Staff Meeting Bulletin
Hospitals of the » » »
University of Minnesota

Peptic Ulcer

Volume XI | Friday, March 15, 1940 | Number 21

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
UNIVERSITY OF MINNESOTA

Volume XI | Friday, March 15, 1940 | Number 21

INDEX

Published for the General Staff Meeting each week during the school year, October to May, inclusive.

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William A. O'Brien, M.D.

I. LAST WEEK

Date: March 8, 1940

Place: Recreation Room
Powell Hall

Time: 12:15 to 1:30 P.M.

Program: Movie: "Little Goldfish"

Fluoroscopy of the Chest
Jack I. Chalek
Solveig Bergh
Discussion
Leo G. Higler
Macnider Wetherby
Owen H. Wangensteen
Charles E. Lyght
D. H. Bessesen

Present: 145

Gertrude Gunn,
Record Librarian

- - - -

II. MOVIE

Title "Autograph Hound"
A Walt Disney Short

Released by: R-K-O

- - -

III. ANNOUNCEMENTS

1. CENTER FOR CONTINUATION STUDY

Continuation Course in Surgery - March 11-16. Welcome to our guests at Staff Meeting today:

Edward D. Churchill, John Homans Professor of Surgery, Harvard Medical School; Chief of the West Surgical Service, Massachusetts General Hospital, Boston.

Burt J. Canfield, Rockford, Ill.; Clark N. Cooper, Waterloo; James W. Cosgriff, Olivia; Lloyd T. Davis, Wadena; Harold J. Dvorak, Milwaukee; C. O. Estrem, Fergus Falls; John C. Feuling, Bovey; Alvin C. Florin, Fonddu Lac; E. E. Gallagher, La Crosse; Silas Giere, Benson; Gilbert Gordon, Rosetown, Sask.; Ralph Gorrell, Clarion, Ia.; William M. Hartman, Macomb, Ill.; A. H. Heidner, West Bend; Ralph B. Kettlewell, Sauk Centre; Severin H. Koop, Richmond; Ernest N. Krueger, Appleton; S. T. Kucera, Lonsdale; Roy C. Little, Mayville, N. D.; Maurice J. McKenna, Grand Rapids; Sidney D. Martin, Carroll, Ia.; George S. Metcalf, Janesville, Wis.; Arthur J. Offerman, Omaha; Clifford A. Olsen, Hammond, Wis.; H. R. Sharpe, Fond du Lac; Guy Wakefield, West Salem, Wis.; William W. Will, Bertha; Herman E. Wolf, La Crosse.

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2. SCORE - COURSES

Medical and Hospital - to date:

Number of Courses - 56
Attendance - 2,027
Time - 3-1/4 years.

- - -

3. SPRING PROGRAM

April 1 - 6	Venereal Disease
April 29-May 1	Obstetrics
May 2 - 4	Health Problems of College Students
May 6 - 11	Electrocardiography
May 20 - 25	Pediatrics
May 23 - 25	Hospital, Medical and Institutional Library Service
June 6 - 8	Gynecologic Tumors

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4. STAFF MEETING PROGRAM
Spring Assignments

April 5	Out-Patient Department
" 12	Medical Photography
" 19	Medicine
" 26	Student Health Service
May 3	Pediatrics
" 10	Radiation Therapy
" 17	Obstetrics and Gynecology
" 24	Physical Therapy
" 31	Biochemistry
June 7	Anesthesiology
" 14	Administration

- - -

Vacations

No staff meetings March 22 or 29 because of spring holiday. Next meeting Apr. 5th,

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5. STUDY

Minnesota State Medical Association Study Subject for April - Cancer of Digestive Tract.

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6. SPECIAL NOTICE (LABORATORIES)

Only emergency requests will be handled on Good Friday, March 22, 1940.

Gerald T. Evans.

IV. PEPTIC ULCER

George S. Bergh
Lyle J. Hay
Benedict Trach

PART I

It has long been recognized that, except for ulcerations due to specific diseases, ulcers of the gastrointestinal tract are confined to those areas exposed to the action of the acid secretions of the stomach.

The secretions elaborated in the stomach vary with the region in which they are produced. The mucous membrane of the stomach is occupied by numerous glands (estimated at 35,000,000) which open into the gastric pits. Based on the type of glands found in each region, the stomach is divided into three zones:-

1. Cardiac zone: a narrow strip in the cardiac region about 0.5 to 3.0 cm. wide. The cardiac glands are compound tubular glands which secrete some acid, but largely mucus.

2. Zone of the gastric glands: This zone comprises the fundus and a large part of the body of the stomach. The glands are of the simple tubular, branched variety and consist of four types of cells:

   a. The chief or zymogenic cells are found in the lower portion of the gland. They are believed to secrete pepsinogen which is converted to pepsin by the action of HCL.

   b. Parietal cells are found throughout the gland, but are more numerous near the neck. They apparently are the acid-secreting cells.

   c. Mucous neck cells are found in the neck of the gland, and secrete mucus.

   d. Argentaffine cells are found singly scattered in the glands. Their function is unknown.

3. Pyloric zone: The glands here are also of the simple, branched, tubular type and secrete mucus. There may be a few acid-secreting parietal cells in the region of the sphincter.

