MEDICAL GRAND ROUNDS
Parkland Memorial Hospital
November 2, 1967

CURRENT STATUS OF SURGERY FOR DUODENAL ULCER DISEASE

Outline of Discussion

I. Normal Physiologic Functions of the Stomach
   A. Reservoir capable of monitoring outflow into proximal small intestine
   B. Secretion of intrinsic factor

II. Relationship of Gastric Acid Secretion to Development of Duodenal Ulcer

III. Physiologic Control of Gastric Acid Secretion
   A. Cephalic or vagal phase
   B. Gastric or antral phase
   C. Intestinal phase

IV. Resume of Factors Which Affect Success or Failure of a Given Operative Procedure
   A. Anatomic distribution of acid producing cells
   B. Physiologic control of acid secretion
   C. Consideration of mechanical problems in various resection procedures

V. Historical Development of Present-Day Operations
   A. Gastroenterostomy alone
   B. Vagotomy alone
   C. Subtotal resection (<75%) with Billroth II anastomosis
   D. Subtotal resection (<75%) with Billroth I anastomosis

VI. The Operations Currently Being Utilized for the Treatment of Duodenal Ulcer
   A. 1) 75% subtotal resection with Billroth II anastomosis
      2) 50% hemigastrectomy (or antrectomy) with a vagotomy
      3) Drainage procedure (gastroenterostomy or pyloroplasty) with a vagotomy
   B. Major problems associated with these three operations
      1) Death as an immediate complication of surgery
      2) Recurrent ulceration at the site of the anastomosis
      3) Nutritional problems and postgastrectomy syndromes
   C. Summary of the relative effectiveness of these three operations

I. Normal Physiologic Functions of the Stomach

Only two functions of the stomach appear to be critical. These are:

1) Secretion of intrinsic factor for promotion of B_{12} absorption in the ileum.
2) To act as reservoir for food and to slowly deliver small aliquots of this food to the proximal small intestine.

While the stomach also secretes a lipase and proteinase (pepsin), neither of these enzymes appears to be necessary for normal digestion and absorption of dietary fat and protein.

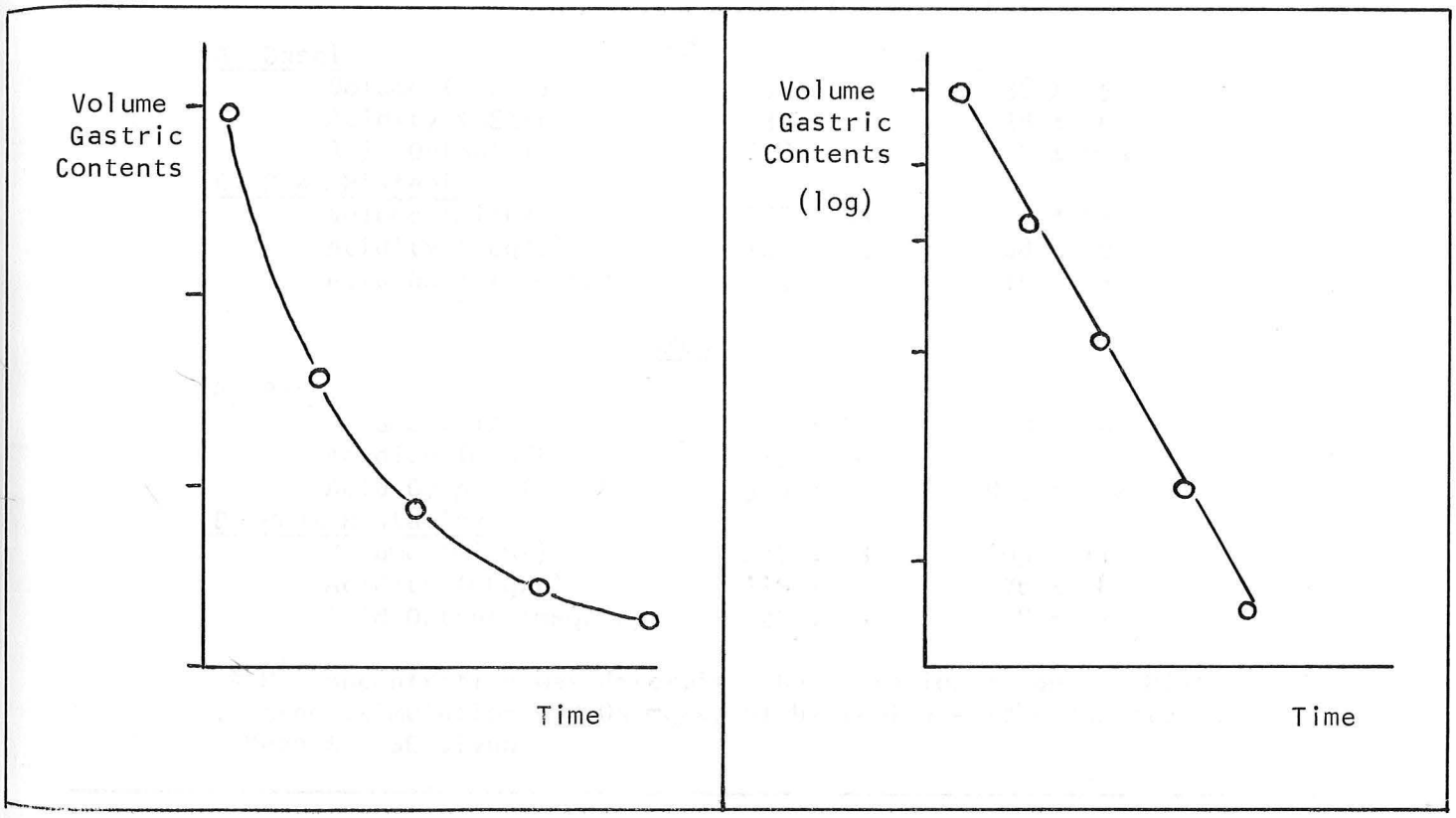


FIG. 1. RELATIONSHIP OF GASTRIC VOLUME AND TIME DEMONSTRATING FIRST ORDER KINETICS OF GASTRIC EMPTYING

1. Williams, J. A., Baume, P. E., and Meynell, M. J.: Partial gastrectomy: The value of permanent vitamin-B_{12} therapy. Lancet 1:342, 1966.

2. Hunt, J. N., MacDonald, J., and Spurrell, W. R.: The gastric response to pectin meals of high osmotic pressure. J. Physiol. 115:185, 1951.

3. Hunt, J. N., and MacDonald, J.: The influence of volume on gastric emptying. J. Physiol. 126:459, 1954.

4. Hunt, J. N., and Spurrell, W. R.: The pattern of emptying of the human stomach. J. Physiol. 113:157, 1951.

II. Relationship of Gastric Acid Secretion to Development of Duodenal Ulcer

TABLE 1*

BASAL AND PEAK HISTAMINE H^+ SECRETION IN NORMAL MEN AND WOMEN

	< 30 y.o.	> 30 y.o.
MEN		
A. Basal		
Volume (ml/hr)	50 ± 16	36 ± 5
Acidity (mEq/L)	36 ± 7	28 ± 6
Acid Output (mEq/hr)	2.8 ± 1	1.0 ± 0.3
B. Peak Histamine		
Volume (ml/hr)	279 ± 43	199 ± 21
Acidity (mEq/L)	121 ± 3	88 ± 9
Acid Output (mEq/hr)	33 ± 6	19 ± 3
WOMEN		
A. Basal		
Volume (ml/hr)	73 ± 24	30 ± 6
Acidity (mEq/L)	35 ± 8	16 ± 4
Acid Output (mEq/hr)	3.0 ± 1	0.5 ± 0.2
B. Peak Histamine		
Volume (ml/hr)	225 ± 23	109 ± 11
Acidity (mEq/L)	119 ± 3	76 ± 8
Acid Output (mEq/hr)	25 ± 3	8 ± 1

* H^+ concentration was determined by titration to pH 7. Histamine stimulation - 0.04 mg/kg of histamine acid phosphate. Mean ± 1 SE given

1. Baron, J. H.: An assessment of the augmented histamine test in the diagnosis of peptic ulcer. Correlations between gastric secretion, age and sex of patients, and site and nature of the ulcer. Gut 4:243, 1963.

