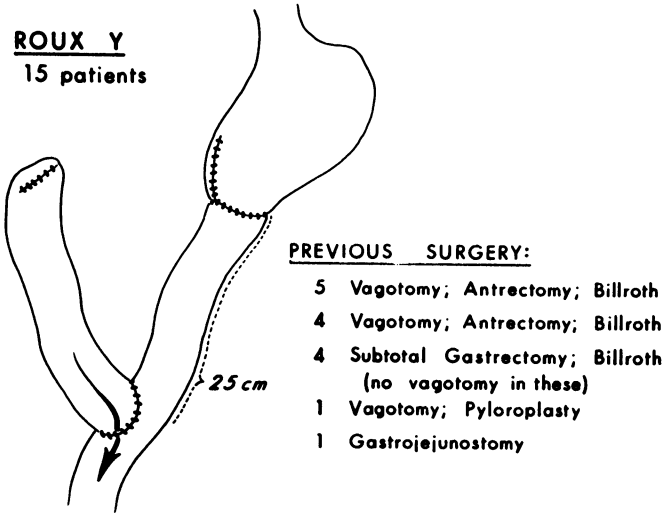

FIG. 4. The Roux-Y technique of bypass of the anastomosis is shown. 25 cm limb of defunctionalized jejunum was utilized in 15 patients. In our experience 25 cm limb was adequate in preventing reflux. Vagotomy should be added if not previously performed in order to prevent recurrent ulceration.

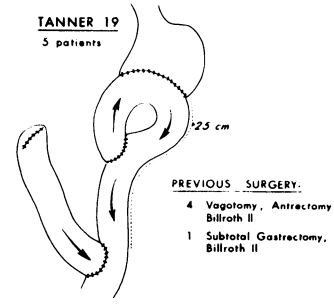

FIG. 5. Tanner 19 technique of reconstruction for reflux gastritis. Advantages of this technique include the limited exposure necessary and implantation of the afferent limb into the efferent limb as shown. Five patients were offered this procedure.

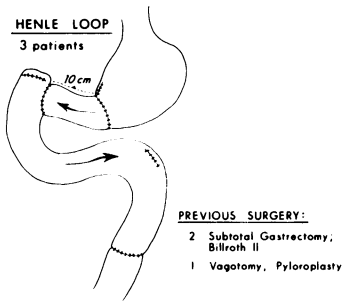

FIG. 6. Henley loop reconstruction utilizing a 10 cm interposed loop of isoperistalic jejunum between the gastric remnant and the duodenum. Vagotomy was added in the 2 patients in whom it had not been previously performed. Three patients were offered this procedure.

TABLE 2. *Results of Surgery in Entire Group of 24 Patients*

Operation	No. Patients	Results		
		Excellent	Good	Poor
Roux Y	15	13	1*	1†
Tanner 19	5	5	0	0
Henley loop	3	3	0	0
Anastomosis Takedown	1	1	0	0

* Continued dumping syndrome
† Post-op death, pulmonary embolus—10 days

Case Report

A 53-year-old Caucasian female underwent a bilateral truncal vagotomy, hemigastrectomy, and gastroduodenostomy in 1957 for intractable peptic ulcer disease. She remained symptom free until 1962 when she developed recurrent nausea, vomiting and severe epigastric pain. Her symptoms were not relieved by antacids. Upper gastrointestinal x-rays demonstrated hypertrophy of the gastric mucosal folds. Gastroscopic examinations revealed multiple superficial perianastomotic ulcerations and diffuse gastritis. Because of these symptoms in 1963 a resection of the previous anastomosis and conversion to a gastrojejunostomy (Billroth II) were performed. She did well following this second procedure until 1971 when her nausea and abdominal pain recurred. She had multiple hospitalizations between 1971 and 1973 but evaluation on each occasion revealed only gastritis and she was discharged on medical therapy. In June, 1973 she was admitted to the Tulane Surgical Service at Charity Hospital with hematemesis, bilious vomiting, severe epigastric pain and chronic weight loss. Admission hematocrit was 24% and stools were guaiac positive. An SMA-12 profile was normal except for hypoalbuminemia. Upper gastrointestinal x-rays were interpreted unremarkable. Gastroscopy revealed a friable, atrophic gastric mucosa with superficial punctate ulcerations (Fig. 8a). Bile freely refluxed into the gastric pouch. A 12-hour gastric analysis and histamine stimulation revealed achlorhydria with a large quantity of bile in the gastric aspirate. After no improvement on medical management she was re-explored at which time there was no evidence of loop obstructions or marginal ulcer. A Tanner 19 diverting procedure was performed and her postoperative course was unremarkable. At five months following her revisional surgery she remains completely asymptomatic. Repeat gastroscopy with biopsy revealed an entirely normal gastric mucosa (Fig. 8b).