The functions of the stomach include secretion, absorption, and motor activity.

Absorption from the stomach is slight. Small amounts of water, sugars, salt solution, alcohol, and certain drugs are absorbed.

Secretion includes HCL, mucus, certain enzymes (pepsin, rennin, lipase), and a non-acid buffer secretion or alkaline component which supplies the base present in the mixed secretion - partly as chloride, and partly combined with the several buffer anions: phosphate, protein, and probably bicarbonate.

The HCl is produced by the parietal cells, and pure parietal secretion is practically only HCl and water. The concentration of HCl in pure parietal secretion is about 170 millimoles - which is numerically the same as clinical units, (0.6205%). It is secreted at a constant maximal strength.

The mucus is secreted by the surface epithelium, and also by neck mucus cells of the gastric glands and the cells of the pyloric and cardiac glands. There are two types of mucus which are chemically different, visible mucus and dissolved mucus.

The pepsin originates from the secretion of the chief cells of the gastric glands. The origin of the other enzymes is not known.

The non-acid buffer secretion apparently constitutes a mixed secretion from the glandular cells exclusive of the parietal cells.

The gastric secretory response to a meal can be divided into 3 phases: (1) cephalic phase, (2) gastric phase, and (3) intestinal phase.

Cephalic phase: The sight, smell, and taste of food set up impulses which reach the stomach by way of the vagi and stimu-

late secretion. Ivy points out that the cephalic phase of gastric secretion is caused by (a) reflexes through the cerebral cortex (psychic), and (b) reflexes through the thalamus, midbrain or medulla - the vagi being the sole pathway of the excitatory impulses. It is generally accepted that the vagus is the secretory nerve for the peptic, acid forming cells, and surface epithelial cells of the stomach (mucus forming cells).

Gastric phase: Mechanical stimulation by distention of the stomach and chemical stimulation by contact with the gastric mucosa of certain secretagogues in the food initiate the gastric phase. It is not known whether these mechanisms excite secretion by stimulating nerve endings in the stomach wall, or by causing a hormone to be produced, or by a combination of these mechanisms.

In 1906 Edkins reported that he had been able to extract from the pyloric mucosa of cats a substance which stimulated gastric secretion in the fundus when injected intravenously (gastrin). There have been objections to this work since the presence of histamine was not excluded, and it is well known that the latter is a powerful excitant of gastric secretion. It has been established, however, that a humoral mechanism for stimulating gastric secretion does exist. Ivy and Farrell showed that in dogs, a fundic pouch transplanted beneath the skin responded by secretion when the animal was fed. Whether the humoral substance is a hormone or a secretagogue is not known. Komarov has reported that he has obtained an extract from pyloric mucosa which contains no histamine and no choline and which stimulates fundic secretion.

Intestinal phase: Food or its digested products acting in the intestine stimulates gastric secretion. Since no such stimulation follows simple distention of the intestine and the latent period of gastric response is 1 to 2 hours, it is assumed that the important factor is chemical in nature. Whether it is an absorbed secretagogue or a hormone elaborated in response to the presence of food cannot be stated.

Factors which regulate gastric acidity

Since the parietal cells secrete acid of a constant strength (0.6205%), the acidity must be reduced to a considerably lower level so that the mucosa can tolerate it without injury.

1. Mathews and Dragstedt state that probably the most important single factory in reducing the acidity and pepsin concentration is the neutralizing and diluting effect of food.

2. Swallowed saliva also acts to reduce acidity of the gastric juice by dilution and by neutralization, since it possesses a considerable degree of titrable alkalinity.

3. Regurgitation of duodenal contents is known to occur (as attested by bile stained specimens of gastric content) and plays some part in control of acidity. The buffering capacities of bile, pancreatic juice, and succus entericus are well known, and the dilution factor is likewise important. The relative importance of duodenal regurgitation is debated but is emphasized by Boldyreff, Wilhelmj, and others. Hollander has pointed out, that "whether or not the duodenal fluids are effective agents in acidity reduction in the stomach, there can be no doubt that once the gastric fluid has entered the small intestine it does come under the influence of these buffer-containing secretions."

4. Another important factor in regulating the acidity of the gastric contents is the diluting and neutralizing effect of the "non-acid buffer secretion" of the stomach.

5. The significance of mucus in regulating gastric acidity is not yet known. It may or may not be important in this regard. At any rate it probably functions to protect the mucosa against mechanical and chemical irritation.

Inhibition of gastric secretion may be induced by several physiologic factors. It has been known for many years

that neutral fat in the upper intestine inhibits gastric secretion and motility. This effect has been shown to be due to the chalone, enterogastrone (Ivy and Gray), and possibly also to nervous reflexes.

Wilhelmj, O'Brien, and Hill report that increased acidity of the stomach contents inhibits the gastric phase, and Babkin states that HCl introduced with food into the duodenum inhibits gastric secretion.