2. Baron, J. H.: Studies of basal and peak acid output with an augmented histamine test. Gut 4:136, 1963.

3. Marks, I. N.: The augmented histamine test (Editorial). Gastro. 41:599, 1961.

TABLE 2

BASAL AND PEAK HISTAMINE H^+ SECRETION IN MEN AND WOMEN WITH DUODENAL ULCER

	< 30 y.o.	> 30 y.o.
MEN		
A. Basal		
Volume (ml/hr)	70 ± 14	60 ± 5
Acidity (mEq/L)	64 ± 8	51 ± 4
Acid Output (mEq/hr)	3.7 ± 1	3.6 ± 0.5
B. Peak Histamine		
Volume (ml/hr)	363 ± 36	351 ± 13
Acidity (mEq/L)	127 ± 3	124 ± 2
Acid Output (mEq/hr)	43 ± 4	42 ± 2
WOMEN		
A. Basal		
Volume (ml/hr)	45 ± 5	49 ± 6
Acidity (mEq/L)	41 ± 6	43 ± 6
Acid Output (mEq/hr)	1.7 ± 0.6	2.3 ± 0.5
B. Peak Histamine		
Volume (ml/hr)	304 ± 40	292 ± 24
Acidity (mEq/L)	116 ± 5	117 ± 3
Acid Output (mEq/hr)	29.7 ± 3	32 ± 3

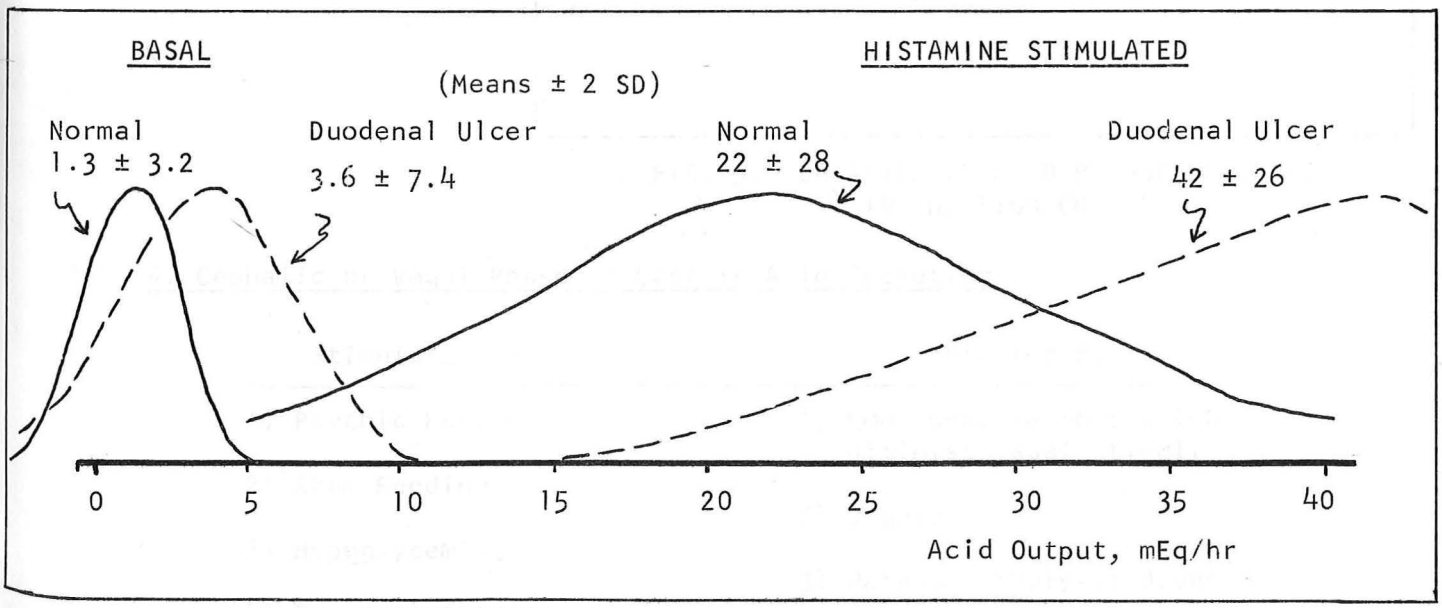


FIG. 2. COMPARISON OF BASAL AND HISTAMINE STIMULATED H^+ OUTPUT IN CONTROL AND DUODENAL ULCER PATIENTS. DATA ARE FOR MEN, ALL AGE GROUPS COMBINED.

4. Zaterka, S., and Neves, D. P.: Maximal gastric secretion in human subjects after Histalog stimulation. Comparison with augmented histamine test. Gastro. 47:251, 1964.

5. Marks, I. N., et al.: The augmented histamine test. An analysis of 672 consecutive tests. S. A. Med. J. 36:807, 1962.

III. Physiologic Control of Gastric Acid Secretion

On the basis of histology of the gastric mucosa, three distinctly different areas of the stomach can be recognized. 1) The cardia is a narrow zone at the esophago-gastric junction which contains mucous producing cells. 2) The fundus, for practical purposes, contains all of the acid producing cells of the stomach. 3) The antrum is essentially devoid of acid producing cells but appears to be the site for the production of the important hormone gastrin.

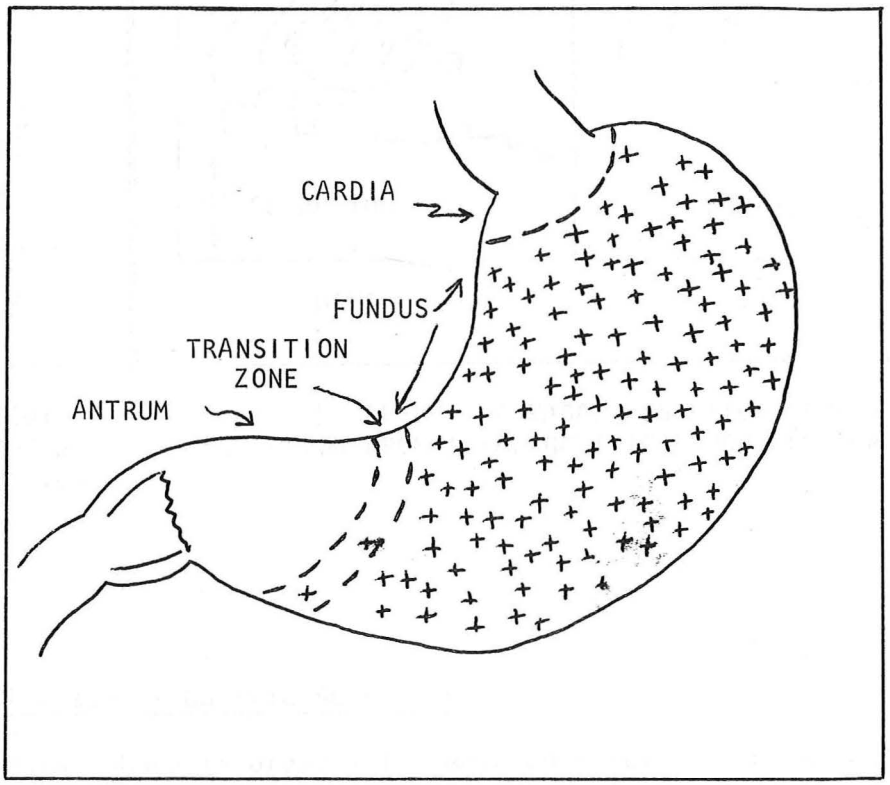


FIG. 3. LOCATION OF ACID PRODUCING CELLS IN THE STOMACH

A. Cephalic or Vagal Phase of Gastric Acid Secretion

Stimulated By	Inhibited By
1) Psychic Factors	1) Emotional factors which diminish vagal stimuli
2) Sham Feeding	2) Vagotomy
3) Hypoglycemia	3) Parasympatholytic drugs
4) Parasympathomimetic drugs	