Figure 9 shows the gastroscopic biopsy taken three years after a Roux-Y bypass procedure in another patient who had perhaps the most severe and distressing preoperative symptoms in this entire series of patients. This reveals a normal gastric mucosa; the patient is now totally asymptomatic, has returned to full work and is extremely gratified with the result.

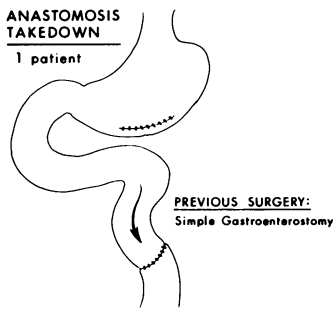

FIG. 7. Technique utilized in one patient with severe reflux gastritis 15 years following gastroenterostomy. Because of the patient's advanced age (81) a simple take-down on the anastomosis was performed and she has remained asymptomatic.

Discussion

For many years it has been pointed out by both gastroscopists and pathologists that gastritis of varying degree may develop in from 5 to 35% of patients following the creation of a stoma between the stomach and the intestine. More recently, this same phenomenon has also been documented after pyloroplasty.[17] However, very little attention was paid to these observations despite the fact that scattered case reports began appearing supporting the fact that such "alkaline" reflux might be responsible for some of the symptoms of abdominal pain, bilious vomiting and weight loss following these operations. It was further supposed that the presence of bilious vomiting was possibly the result of an afferent or efferent loop syndrome, representing a mechanical obstruction or some other functional abnormality of the anastomosis and such patients were often group classified as "afferent loop" syndrome.[13,35]

Largely due to the initial reports by Tanner,[31] Lawson,[18–20] DuPlessis,[8,9] Toye and Williams,[32] Wells and Johnston,[34] Van Heerden and his associates,[33] and Bartlett and Burrington,[1] the clinical pattern of reflux gastritis evolved. Most of the early investigators presumed that the deleterious effect of duodenal or proximal small bowel content upon the stomach was related to its alkalinity although the mechanisms involved were unknown. It remained for Davenport[6] in 1968 to demonstrate experimentally that bile and bile salts were capable of disrupting the ability of the gastric mucosa to contain hydrogen ion within its lumen and he proposed the hypothesis of back diffusion of hydrogen ion as the possible culprit initiating the train of events leading to gastritis. Davenport further demonstrated that the back effusion of hydrogen ion into the wall of the stomach was associated not only with the outpouring of water, sodium and protein into the lumen but also with the release of pepsin into the gastric juice.