The motor activity is complex and not yet completely understood. Individual variations in the normal are great. There are apparently several types of motor activity which control tone and intragastric pressure and peristaltic activity. When food is swallowed there is a receptive relaxation of the stomach in order to allow the food to enter. Then the tone progressively increases during digestion. The rate of tonus waves is usually less than three per minute. According to Gordon and Singleton, there are two types of peristaltic waves - the vigorous type involving all coats of the stomach, and a type involving mucosa and muscularis mucosae only. Peristalsis, like tone, increases as digestion progresses.

The musculature has both vagus and sympathetic innervation. The effects of stimulating these nerves is variable and depends upon the state of muscular activity present. The vagus is inhibitory to contractions of the stomach when the tone is high, and the opposite effect is obtained when the stomach is relaxed. It is usually stated, however, that the vagus is predominantly motor and the sympathetic is predominantly inhibitory. If the pyloric sphincter is contracted, relaxation may follow stimulation of either the vagus or the sympathetics.

Emptying of the stomach depends upon several factors among which are: 1) intragastric pressure, 2) intraduodenal pressure, and 3) state of relaxation or contraction of the pylorus. It is influenced by the consistency of food (fluids and finely divided solids empty most readily), by the character of the food (carbohydrates leave the stomach more rapidly than proteins, and proteins leave more rapidly than fats), by emotional factors, physical exercise, fatigue, general condition, etc. The acid-control theory of pyloric relaxation regulation gastric emptying has been largely discarded since the pylorus is apparently patent during the greater part of the time and emptying seems to be dependent on peristaltic activity and the ability of the duodenum to accept the chyme.

Etiology of Ulcer

The etiology of ulcer is obscure, and many theories have been elaborated to explain it. The most widely accepted view is that there exists some imbalance between the acid factor and the defense mechanism against the acid. However, a number of factors have been extensively studied and will be reviewed briefly.

1. Constitutional factor: Since this factor is apparently such an important one it has received intensive study from many angles.

Although Gibelli and others failed to produce ulcers by the oral administration of acid, Matthes, Langenskiold, Bolton, Ivy, and others have shown that both natural gastric juice and HCl in stengths approximating that of the acid in gastric juice are irritating to the mucosa of the stomach and intestine. Mann and Bollman have produced chronic ulcers in gastric fistula dogs by injecting 0.4% HCl by constant drip for eight hours a day over periods of several weeks.

Silbermann reported the development of ulcers in dogs which were sham fed daily for three weeks, presumably because the mucosa was exposed to the action of natural gastric juice of high free acidity undiluted by food. Schmidt and Fogelson have repeated and extended these experiments with negative results. They concluded that acid gastric juice, per se, is not sufficiently destructive to overcome the normal protective factors which inhibit or prevent gastro-duodenal ulceration.

A number of investigators, by means of transplantation experiments, have exposed the mucosa of different parts of the ali-

mentary tract, as well as parenchyma of the spleen, kidney, and other tissues to the action of gastric contents, and in general, when the circulation to the graft was intact, no digestion of the transplanted tissues occurred. By introducing a valve to prevent duodenal regurgitation, however, Matthews and Dragstedt were able to delay the healing of acute ulcers produced in the gastric mucosa.

It has been observed that ulcers which occasionally occur in a Meckel's diverticulum are almost invariably associated with ectopic gastric mucosa. Experimentally, Matthews and Dragstedt implanted small gastric pouches into the jejunum and ileum of dogs and obtained chronic, progressive ulcers in 85 to 100% of the cases depending upon the site of implantation.

Experimental ulcers have been produced by a variety of operative procedures excluding one or more of the alkaline juices. Early investigators observed occasional ulcerations when either the bile or pancreatic juice or both were excluded. Bickel (1909) was the first to carry out triple exclusion (duodeno-pancreatico-biliary exclusion) by external fistulization of the pancreatic and biliary ducts, extirpation of the duodenum, and gastro-jejunostomy. Exalto (1911) was the first to carry out the procedure of "duodenal drainage" which Mann and Williamson independently showed to be followed almost invariably by the development of a chronic ulcer. This has been confirmed by many investigators.

Histamine has also been used in the study of experimental ulcer. McIlroy reported extension of a mucosal lesion produced in the stomach of cats by cauterization after the subcutaneous injection of histamine on alternate days. Buechner, Siebert, and Molloy produced gastric erosions in rats by starving them every other day and injecting them with histamine, and Harde reported subacute and chronic ulcers in mice following a somewhat similar procedure. Buerkle de la Camp also produced acute ulcers in rats by this method and also stated that the repeated injection of histamine prolonged the healing time of acute ulcers artificially produced in the stomachs of dogs. Friedenwald, Feldman, and Morrison found opposite results. O'Shaughnessy failed to produce chronic ulcers by subcutaneous injection of histamine, but obtained lesions by injecting the drug locally into the stomach wall of cats and subsequently giving the animal large doses of histamine beneath the skin. Flood and Howes produced artificial lesions in the mucosa of the stomach of cats and found that histamine in doses of 1 to 1.2 mgm. per kilo twice a day delayed the healing of pre-pyloric defects but not of mucosal defects high on the greater curvature. Orndorff, Bergh, and Ivy injected histamine into dogs every two hours night and day for 60 days without producing ulcerations indicating that the protective factors in normal animals are sufficient to withstand even such continuous exposure to high acid gastric secretions.