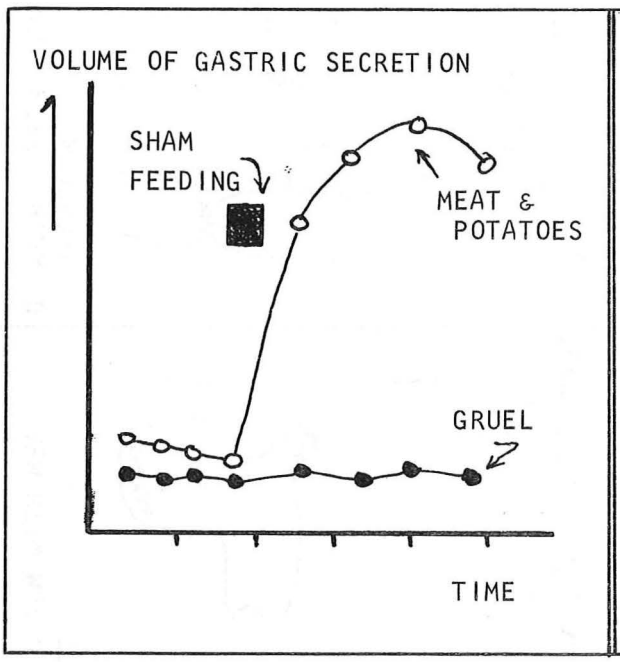


FIG. 4. EFFECTS OF SHAM FEEDING TWO KINDS OF MEALS TO PATIENT WITH COMPLETE ESOPHAGEAL OBSTRUCTION

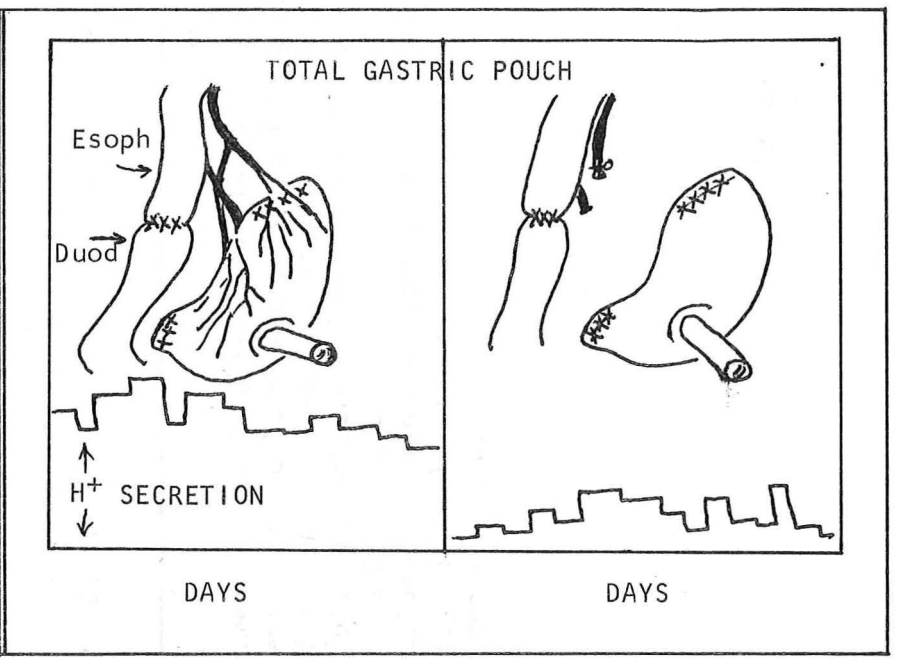
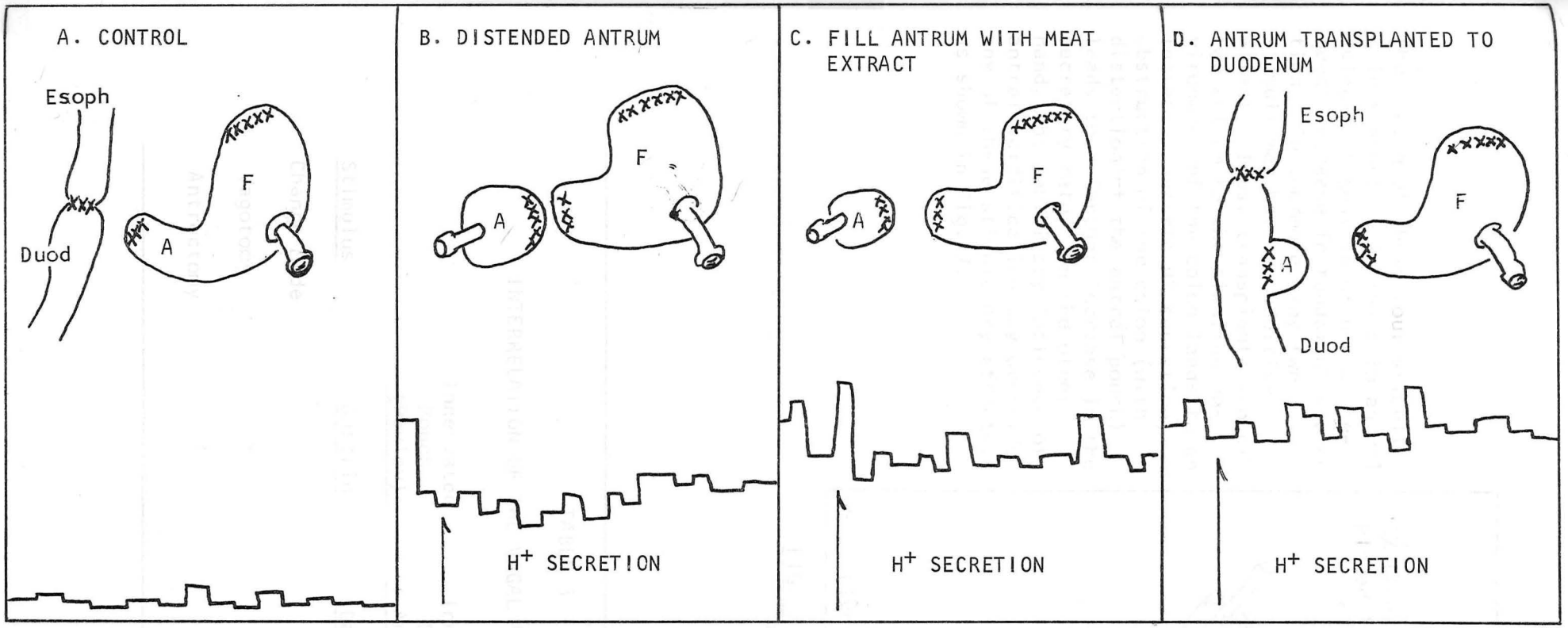


FIG. 5. EFFECT OF VAGAL RESECTION ON ACID SECRETION FROM TOTAL GASTRIC POUCH PREPARATION

B. Gastric or Antral Phase of Gastric Secretion

The gastric or antral phase is presumably mediated solely by the hormone gastrin. This substance is released from the antrum (an area nearly devoid of acid secreting cells) and acts on the cells of the gastric fundus (an area devoid of intrinsic gastrin activity). The release of gastrin is dictated by events occurring in the antrum according to the following schedule:

Stimulated By	Inhibited By
1) Chemical stimuli such as meat extracts or ethanol in antrum	1) Painting the antral mucosa with local anesthetics
2) Distention of antrum	2) Collapse of the antrum
3) Alkalinization of antrum	3) Acidification of the antrum
4) Vagal stimulation	4) Vagotomy



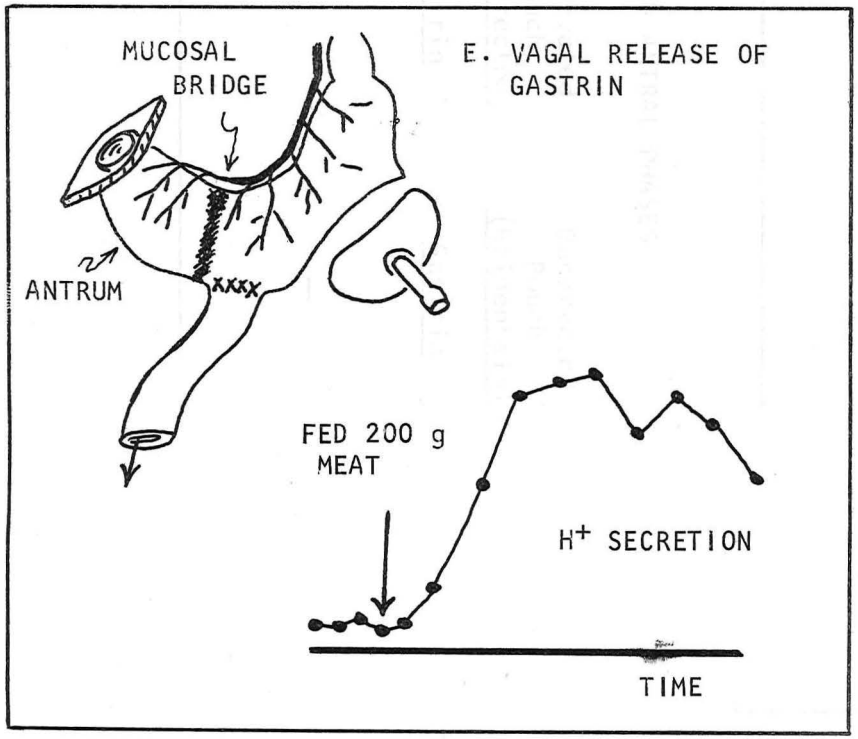


Fig. 6. Experimental data illustrating release of gastrin with subsequent increase in H^+ secretion following (B) distention of antrum, (C) exposure of antrum to chemical stimuli and (D) transplantation of antrum to alkaline environment. In E a surgical preparation with an intact vagal innervation to an isolated antrum is shown; vagal stimulation (by feeding) leads to increased gastric secretion in denervated pouch.