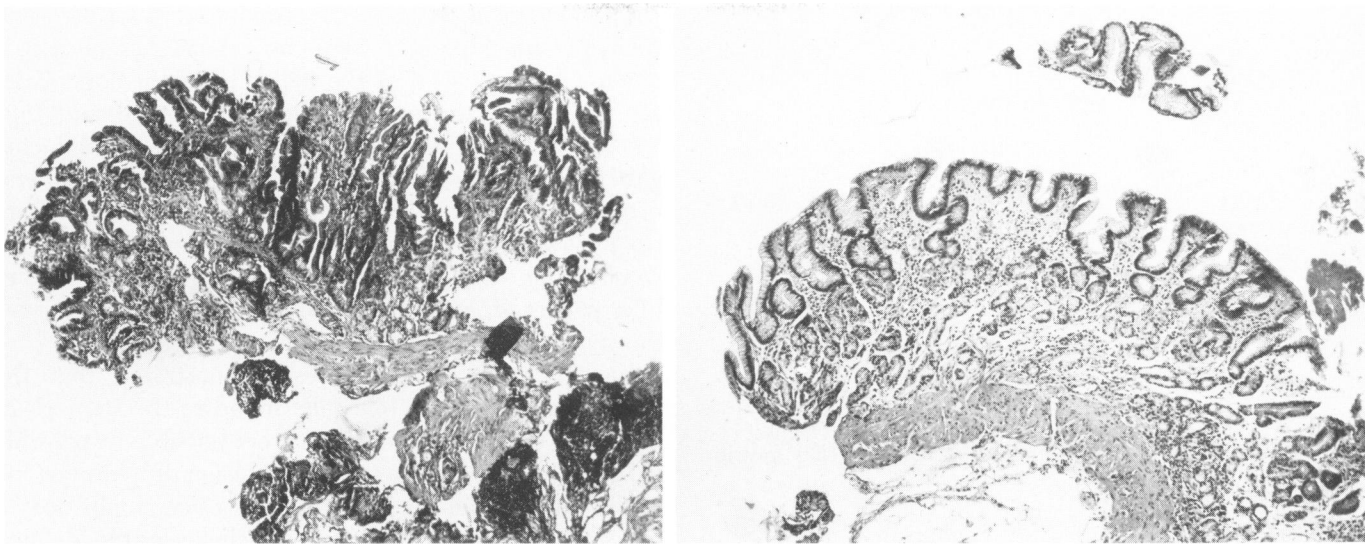

FIG. 8a, b 8a (left) shows preoperative gastric biopsy in 53-year-old female with reflux gastritis. The inflammatory and ulcerative changes in the mucosa are well shown. 8b (right) shows repeat gastric biopsy five months following Tanner 19 diverting procedure. The gastric mucosa appears normal and patient is asymptomatic. (45×)

Since these initial observations numerous investigators have studied experimentally the possible mechanisms involved in the production of diffuse and/or atrophic gastritis in these patients. DuPlessis[9] measured bile acid conjugates in gastric aspirates and found that patients with gastric ulcer had considerably larger amounts of bile acids in overnight gastric juice collections compared to normal individuals or even patients with duodenal ulcer.

Rhodes and his group were able to document by an ingenious technique utilizing an intravenous dose of carbon[14] tagged bile salts and measuring the concentration of radioactive bile salts in the gastric aspirate that regurgitation of duodenal contents through the pylorus into