3. Nervous factor: It is well known to clinicians that recurrence of an ulcer is often associated with sustained anxiety or mental distress. The mechanism of this influence is obscure, and experimentally it is difficult to study since animals do not worry. Alterations in motility, gastric secretion, and secretion of the alkaline juices (bile and pancreatic juice) have been suggested but not proved.

In addition to the undoubted deleterious influence of anxiety states, other experimental and clinical evidence lends credence to the importance of the neurogenic theory of ulcer formation as originated by Rokitansky. For example experimental ulcers have been produced by bilateral vagotomy, by splanchnic section, and by the production of a variety of brain lesions. Clinically, also, ulcers have been observed to follow brain lesions (tumors, injury, aneurysms, haemorrhage and thrombosis).

4. Traumatic factor: Aschoff emphasized the importance of mechanical factors in the development of chronic ulcers and pointed out that the location of the majority of the ulcers sustain this theory. Mann has also contributed experimental evidence indicating the importance of trauma. The consistency of the ingested food is important, as shown by Fauley and Ivy, both because of actual

mechanical injury from rough particles, and because coarse particles remain in the stomach longer than fine particles, resulting in more prolonged motor activity of the stomach, and thereby more trauma. It is also true that in cases of diaphragmatic hernia ulcers frequently develop at the site of constriction of the stomach. Violent external trauma occasionally is followed by the development of an ulcer.

5. Vascular factor: Circulatory changes have been suggested as a factor decreasing tissue resistance and, therefore, as being important in the etiology of ulcer. Boles, Riggs, and Griffiths have pointed out that alterations in circulatory efficiency may be (a) mechanical or quantitative as in cardiovascular disease, (b) nutritional or qualitative as in metabolic disturbances, anemia, blood dyscrasias and infection, or (c) functional or vasomotor.

6. Infectious factor: A number of clinicians have pointed out that foci of infection are often found associated with peptic ulcer. Rosenow has produced ulcers experimentally by injecting strains of streptococci isolated from septic foci. These strains appear to have a selective affinity for certain tissues and produce the ulcers because of this "elective localization." Others have emphasized the importance of non-specific infections.

7. Hepatic factor: Numerous observations from the laboratory and from the clinic have indicated that the liver and its secretions may be related in some way to the etiology of peptic ulcer. It is well known that spontaneous duodenal or gastric ulcer is often associated with hepatitis and liver insufficiency in the dog. Ulcers may develop in dogs with common duct obstruction, with external or internal biliary fistula, following plastic operations upon the bile ducts, after establishment of an Eck fistula or other interference with the hepatic blood supply, after partial hepatectomy, or following the administration of certain drugs which damage the liver. Observations of peptic ulcer associated with liver disease or disturbed hepatic physiology in man have been reported also.

Almquist has found a cholic acid deficiency in the gizzard lining of chicks with gizzard erosion, and the administration of bile proved effective in combating the erosive tendency. Whether or not this observation is significant as applied to human peptic ulcer is not yet known.

8. Deficiency factors: Deficiency factors have been emphasized by a number of investigators, and some experimental work has been done to support these contentions. Among the substances which have been suggested as possibly having some relationship in this regard are: Vitamin B, Vitamin C, certain essential amino acids, proteins, and certain glandular products (posterior pituitary, anterior-pituitary-like hormone, estrogenic hormone, etc.). Deficiency of protective mucin may also be an important factor. Cholic acid deficiency has already been mentioned. Up to the present, no specific deficiency factor has been absolutely established as the cause of the disease; but it is possible that impaired tissue resistance may be the result of some such deficiency.

9. Allergic factor: An allergic factor has been suggested, but is not established.

PART II

The treatment of uncomplicated peptic ulcer is medical, but the great variety of therapeutic procedures which are in use indicates that there is still no one entirely satisfactory method of treatment. Perforation and severe cicatricial obstruction are clear indications for surgical intervention. Intractability and massive hemorrhage are complications requiring the cooperative judgment of the internist and the surgeon.

In an effort to gain better orientation concerning the complications and results of therapy, the charts of 538 patients with peptic ulcer have been reviewed, and a number of clinical and laboratory investigations are now in progress under the direction of Dr.

Wangensteen.

Among the cases reviewed there were 415 males (77.1%) and 123 females (22.9%). 418 patients had duodenal ulcer, 96 had gastric ulcer, and both duodenal and gastric ulcers were present in 24 cases. The average age was 47.6 years.

In the entire group there were 76 perforations in 72 patients with 30 deaths (39%). The operative group included 50 cases with 17 deaths (34%). The remaining cases were not operated upon either because the patient was moribund when admitted, the diagnosis was incorrect, or conservative measures were believed to be advisable. The last group included cases in which the perforation was apparently walled-off.

This mortality is considerably higher than the figure of 22.8% which we have found in 2,081 collected cases from the recent literature, with 475 deaths. The probable explanation for this discrepancy is the fact that our patients coming from great distances usually arrive here relatively late. The mortality following rupture of the gastrointestinal tract is governed by several factors:

(I) The number and virulence of the escaping organisms.