Thus, each of these four stimuli brings about an increase in antral release of gastrin with a subsequent increase in fundal H^+ secretion. Furthermore, any two stimuli may have an additive effect. Thus, transplantation of the antrum to the alkaline environment of the colon leads to an increased gastric H^+ secretion; obstruction of the colon (with distention of the antral pouch) leads to a further increase in the secretory rate. On the other hand, the inhibitory influence of antral acidification may override any of these stimulatory effects, as shown in Fig. 7.

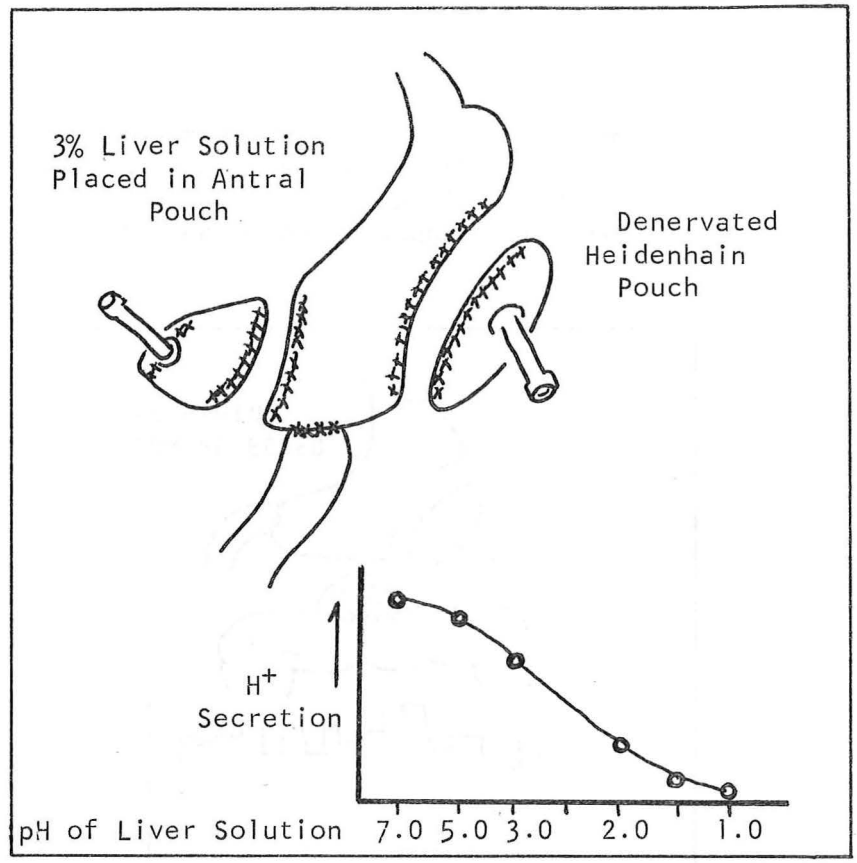


FIG. 7. Effect of Acidification of Antral Perfusate on Secretion of H^+

TABLE 3

INTERRELATION OF THE VAGAL AND ANTRAL PHASES

	Innervated Pouch (Pavlov)	Innervated Pouch + Antrectomy	Denervated Pouch (Heidenhain)
Stimulus	Gastrin	Gastrin	Gastrin
Change Made			
Vagotomy	↓	↓	—
Antrectomy	↑	—	↑

Finally, there is evidence that the presence of acid in the antrum not only inhibits the release of gastrin, but in addition it may stimulate the release of a presumed "inhibitor hormone". The presence of such a hormone is still controversial; however, experiments of the type presented below suggest the presence of such a substance.

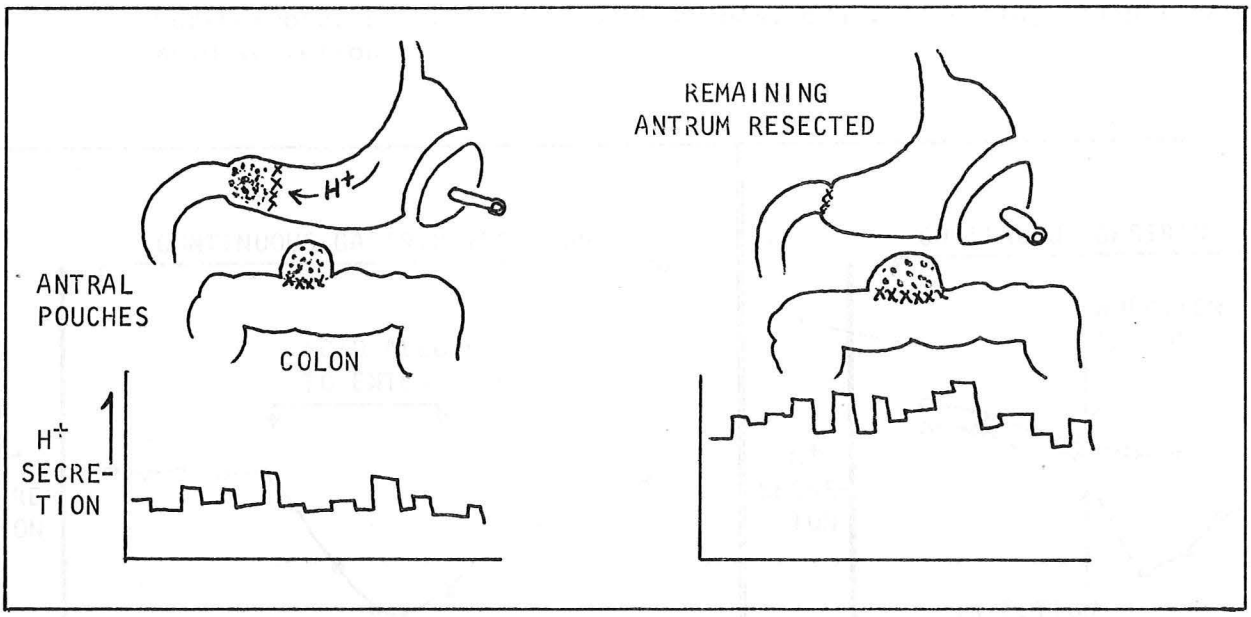


FIG. 8. Experimental Evidence for an Antral Inhibitory Hormone

TABLE 4

Summary: In order to minimize the antral phase of gastric secretion, it is important that:

1) The antrum be denervated

2) Drainage is efficient so that the antrum does not become distended

3) The antrum must remain in contact with the acid stream and not be excluded to an alkaline area of the gastrointestinal tract

C. Intestinal Phase of Gastric Secretion

Finally, evidence exists that a hormone(s?) is (are) liberated from the duodenal mucosa which inhibit(s) gastric H^+ secretion. Such a substance is secreted in response to acidification of the proximal small bowel and this response is abolished only after complete excision of the duodenum. Furthermore, other intestinal hormones, e.g., secretin, inhibit gastric acid secretion.