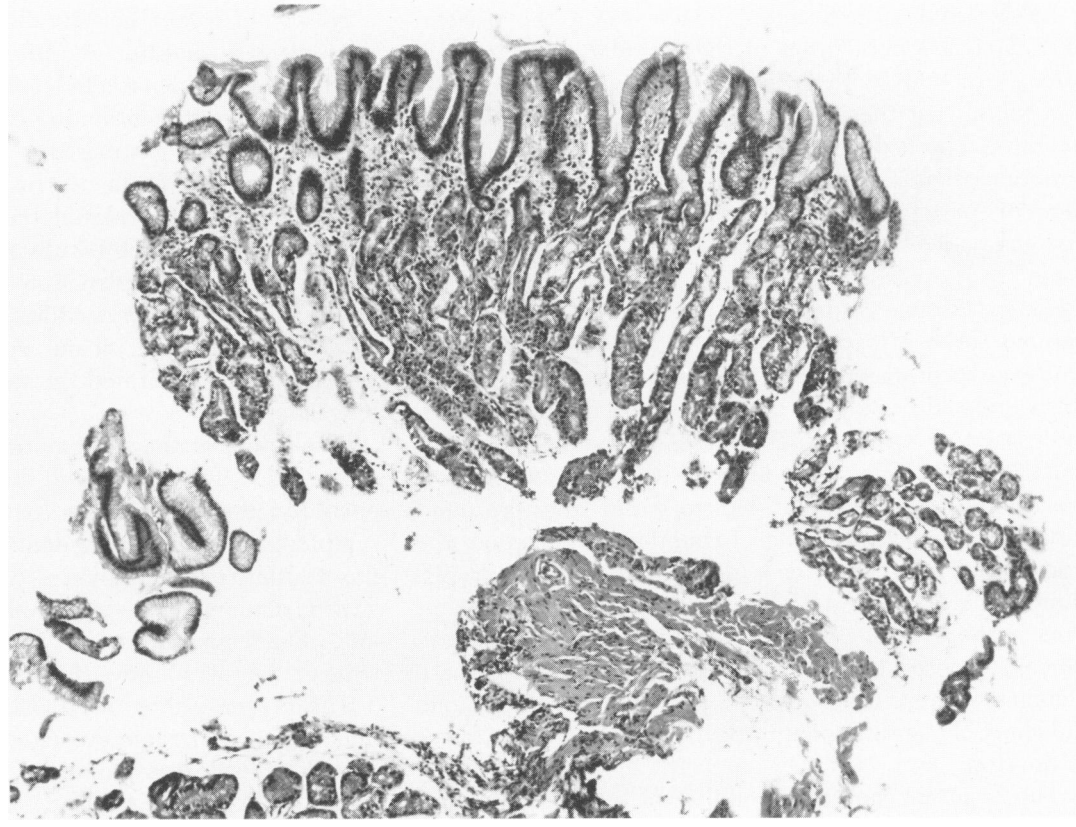

FIG. 9. Gastric biopsy obtained three years following Tanner 19 diverting procedure for reflux gastritis. This patient had symptoms of reflux gastritis for 8 years following truncal vagotomy, hemigastrectomy and Billroth II reconstruction. He became asymptomatic within a few days following surgery and has remained asymptomatic. The biopsy at three years reveals normal gastric mucosa. (45×)

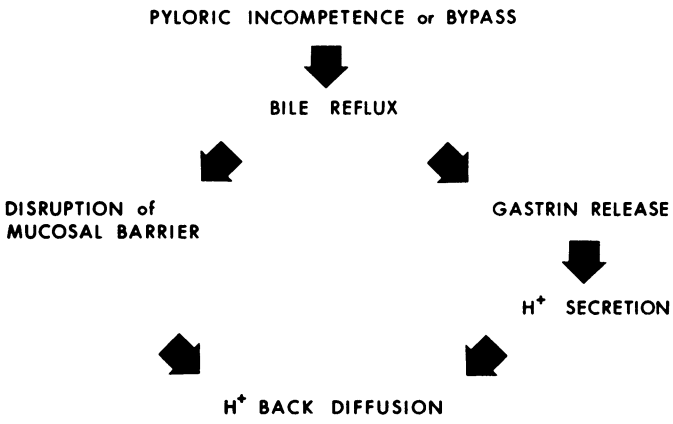

FIG. 10. Postulated mechanism of pathogenesis of reflux gastritis.

the stomach was considerably higher in patients with gastric ulcer as compared to patients with normal stomachs or with duodenal ulcer.[23]

Van Geertruyden[10,11] demonstrated that following the partial gastrectomy with Billroth II reconstruction in humans, the residual gastric pouch showed a progressive diminution in its ability to secrete acid and this correlated well with the pathologic finding of gastritis. Lawson further demonstrated that in dogs with Billroth II reconstruction extensive gastritis could be found in the gastric pouch whereas following a Billroth I reconstruction the gastritis appeared to be limited to the area surrounding the anatomosis.[18]

Ritchie and his associates[24–26] measured the parietal and chief cell population of the residual gastric pouch after antrectomy with a Billroth I type of reconstruction and found that these are unchanged up to one year following the procedure. However, after a Billroth II reconstruction, there was a progressive diminution in the numbers of parietal and chief cells and there appeared characteristic changes of atrophic gastritis in the area adjacent to the gastric jejunostomy. In subsequent studies Delaney, Ritchie and their associates[7] reported an ingenious series of experiments in which a tube of gastric wall was reconstructed from the greater curvature of the dog's stomach, pedicled upon the short gastric vessels, interposed at various sites in the gastrointestinal tract and biopsied. They were able clearly to show that exposure to jejunal content led to rapid and profound mucosal changes consisting of round-cell infiltration, the appearance of mucous cells deep in the gastric crypts, diminution of parietal and chief cells, and cystic dilatation of the glands. On the other hand, tubes of gastric mucosa exposed to pure bile did not demonstrate these changes whereas tubes exposed to pancreatic juice alone did show changes, although they were milder and slower to develop.

The apparent lack of the ability of bile alone to produce such changes was also supported by Byers and Jordan[4] who implanted vascularized patches of gastric mucosa into the wall of the gall bladder in dogs. Even after one year the mucosa appeared normal and they concluded that bile alone was incapable of inducing gastritis but theorized that something additional was needed, possibly acid. Nevertheless, a considerable controversy arose concerning whether or not bile alone, in combination with pancreatic juice, or the combination of bile, pancreatic juice and acid were needed to produce this pattern of gastritis.