1. Size of the perforation.
2. Length of time the perforation remains open.
3. Number of organisms at the level of perforation. (Length of time after ingestion of food).
4. Amount and fluidity of material in the viscus at the time of perforation.
5. Forces tending to carry contents of the viscus into the peritoneal cavity.

(II) Resistance of the host.

1. General.
2. Local - may be affected by duodenal juices.

The operation of choice in case of perforated ulcer is simple closure. Before widespread use of gastric suction as introduced by Wangensteen was adopted, it was sometimes necessary to perform gastrojejunostomy in order to relieve obstruction. Decompression can now be carried out more safely by simple suction and if obstruction persists elective operative procedures can be carried out under more favorable circumstances.

There was a history of bleeding in 186 of the 538 cases; hematemesis in 32, melena in 55, and both hematemesis and melena in 99. The bleeding was considered to be severe as judged by symptoms of syncope or shock, by rapid fall in hemoglobin, or by actual observation of massive blood loss in 83 cases. This high percentage with massive hemorrhage is explained by the fact that only the most critically ill patients have been admitted to the hospital, and most of the cases included in this review were hospital patients.

In 70 patients with massive bleeding the treatment was entirely medical. Seven of these (10%) died, and the ages in the group with fatal hemorrhage were 47, 60, 60, 60, 65, 66, and 71 years.

Thirteen patients were treated surgically when the bleeding continued in spite of medical therapy. There were 4 deaths (30.7%), and their ages were 30, 48, 54, and 80 years. One other patient, aged 24, survived for several months and subsequently died of a perforated jejunal ulcer. The operative procedures were: gastric resection, 8 cases with 2 deaths; gastroenterostomy, 4 cases with 1 death; and ligation of the bleeding vessel in 1 case with one death. It should be pointed out that the patients treated surgically were essentially those who had already been treated medically but who, the staff believed, would not survive unless surgical therapy were undertaken.

It is interesting that our mortality statistics agree almost exactly with figures which we have collected from the recent literature. In 2565 collected cases of massive hemorrhage treated medically there were 263 deaths (10.2%) and in 214 collected cases treated surgically there were 64 deaths (29.8%).

It is obvious that when a large vessel has been eroded and continues to bleed, conservative therapy cannot succeed. Persistent bleeding is especially common and exceedingly dangerous in elderly individuals with sclerotic vessels. The indications for surgery in cases of massive hemorrhage still constitute a moot question. Rankin has advised that operation be undertaken when:

1. Massive hemorrhage occurs in a known peptic ulcer patient over 50 years of age.

2. Massive hemorrhage occurs in a known peptic ulcer of long standing which has failed to respond to careful, continuous medical treatment.

3. Massive hemorrhage follows one or more previous episodes of severe bleeding from a peptic ulcer; and

4. Massive hemorrhage from a known ulcer of long standing or in an arteriosclerotic patient fails to respond to one or two transfusions and there is evidence of continuous or repeated bleeding.

If a surgical procedure is to be undertaken for hemorrhage, it is best to operate within the first 48 hours. Delay beyond that time greatly increases the risk. Incomplete procedures such as an attempt to ligate the bleeding vessel, gastroenterostomy, etc. are inadequate. The operation of choice is excision of the ulcer together with extensive gastric resection, but in many cases the poor condition of the patient necessitates the use of less radical resection.

There were 138 patients with gastroenterostomy or gastric resection for ulcer with 14 cases of anastomotic ulcer. In the group of 100 patients who had had their operation here (38 patients with gastroenterostomy elsewhere), there were 5 with anastomotic ulcer (5%).

It is impossible to judge the true incidence of stomal ulcers in the group since some of our cases may have developed them without our having an opportunity to secure further information concerning the complication. Others might develop a stomal ulcer at a later date.

The seriousness of this complication is apparent from the fact that the stomal ulcer led to the death of 6 of these 14 patients (42.8%). Four of them died following operation, a fifth died from perforation, and the sixth from hemorrhage. In the entire group of 538 patients, deaths from gastrojejunal ulcer constituted 1.1%.

Gastroenterostomy

Gastric acidity studies have been carried out in 21 patients with gastroenterostomy for ulcer (19 posterior, and 2 anterior gastroenterostomies). Histamine (0.5mg) was used to stimulate gastric secretion and 4 aspirations were carried out at half-hour intervals. Acid neutralization tests were also performed. 150 cc. of 0.5% HCl was introduced into the stomach and 15 cc. specimens were withdrawn at 15 minute intervals for 90 minutes. At the last aspiration all of the fluid remaining in the stomach was withdrawn. The samples were titrated for free and total acidity using Topfer's solution and phenolphthalein as indicators. The reduction of acidity during the period of observation thus was measured. The results are listed in Table I.

The effect of gastroenterostomy was found to be variable, but there was often a decreased acidity and, in 5 cases (three with duodenal and two with gastric ulcer), even achlorhydria. (See Table I.)

Snell states that, as a rule, there is a permanent reduction of 30 to 50% in gastric acidity after gastroenterostomy. Holman and Sandusky, on the other hand, reviewed the literature and carried out studies in 75 gastroenterostomy patients, and concluded that gastric acidity was not appreciably altered by gastroenterostomy in 92% of the patients. Acid loss, as estimated from the neutralization test, was rapid. This confirms the work of Elman who found that there was delayed neutralization in all cases of duodenal ulcer, but that after gastroenterostomy the neutralization was more rapid even than in the normal.