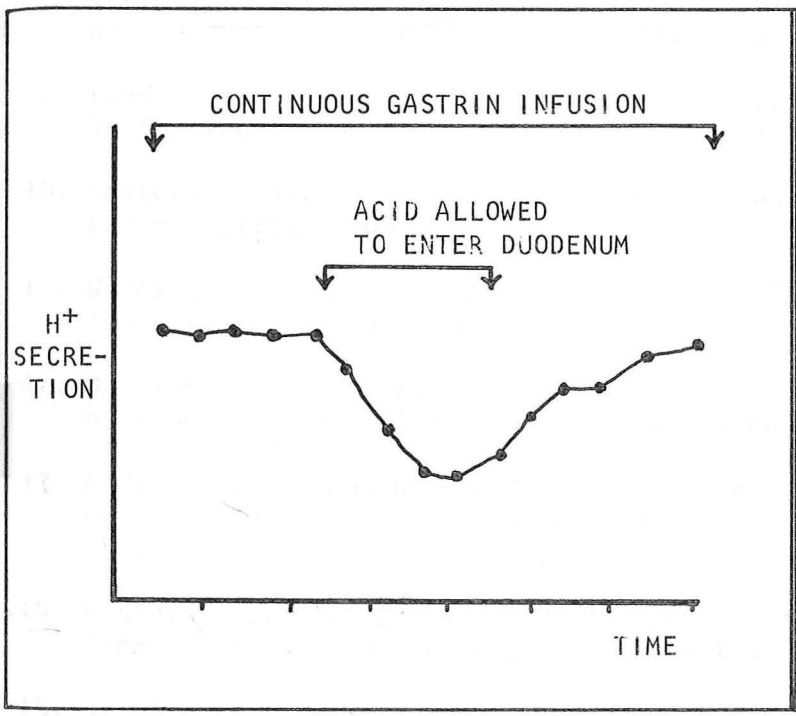


FIG. 9. Effect of Acidification of Proximal Small Bowel on Gastrin-Induced H^+ Secretion From Heidenhain Pouches

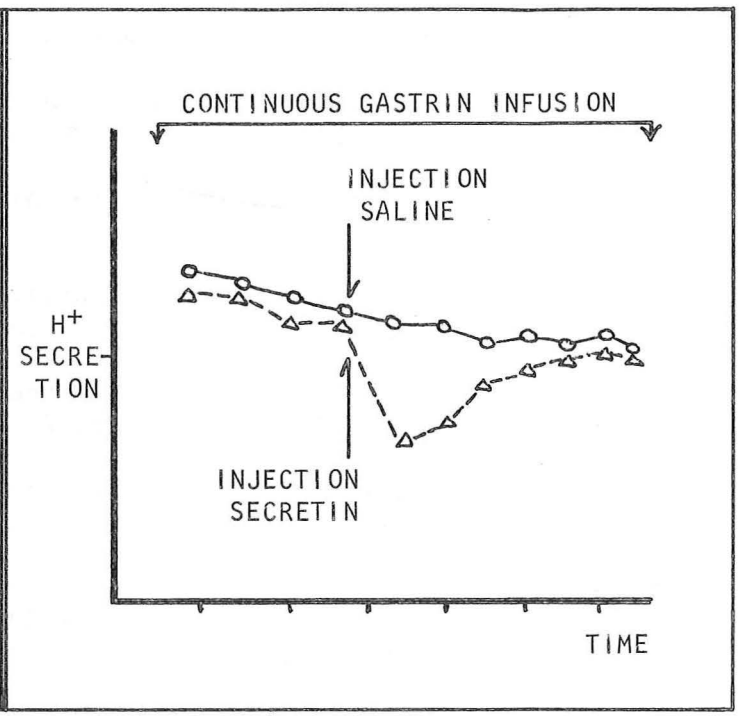


FIG. 10. Effect of Secretin on Gastrin-Induced H^+ Secretion From Heidenhain Pouches

1. Davenport, H. W.: Physiology of the Digestive Tract. Chicago: Year Book Medical Publishers, 1961.

2. Grossman, M. I. (Editor): Gastrin. Berkeley: Univ. of Calif. Press, 1966.

3. Grossman, M. I., Robertson, C. R., and Ivy, A. C.: Proof of a hormonal mechanism for gastric secretion—The humoral transmission of the distention stimulus. Am. J. Physiol. 153:1, 1948.

4. Dragstedt, L. R., et al.: Quantitative studies on the mechanism of gastric secretion in health and disease. Ann. Surg. 132:627, 1950.

5. Dragstedt, L. R., Oberhelman, H. A., Jr., and Smith, C. A.: Experimental hyperfunction of the gastric antrum with ulcer formation. Ann. Surg. 134: 332, 1951

6. Dragstedt, L. R , Oberhelman, H. A., Jr., Zubiran, J. M., and Woodward, E. R.: Antrum motility as a stimulus for gastric secretion. Gastro. 24: 71, 1953.

7. Oberhelman, H. A., Jr., Rigler, S. P., and Dragstedt, L. R.: Significance of innervation in the function of the gastric antrum. Am. J. Physiol. 190:391, 1957.

8. Oberhelman, H. A., Jr., Rigler, S. P., and Dragstedt, L. R.: The significance of innervation in the function of the gastric antrum. Surg. Forum 7:353, 1957.

9. Landor, J. H., Ross, J. L., and Gay, G. R.: The importance of acid inhibition in the regulation of gastric secretion. Arch. Surg. 85:695, 1962.

10. Køster, K. H., and Rune, S. J.: Antral control of gastric acid secretion. Lancet 2:1183, 1963.

11. Wormsley, K. G., and Grossman, M. I.: Inhibition of gastric acid secretion by secretin and by endogenous acid in the duodenum. Gastro. 47:72, 1964.

12. Quintana, R., Kohatzu, S., Woodward, E. R., and Dragstedt, L. R.: Mechanism of duodenal inhibition of gastric secretion. Arch. Surg. 89:585, 1964.

13. Andersson, S., and Grossman, M. I.: Effect of vagal denervation of pouches on gastric secretion in dogs with intact or resected antrums. Gastro. 48: 449, 1965.

14. Konturek, S., and Grossman, M. I.: Localization of the mechanism for inhibition of gastric secretion by acid in intestine. Gastro.49:74, 1965.

15. De La Rosa, C., Manzano, C. A., Woodward, E. R., and Dragstedt, L. R.: Effect of variations in antrum acidity on gastrin release. Arch. Surg. 93:286, 1966.

16. De La Rosa, C., Linares, C. A., Woodward, E. R., and Dragstedt, L. R.: Effect of vagotomy on the gastric secretory response to endogenous gastrin. Arch. Surg. 93:583, 1966.

In addition to considerations of the anatomic location of the acid-producing cells of the stomach and the physiologic means of controlling the rate of H^+ secretion, there is also evidence that various levels of the small bowel have varying degrees of susceptibility to peptic ulceration, as shown below.