Ivey[14,15] and his associates demonstrated that the high concentration of bile salts found in the stomach of patients with gastric ulceration were possibly responsible for the marked increase in back diffusion of hydrogen ion in the human stomach. Johnson[16] in a very sophisticated series of experiments showed conclusively the definite disruption of the gastric mucosal barrier to hydrogen ion causing release of large quantities of pepsin, thus providing another link in the chain of processes caused by back diffusion as previously postulated by Davenport. Disruption of the gastric mucosal barrier to hydrogen ion with consequent back diffusion has also been recently implicated in the development of stress ulcerations by Skillman, Silen and their associates in a large percentage of seriously ill surgical patients.[29,30] Similarly, Hamza and DenBesten have incriminated bile and bile salts in the destruction of the gastric mucosal barrier.[12]

Nahrwald in another significant series of experiments demonstrated that bile and bile salts were able to release gastrin from the denervated antrum.[22] This demonstration added another dimension to our knowledge of the pathogenesis of reflux gastritis, for not only could direct injury to the gastric mucosa by bile, bile salts and pancreatic juice be postulated but also the stimulation of gastrin release by these substances might further agravate the mucosal destruction by added production of hydrogen ion and back diffusion through the mucous blanket producing a vicious cycle. Bedi and his associates have also confirmed the release of antral gastrin by bile salts.[2]

Regardless of the mechanism involved, in patients in whom free reflux of bile and duodenal juice can be demonstrated along with the presence of diffuse gastritis and symptoms of vomiting, abdominal pain and weight loss, the salutary effect of bypass of the secretions from the gastric remnant has been amply demonstrated by Bartlett,[1] DuPlessis,[8] Joseph,[17] Lawson,[20] Mackman,[21] Rutledge,[27] Tanner,[31] Toye,[32] Van Heerden,[33] and Wells.[34]

The present series of 24 patients in which every patient had gastroscopic evidence of gastritis, and the further documentation that in every one of these patients relief of this pattern could be accomplished by diversion

of duodenal and/or proximal jejunal contents from the stomach lends overwhelming support to the observations previously cited by others. In fact, the responses of our patients to such surgery were so prompt and so gratifying to the patients that, in our opinion, little need exists at the present time to continue to procrastinate with other forms of therapy in such patients.

We continue to favor either the Roux-Y reconstruction in such patients or the Tanner 19 procedure which represents a modification of the Roux-Y technique but is simpler and more quickly performed for the anastomosis does not have to be dissected free completely. Nevertheless, caution must be raised in recommending such procedures, particularly in patients who have a large gastric remnant or in whom the antrum is present for it has been aptly demonstrated by many surgeons during the past three decades that diversion of duodenal contents from the anastomosis in such patients may lead to recurrence of the ulcer diathesis. We therefore feel, as does DuPlessis, that it is important to perform a vagotomy in all patients in whom it had not previously been performed.

Although the results with the interposed, isoperistaltic, 10 cm Henley loop in three of our patients are similar to those in which the Roux-Y bypass or the Tanner 19 loop was utilized, we do not see any advantages in the Henley loop and it is considerably more difficult to perform.

Further caution must be instilled, lest the unwary surgeon begins to ascribe all symptoms of pain, vomiting and weight loss after gastric operations to reflux gastritis. In our experience, the majority of patients with such symptoms still have other causes responsible for these symptoms, including afferent loop syndrome and recurrent marginal ulcer. For these reasons we feel strongly that a complete work-up should be performed in all such patients including careful roentgenologic studies, gastroscopy and biopsy along with studies of the gastric secretion. If other causes can be ruled out, and if the typical pattern of gastritis as shown in gastroscopic biopsies is present along with symptoms of abdominal pain, weight loss and the demonstration of free reflux of bile into the stomach, then the diagnosis of reflux gastritis can be firmly established.

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Discussion

Dr. J. Lynwood Herrington, Jr. (Nashville): As all of you know, bilious vomiting was recognized even before the turn of the century, shortly after Anton Wolfler performed the first gastroenterostomy in 1881. As a result, surgeons over the following decades have used various modifications of gastric resection and various modifications of gastroenterostomy with hopes of allevating this distressing problem. As Dr. Drapanas stated, it is, however, only within the last few years that these cases have been documented as the result of the use of the fibreoptic gastroscope and biopsy examinations.

I think, also we are indebted to our British and European friends for bringing this problem to our attention. They were fully aware of this entity 20 years ago. As a matter of fact, Frank Henley in London, and Professor Stauve Haddenstedt in Stockholm, were advocating isoperistaltic jejunal segments and Roux-en-Y reconstructions, in addition to vagotomy and gastric resection, as the primary surgical procedure for the complications of duodenal ulcer in order to avoid this distressing long-range complication.