TABLE I

PATIENTS HAVING GASTROENTEROSTOMY

CASE NO. AGE DATE OF OPERATION	DATE	FASTING FREE ACID	MAXIMUM FREE ACID WITH HISTAMINE	MAXIMUM TOTAL ACID WITH HISTAMINE	VOLUME OF SECRETION	ACID OUTPUT mgm/1/2h FREE	ACID OUTPUT mgm/1/2h TOTAL	ACID NEUTRALIZ. % ACID LOSS	DUODENAL REGURGITATION PSP: Ba	MOTILITY EMPTYING TIME Ba
#1 53 - D.U. 11/25/38	2/8/40	48°	50°	68°	14cc	25mg	35mg	92%	34% PSP supine	
#2 67 - D.U. 7/25/34	1/22/34	51°	-	84°						
	2/8/40	60°	37°	77°	4.6cc	6.3mg	13mg	99%		
#3 58 - D.U. 12/21/39	12/20/39	64°	96°	110°	21cc	68mg	79mg	89%		
	1/20/40	0°	25°	55°	5.8cc	4.7mg	10mg	83%	5% Ba erect Duo.empty.20min.	
#4 29 - D.U. 10/25/38	9/8/38	44°	-	56°						
	2/19/40	0°	0°	20°	2.3cc	0mg	1.5mg	96%	75% Ba erect	1 hour
#5 G.U., 71 - D.U. 11/5/35	8/23/35	0°	54°	70°	9cc	18mg	23mg			
	2/21/40	13°	50°	60°	22cc	40mg	48mg	86%		1hr.55min.
#6 40 - D.U. 1927	1/23/40	20°	20°	40°	23cc	16mg	33mg	94%		3hr.15min.
#7 30 - D.U. 12/28/38	10/6/38	80°	-	104°						
	2/7/40	68°	84°	94°	16cc	49mg	54mg	88mg	2.5% PSP 0% Ba erect Duo.empty,25min.	40 min.
#8 59 - D.U. 1919	10/24/39	0°	0°	32°	12cc	0mg	14mg	93%	2.6% PSP	
#9 55 - G.U. 9/30/36	9/15/36	0°	75°	85°	10cc	28mg	31mg			
	2/20/40	50°	98°	110°	14cc	50mg	56mg	67%		1 hour

(Over)

TABLE I (Cont.)

PATIENTS HAVING GASTROENTEROSTOMY

CASE NO. AGE DATE OF OPERATION	DATE	FASTING FREE ACID	MAXIMUM FREE ACID WITH HISTAMINE	MAXIMUM TOTAL ACID WITH HISTAMINE	VOLUME OF SECRETION	ACID OUTPUT mgm/1/2h FREE	ACID OUTPUT mgm/1/2h TOTAL	ACID NEUTRALIZ. % ACID LOSS	DUODENAL REGURGITATION PSP: Ba	MOTILITY EMPTYING TIME Ba
#10										
39- D.U.	3/28/35	10°	117°	130°						
12/19/38	2/21/40	0°	12°	22°	24cc	10mg	19mg	97%	Trace PSP erect 0% Ba supine Duo.empty,32min.	
#11										
69- G.U.	4/28/36	0°	22°	82°	25cc	20mg	75mg			
7-28-37	2/21/40	0°	0°	22°	9.8cc	0mg	11mg	98%		1 hour
#12										
73- G.U.	5/10/38	24°	-	38°						
7/13/39	10/24/39	0°	0°	18°	18cc	0mg	12mg	94%	7.6%PSP erect 0% Ba erect or supine Duo.empty,18min.erect 43min.supine	30 min.
#13										
50- D.U.	6/15/37	30°	-	52°						
8/18/37	2/19/40	15°	27°	40°	4.4cc	4.3mg	5.5mg		3% PSP	15 min.
#14										
60- D.U.	2/6/40	14°	80°	98°	11cc	32mg	40mg	88%		30 min. (all thru stoma)
#15										
35- D.U.	4/14/39	15°	-	37°					Trace PSP erect 0% Ba erect 95% Ba supine	
1929										
#16										
50- D.U.	12/21/39	55°	65°	96°	18cc	42mg	48mg	88%		
12/22/39	2/5/40	0°	38°	72°	2.8cc	4mg	8mg	69%		
#17										
51- D.U.	10/31/39	28°	86°	100°	67cc	210mg	245mg	45%		
#18										
54- D.U.	1/29/38	37°	71°	80°	17cc	44mg	51mg			
1/31/39	3/5/40	18°	18°	22°	28cc	27mg	22mg	100%	Trace PSPerect 95% Ba supine	1h,25min.

(Over)

TABLE I (Cont.)