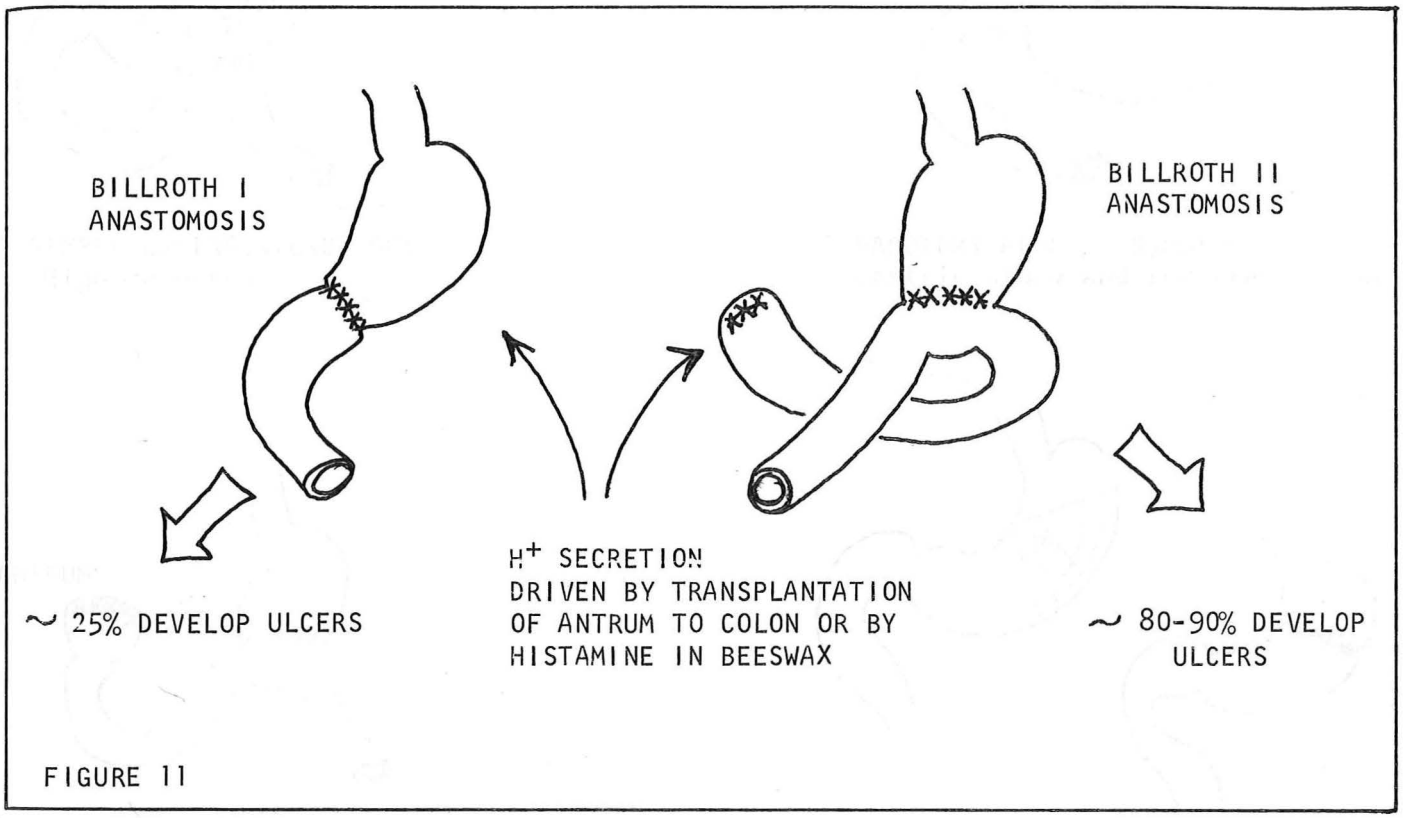


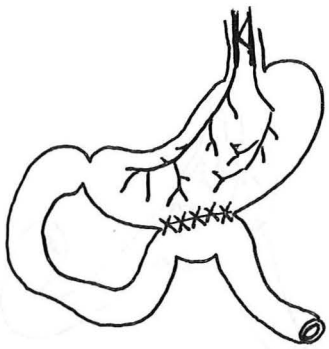
FIGURE 11

It should be stressed that this type of experiment does not prove enhanced susceptibility of the jejunum to ulceration; alternatively, it is possible that in the Billroth I anastamosis acid in the duodenum releases an inhibitor of gastric secretion.

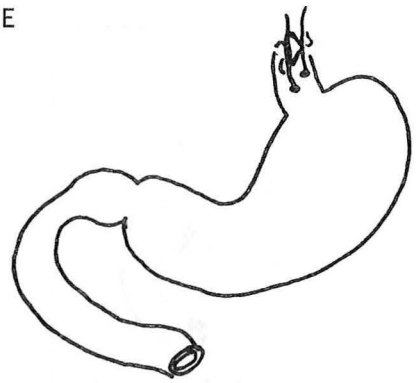
1. Harkins, Schmitz, Nyhus, Kanar, Zech and Griffith: The Billroth I gastric resection: Experimental studies and clinical observations on 291 cases. Ann. Surg. 140:405, 1954.

2. Dragsted, Oberhelman, and Smith: Experimental hyperfunction of the gastric antrum with ulcer formation. Ann. Surg. 134:332, 1951.

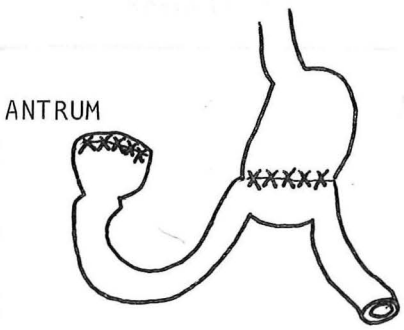
OPERATIONS WHICH HAVE FAILED
BECAUSE OF POSTOPERATIVE
COMPLICATIONS



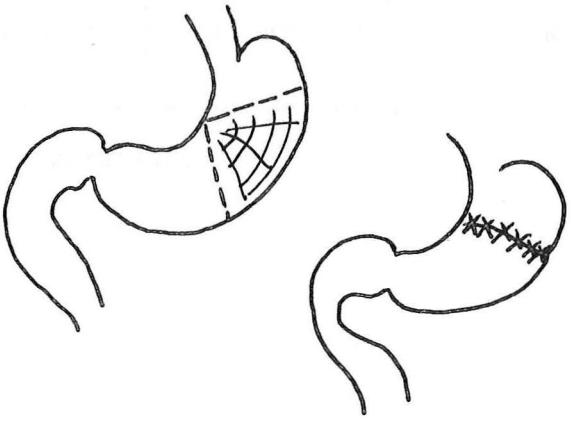
1) SIMPLE GASTROENTEROSTOMY -
High recurrence rate



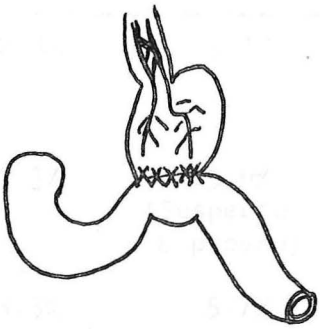
2) VAGOTOMY ALONE - Symptoms of
gastric atony and recurrent ulcers



3) ANTRAL EXCLUSION OPERATIONS -
Very high recurrence rate



4) TUBULAR RESECTION -
High recurrence rate



5) GASTRECTOMIES OF LESS THAN 75% -
High recurrence rate

FIGURE 12

THE THREE OPERATIONS CURRENTLY BEING UTILIZED TO TREAT DUODENAL ULCER

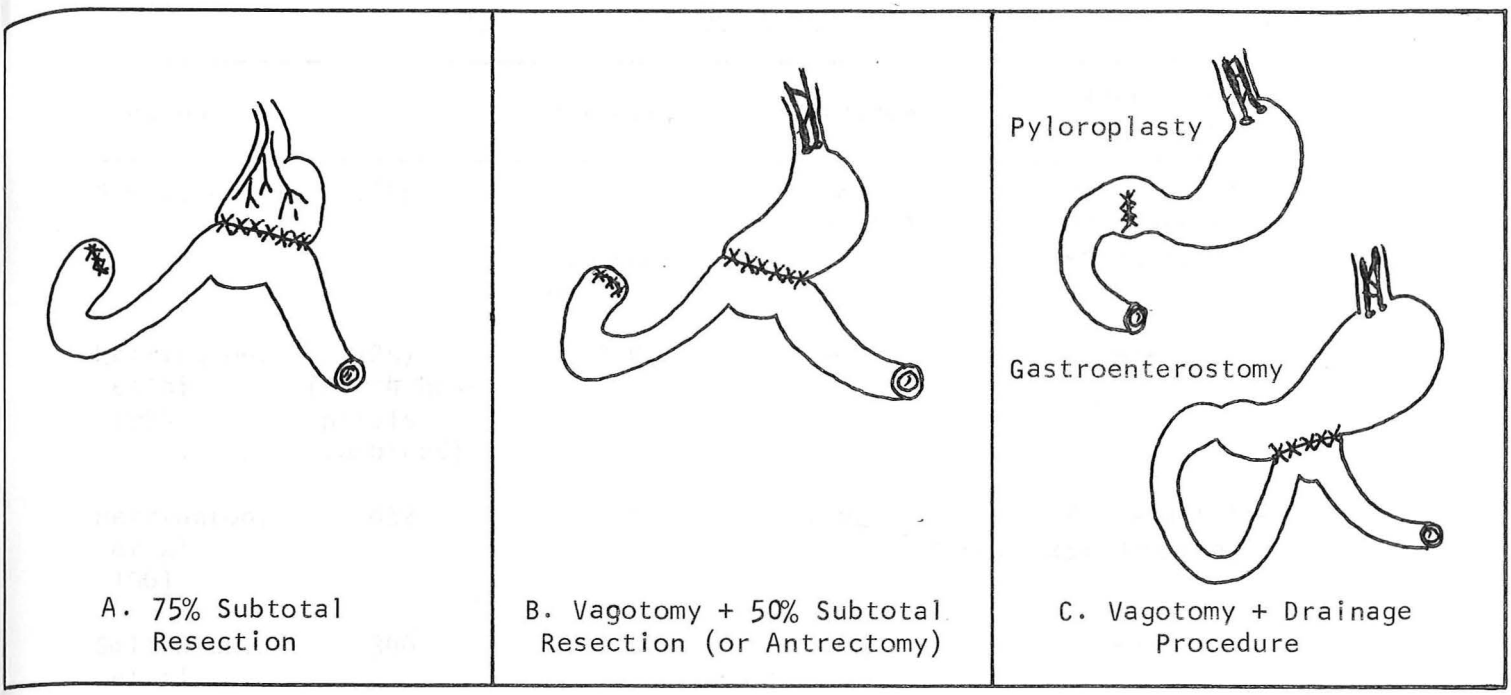


FIG. 13. Currently Utilized Operations

REPRESENTATIVE DATA ON THESE OPERATIONS

Table 5

A. 75% Subtotal Gastrectomy

Author	No. Cases	Mortality	Recurrence	Nutritional-Postgastrectomy Problems
Clark, et al	331	2-3%	5.1%	16% vomiting 12% dumping 20% diarrhea
Magnuson, et al	230 (16 were Billroth I)	0.3%	9.0% (Suspected & proven)	51% lost weight 40% dumping
Smithwick, et al	510	3.3%	5.7%	—