I certainly agree wih Dr. Drapanas that bile reflux is not infrequent and it is by no means rare after gastric operations. It is like the dumping syndrome; if we question our patients, we will detect it and I think we will find it to occur to some degree in from 5 to 35% of the cases. In the past we were simply overlooking this problem, and were labeling these patients variants of the afferent loop problem, or else calling them neurotics.

During the past 10 months we have operated upon 11 documented cases of reflux gastritis. In the past few years prior to that, we had 17 additional cases, but they were not completely documented. We have treated these patients with either a Roux-en-Y reconstruction or a Henley loop. We have made the Henley loop a little longer than Dr. Drapanas, usually about 15–20 cm. However, Dr. Sawyers and I both prefer the Roux-en-Y reconstruction, as does Dr. Drapanas, because it is easier to carry out.

The 11 patients encountered with this entity during the past 10 months experienced symptoms which were really crippling. There were six males and five females in the group.

(Slide) They had the same set of symptomatology as Dr. Drapanas' patients: epigastric pain, nausea, and vomiting, and weight loss. Weight loss was appreciable in four patients; anemia was a striking feature in several, and two patients had upper gastrointestinal bleeding.

(Slide) This slide shows you the 11 patients, and the type of procedure used. The Roux-en-Y reconstruction was carried out in all but one and in this case we used the Henley loop. As you can see, the original operation was either a Billroth I or Billroth II type reconstruction with vagotomy or else vagotomy and pyloroplasty. In patients undergoing pyloroplasty we have always removed the antrum in carrying out the Henley loop or Roux-en-Y reconstruction. Four of the patients who had the Roux-en-Y reconstruction also had in addition a Hunt-Lawrence pouch constructed below the gastric remnant because these four patients had lost considerable weight and were malnourished. This latter problem is not unique.

One of our 11 patients had not undergone a previous gastric operation. On endoscopic study he had a wide, relaxed, patulous pylorus with regurgiation of bile and duodenal contents and diffuse gastritis with ulceration throughout the stomach.

(Slide) This is a photograph showing the mucosal ulceration and nodular hyperplasia with areas of intervening atrophic gastritis. This is a gross picture of the typical far advanced case.

I would like to ask Dr. Drapanas if he has any explanation why these patients sometimes get along for months or years following the original gastric surgery before developing symptoms of reflux gastritis. Also, would you comment on the postoperative correlations comparing your subsequent gastroscopic examinations with the clinical appraisal of the patient's result. In other words, have all your patients with excellent results had disappearance of the gastritis on endoscopic study, or have you seen some degree of gastritis with bile reflux persisting but now your patient is asymptomatic. From my perusal of the literature on this matter it is my impression that patients have been subjectively improved, but yet areas of gastrititis have persisted.

Lastly, I would like to exert a word of caution. The presence of bile reflux after any definitive gastric operation is a common finding. We should not go overboard in diagnosing this problem and should not subject patients to a remedial operation unless the symptoms are severe and unresponsipe to conservative treatment.

Dr. John A. van Heerden (Rochester, Minnesota): Our initial interest in Rochester, in reflux gastritis, was stimulated by Dr. Jim Priestley, who, I am sure, is well known to all members of this society.

Our management of this problem (slide) has essentially been the same as that so adequately outlined by Dr. Drapanas today. Roux-en-Y, in our group, with vagectomy is still the most important mode of therapy. Early in our experience with this syndrome, a number of cases underwent either jejunal interposition or conversion to a gastroduodenostomy. We feel that neither of these procedures are indicated today in the management of reflux gastritis.

(Slide) Of the 31 patients, we achieved excellent results in 83% of the Roux-en-Y's; and in approximately 72% of the interposition procedures.

(Slide) Our surgical treatment has been essentially the same as outlined by Dr. Drapanas; Roux-en-Y being by far the most common, and certainly the easiest to perform.

Certain facets of this syndrome, however, continue to fascinate and frustrate our group.

1. The difficulty in correlating symptoms with objective findings. I think we have all seen gross reflux in an asymptomatic patient, and in contrast, a patient who is severely symptomatic, with minimal reflux.

2. The difficulty in sorting out functional symptoms from those that are truly organic. So many of these patients are labeled as crocks. Twenty-five per cent of the patients in our series had highly abnormal MMPI's—Minnesota Multiphasic Personality Indexes. Yet this in no way correlated with good or bad postoperative results. Some of the patients who had the worst MMPI's did the best.

3. Why is the pylorus incompetent in the primary case and in stress ulceration? Is this the primary defect, I think continued investigation of the role of the pylorus may well prove most rewarding.