PATIENTS HAVING GASTROENTEROSTOMY

CASE NO. AGE DATE OF OPERATION	DATE	FASTING FREE ACID	MAXIMUM FREE ACID WITH HISTAMINE	MAXIMUM TOTAL ACID WITH HISTAMINE	VOLUME OF SECRETION	ACID OUTPUT mgm/1/2h FREE	TOTAL	ACID NEUTRALIZ. % ACID LOSS	DUODENAL REGURGITATION PSP: Ba	MOTILITY EMPTYING TIME Ba
#19 40- D.U. 6/1937	3/5/40	0°	0°	100°	5cc	0mg	18mg	90%		1hr.35min.
#20 50- D.U. 9/9/37	6/4/37	20°	-	40°						
	12/8/39	0°	36°	68°	3.1cc	3.8mg	7.5mg	96%		14 min.
#21 59- D.U.	2/23/40	12°	56°	74°	18cc	37mg	49mg	93%	Trace PSP erect 5% Ba erect 30% Ba supine	

Elman regarded regurgitation of intestinal juices through the stoma as the most important factor in the more rapid acid loss after gastroenterostomy. In order to study the regurgitation factor, several patients were examined by passing a duodenal tube through the pylorus into the descending portion of the duodenum, and observing the amount of regurgitation into the stomach after the introduction of barium or phenolsulphonphthalein into the duodenum. The amount of barium regurgitation was estimated from roentgenoscopic examination (in collaboration with Dr. Nessa), and the PSP regurgitation was estimated colorimetrically on specimens withdrawn at intervals of 2, 4, 6, 8, 10, 20, 40, and 60 minutes. The results, shown in Table I, were variable. The barium studies appeared to be the more reliable. Position exerted a definite effect. When a patient was in the supine position regurgitation was much greater than when he was erect. Similar studies were performed on two dogs after gastroenterostomy, and these showed comparable results. (Table II.)

TABLE II. Regurgitation from Duodenum in Dogs with Gastroenterostomy

Position of dog	% PSP in stomach in 1 hour
Prone (standing on four legs)	8.5%
Standing on hind legs . . .	7.0%
Lying on right side	12.5%
Lying on left side	15.5%

Gastroscopists have observed regurgitation of bile-stained fluid into the stomach through a gastroenterostomy stoma to be much more profuse than reflux through the normal pylorus. They also frequently have noted the development of a postoperative gastritis with an oedematous, inflamed mucosa, which Schindler suggests may be due to the action of regurgitated intestinal juices. Atrophic gastritis occurs less frequently.

It is common knowledge that regurgitation through the pylorus is a normal event. Bile-stained gastric content is frequently obtained by aspiration, and gastroscopists actually have observed such regurgitation.

When cholangiography is carried out in patients with choledochostomy, but without other apparent gastrointestinal disturbances, the contrast medium often is seen to have entered the stomach. In 57 such cases reviewed by Dr. Nessa, the films showed opaque medium in the stomach in 12 cases (21%). Evidence at hand, however, indicates that regurgitation is probably increased after gastroenterostomy

Kirklin has pointed out that there is usually a reduction of gastric emptying time after gastroenterostomy, but this is an individual matter and no standards can be set for it. Our observations in 14 cases confirm this statement. The emptying time (150 cc. of a thin barium sulphate suspension at room temperature) varied from 7 to 195 minutes and the average was 59 minutes (average in normals was 83 minutes).

Gastric Resection

(1). Antral resections:

Similar studies have been carried out in 5 patients with antral resection (Polya type) for ulcer, and the results are listed in Table III. All of these were done as emergency operations for hemorrhage either (a) supplementary to a previous gastroenterostomy, or, (b) as primary operations for bleeding. None of the patients who had had free acid before operation became achlorhydric to histamine stimulation after antral resection, but all showed a decreased acid secretion. These results agree with the findings of other investigators, except that some cases have been reported to develop achlorhydria after antral resection [81].

The explanation for the reduced acidity after antral resection is not entirely clear since relatively little of the acid-secreting portion of the stomach is removed by this procedure. In the light of the experimental work of Ivy and others, as well as the work of Wangensteen, Varco, et al in our own

TABLE III

PATIENTS HAVING ANTRAL RESECTION

CASE NO. AGE DATE OF OPERATION	DATE	AMOUNT* RESECTED	FASTING FREE ACID	MAXIMUM FREE WITH HISTAMINE	MAXIMUM TOTAL WITH HISTAMINE	VOLUME OF SECRETION 1/2 hour	ACID OUTPUT mgm 1/2 hr. FREE	ACID OUTPUT mgm 1/2 hr. TOTAL	ACID NEUTRALIZ. % ACID LOSS	MOTILITY EMPTYING TIME Ba
#22										
56- D.U.	6/28/39		15°	82°	91°	12cc	36mg	40mg		
9/18/39	9/30/39	65%	0°	62°	78°	35cc	79mg	100mg	72%	
	11/7/39	117sq.cm.	0°	40°	116°	90cc	135mg	384mg	18%	5hr.10 min.
#23										
41- D.U.	7/9/39		27°	-	39°					
7/10/39	11/14/39	32%	24°	56°	88°	26cc	49mg	67mg	82%	40 min.
	2/15/40	143sq.cm.	20°	56°	64°	22cc	45mg	51mg	84%	
#24										
50- D.U.	8/29/39		25°	-	71°					
9/3/39	11/8/39	60%	0°	0°	60°	17cc	0mg	33mg	58%	
	2/21/40	130sq.cm.	0°	18°	36°	11cc	0mg	7mg	79%	2hr.35 min.
#25										
49- D.U.	1/20/34		0°	53°	63°					
G.E.- -11/14/38 Ant.Res.- 12/17/38	2/26/40	52% 138 sq.cm.	0°	34°	52°	13cc	15mg	25mg	95%	30 min.
#26										
24- D.U.	12/1/38		37°	80°	100°					
G.E.- -2/14/39 Ant.Res.- -2/21/39	5/8/39	150 sq.cm.	31°	38°	50°	13cc	18mg	23mg		