Table 6

B. Vagotomy + 50% Subtotal Resection

Author	No. Cases	Mortality	Recurrence	Nutritional-Postgastrectomy Problems
Scott, et al	1600	2.3% (1% in younger patients)	0.8% (3 had ZE)	10% weight loss 10% diarrhea 25% dumping
Herrington, et al 1964	1257 (For 4 hospitals combined)	3.0%	—	—
Herrington, et al 1961	832	2.8%	0.6%	9% weight loss 25% dumping
Smithwick, et al	346	2.0%	1.4%	—

Table 7

Vagotomy + Drainage Procedure

Author	Drainage Procedure	No. Cases	Mortality	Recurrence	Nutritional-Postgastrectomy Problems
Clark, et al	Gastroenterostomy	146	0.7%	4.0%	26% vomiting 13% dumping 31% diarrhea
Evans, et al	Gastroenterostomy Pyloroplasty	466 61	0.9% 0	8.1% 1.6% (8/36 gastric)	4.3% dumping 2% diarrhea
Holt, et al	Gastroenterostomy	100	0	1.0%	6% dumping 20% diarrhea 23% vomiting
Austen,	Gastroenterostomy 90% Pyloroplasty 10%	100	1.0%	5.0%	6% dumping 13% weight loss 15% diarrhea
Weinberg, et al	Pyloroplasty	200	0.4%	5.0%	5% "disabling sequelae"

1. Dragstedt, L. R., and Woodward, E. R.: Appraisal of vagotomy for peptic ulcer after seven years. JAMA 145:795, 1951.

2. Weinberg, J. A., Stempien, S. J., Movius, H. J., and Dagradi, A. E.: Vagotomy and pyloroplasty in the treatment of duodenal ulcer. Am. J. Surg. 92:202, 1956.

3. Edwards, L. W., et al.: Duodenal ulcer: Treatment by vagotomy and removal of the gastric antrum. Ann. Surg. 145:738, 1957.

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5. Smithwick, R. H., Harrower, H. W., and Farmer, D. A.: Hemigastrectomy and vagotomy in the treatment of duodenal ulcer. Am. J. Surg. 101:325, 1961.

6. Herrington, J. L., Jr., Edwards, W. H., and Edwards, L. W.: Re-evaluation of the surgical treatment of duodenal ulcer. Surgery 49:540, 1961.

7. Holt, R. L., and Lythgoe, J. P.: Ten-year results of vagotomy and gastro-jejunostomy in the treatment of chronic duodenal ulcer. Brit. J. Surg. 49: 255, 1961.

8. Austen, W. G., and Edwards, H. C.: A clinical appraisal of the treatment of chronic duodenal ulcer by vagotomy and gastric drainage operation. Gut 2:158, 1961.

9. Stafford, E. S., and Finney, G. G.: Results of surgical treatment of peptic ulcer. Ann. Surg. 155:687, 1962.

10. Orr, I. M.: Selective surgery for peptic ulcer: A review. J. Brit. Soc. of Gastroenterology - Gut 3:97, 1962.

11. Herrington, J. L., et al.: Etiologic factors influencing the operative mortality after vagotomy and antrectomy for duodenal ulcer. Am. J. Surg. 107:289, 1964

12. Clark, C. G., Murray, J. G., Slessor, I. M., and Wyllie, J. H.: Complete vagotomy and its consequences: Follow-up of 146 patients. Brit. Med. J. 2: 900, 1964.

13. Scott, H. W., Jr., et al.: Vagotomy and antrectomy in surgical treatment of duodenal ulcer disease. Surg. Clin. No. Amer. 46:349, 1966.

14. Magnuson, F. K., Judd, E. S., and Dearing, W. H.: Comparison of postgastrectomy complications in gastric and duodenal ulcer patients. The American Surgeon 32:375, 1966.

15. Kemp, D.: An evaluation and comparison of the early and late results of standardized Polya gastrectomy. Gut 8:151, 1967.

Complications of the 3 accepted operations with respect to 1) immediate operative mortality, 2) recurrent ulceration, and 3) postoperative nutritional and post-gastrectomy syndromes.

1) IMMEDIATE OPERATIVE MORTALITY

Table 8

Causes of Death in Postoperative Period Following 1537 Gastric Resections for Gastric and Duodenal Ulcer (Overall Mortality in This Series was 1.5%)

35% Pancreatico-Duodenal

- Pancreatic disease
- Duodenal stump leaks
- Intraabdominal infection from leaking suture lines

65% Cardio-vascular

- Pulmonary
- Underlying diseases

Table 9

Technical Complications in Transabdominal Vagotomy

A. > 4700 Operations

—Esophageal rupture	0.3%
—Splenic injury	0.8%
—Hemorrhage	0.1%
—Pleural injury	0.1%
—Hepatic injury	0.1%
—Pericardial effusion	0.1%

B. 883 Operations

—Overall rate of technical complications (mainly splenic damage and hemorrhage)	4.7%
—Reoperation or prolonged morbidity	0.8%
—Death attributable to vagotomy	0.1%

1. Harvey, H. D.: Safety in performing partial gastrectomy for peptic ulcer. Ann. Surg. 153:256, 1961.

2. Turner, F. P.: Postoperative complications following gastric resection. Relationship to activity of peptic ulcer and duration of preoperative therapy. Am. J. Surg. 101:711, 1961.

3. Ballinger, W. F., II: The small intestine following vagotomy. SGO (Editorial), p. 115, January 1963.

4. Pearson, S. C., MacKenzie, R. J., and Ross, T.: The use of catheter duodenostomy in gastric resection for duodenal ulcer. Am. J. Surg. 106:194, 1963.

5. Belding, H. H.: Mechanical complications following subtotal gastrectomy. SGO, p. 578, November 1963.

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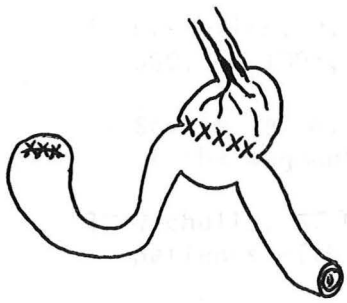
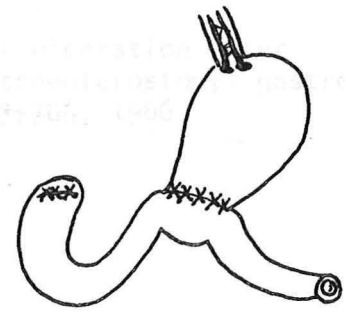
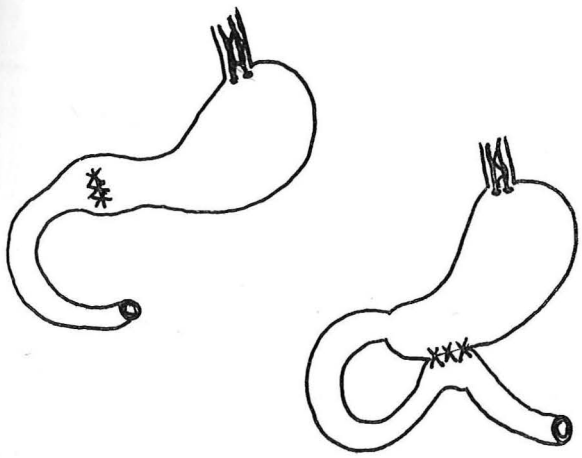
2) RECURRENT ULCERATION DUE TO:



A) 75% Subtotal Resection
   1) Inadequate resection
   2) Basal secretory drive due to:
      a) Excluded or retained antrum
      b) Multiple adenoma syndrome



B) Vagotomy + 50% Gastrectomy
   1) Inadequate antral resection
   2) Inadequate vagotomy
   3) Basal secretory drive due to:
      a) Excluded or retained antrum
      b) Multiple adenoma syndrome



C) Vagotomy + Pyloroplasty or Gastroenterostomy
   1) Inadequate vagotomy
   2) Incorrectly placed or too large gastroenterostomy
   3) Poorly functioning pyloroplasty
   4) Basal secretory drive due to:
      a) Very hypersecreting duodenal ulcer patient
      b) Multiple adenoma syndrome

FIGURE 14

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3) POSTGASTRECTOMY NUTRITIONAL PROBLEMS

CAUSES OF MALABSORPTION FOLLOWING GASTRIC SURGERY

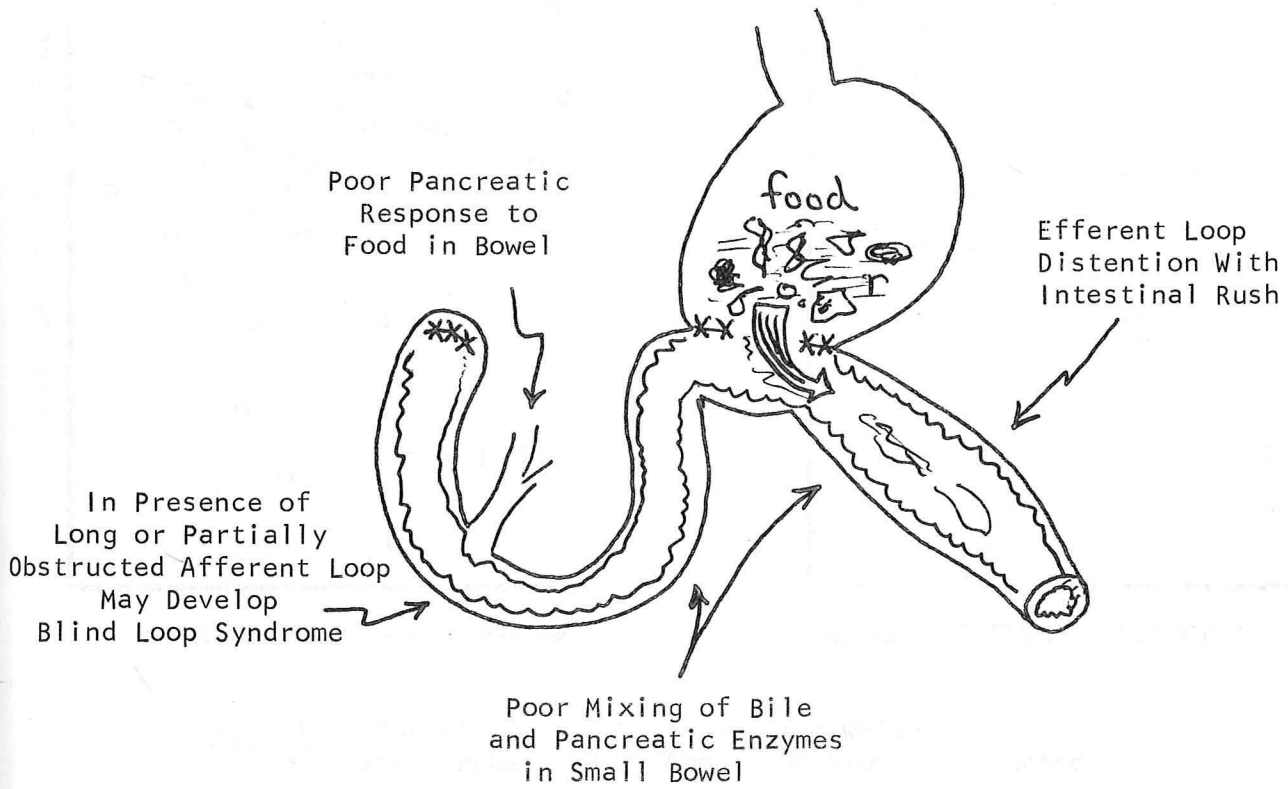

FIGURE 15

Table 10

Comparison of the Amount of Stomach Resected With Postoperative Nutritional Problems

	1936-1948	1949-1953
No. Patients	486	462
% Stomach Resected	50%	60-75%
% Patients Lost > 5 lbs.	14%	31%
% Patients with Significant Weight Loss	2.1%	11.9%
% Patients with Serious Weight Loss	1.6%	6.3%
% Dumping	9%	27%

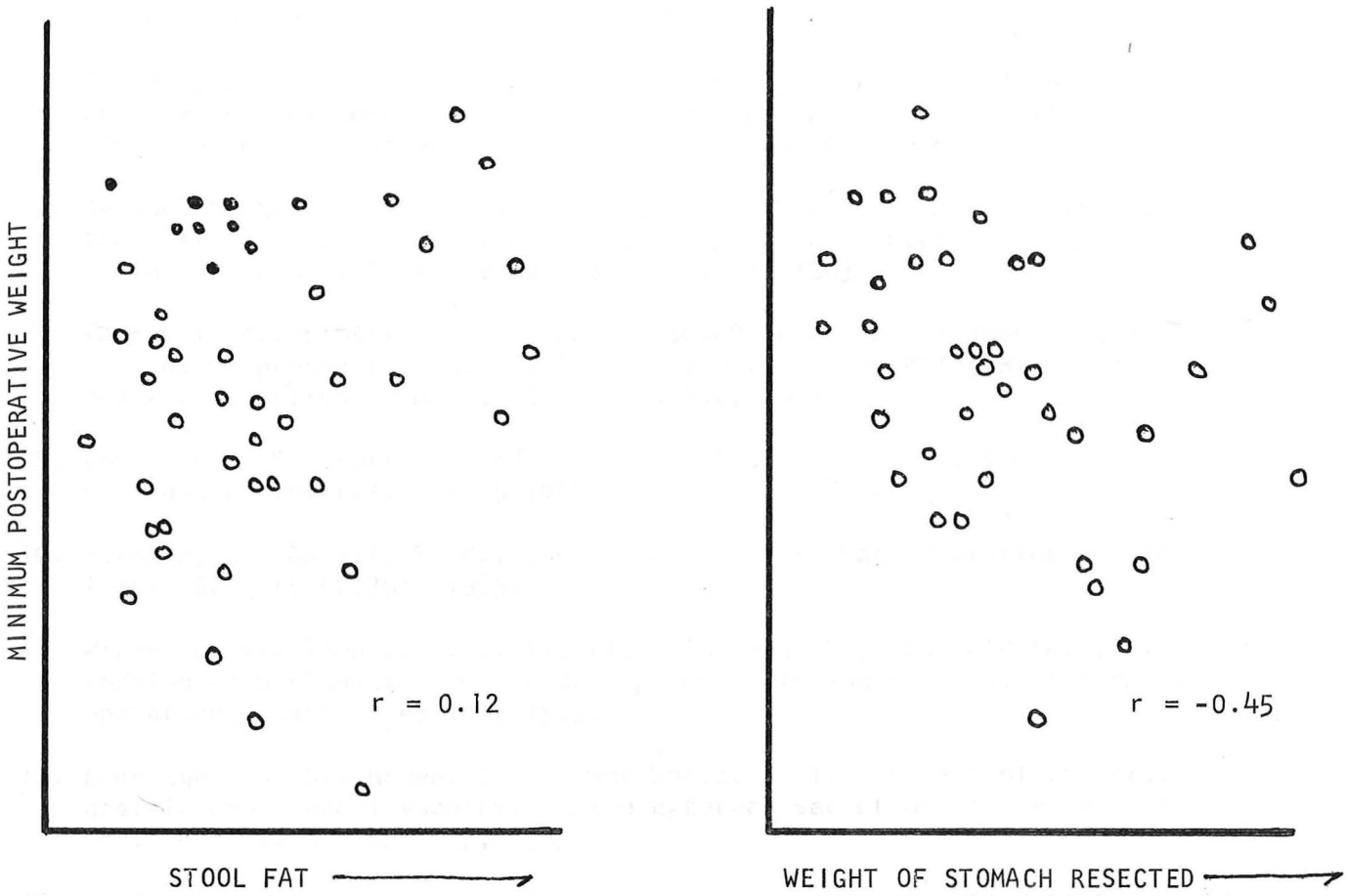

FIG. 16. Correlation of Postoperative Weight Loss With 1) Degree of Steatorrhea and 2) Amount of Stomach Resected

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TABLE 11

Summary of Approximate Statistics for
Elective Surgery by the 3 Currently Utilized Operations

	Mortality Rate		Recurrence Rate	Nutritional and Postgastrectomy Problems
75% Subtotal Resection	Community at Large	5%	4-5%	++++
	Medical Center	1.5-2.0%		
Vagotomy + 50% Subtotal Gastrectomy	1.5-2.0%		< 1%	++
Vagotomy + Drainage Procedure	< 1.0%		>> 5%	+