*Where actual measurements are not given, the amount resected is estimated from planimeter measurements of x-rays. This method is inaccurate not only because of differences in filling but also because the remaining stomach dilates.

laboratory, Edkins' theory of gastrin stimulation of the parietal cells is not generally accepted. St. John, Flood, and Guis suggest that the reduced acidity is probably due to the abolition of the chemical phase of gastric secretion as a result of accelerated gastric emptying, and also to regurgitation of the alkaline intestinal juices. The development of a postoperative gastritis may also actually decrease the amount of acid secreted. It has been suggested that the operative interference with the blood supply to the stomach may account for the decreased acidity. Results of experiments in our laboratory (in collaboration with Dr. Layne) contradict this theory, since three of the four main arteries can be ligated without decreasing the acid secreted or changing the results of the neutralization test.

Elman and MacLeod found that three out of four cases with gastric resection showed very rapid neutralization rates. In 4 of our 6 cases the rate of neutralization was increased.

According to Meyer and Schmitt, Vitkin, and others, the gastric emptying is more rapid after gastric resection than in the normal, and the type of resection is immaterial. In four of our cases of antral resection, two showed fairly rapid emptying, one emptied somewhat slowly, and one showed delayed emptying. The last had the highest acid values, and neutralization was delayed.

(II). Extensive gastric resections:

Extensive resections have been done in 5 patients; four with gastric ulcer and one with duodenal ulcer. It might be expected that this procedure would effectively reduce gastric acidity since it removes a considerable portion of the acid secreting glands. The cases listed in Table IV confirm this assumption, three of them becoming achlorydric to histamine after the operation, and a fourth having a relatively low acid secretion. The fifth case was achlorhydric on one examination before operation. Even a high resection, however, may fail to produce achlorhydria as is indicated by one case in our group and also by two cases reported by St. John, Flood, and Guis. Finsterer, however, reports excellent results after extensive resection (at least two-thirds of the stomach being resected) and states that in more than 1500 cases only one patient had to be reoperated upon for gastrojejunal ulcer.

The neutralization tests showed very rapid acid loss in these patients, even more marked than in the cases of antral resection.

Gastric emptying time was measured in one of the three cases with extensive resection and in this case the stomach emptied the test meal in 10 minutes.

The emptying time following gastric resection is variable. It may be normal or delayed, but in the majority of cases it is reduced and is usually shorter even than in the case of gastroenterostomy.

(III). Extensive resection with antral exclusion:

In two patients, extensive gastric resection with antral exclusion (Finsterer operation) was done. The operation was followed by achlorhydria in both instances. The neutralization test was carried out in one case and showed very rapid acid loss. (Table V).

(IV). Schmilinsky Operation:

In three cases with gastrojejunal ulcers following gastroenterostomy, a partial gastrectomy was performed, and the afferent and efferent intestinal loops were implanted into the stomach at some distance apart so that complete intragastric regurgitation of the duodenal contents was provided. None of these developed achlorhydria, but the acid values were reduced in each case and the anastomotic ulcers healed. (Table VI.) In spite of this, two of the three cases later developed new jejunal ulcers with perforation in one and massive hemorrhage in the other. The third patient has remained well for 5 years.

TABLE IV

PATIENTS HAVING EXTENSIVE GASTRIC RESECTION

CASE NO. AGE DATE OF OPERATION	DATE	AMOUNT* RESECTED	FASTING FREE ACID	MAXIMUM FREE WITH HISTAMINE	MAXIMUM TOTAL WITH HISTAMINE	VOLUME OF SECRETION 1/2 hour	ACID OUTPUT mgm 1/2 hr. FREE	ACID OUTPUT mgm 1/2 hr. TOTAL	ACID NEUTRALIZ. % ACID LOSS	MOTILITY EMPTYING TIME Ba
#27										
49- G.U.	5/5/38		20°	45°	54°	15cc	24mg	29mg		
3/4/39	3/4/39		0°	0°	18°	18cc	0mg	5.3mg	97%	
	10/18/39	70%	8°	0°	22°	7cc	1.3mg	5.6mg	95%	
	2/20/40	200sq.cm.	0°	0°	16°	3cc	0mg	1.7mg	95%	10 min.
#28										
44- D.U.	6/11/36		44°	-	72°	5cc	8mg	13mg		
	10/24/39	53%	0°	0°	38°	15cc	0mg	20mg	90%	
#29										
67- G.U.	12/30/39		26°	36°	63°	20cc	26mg	46mg	45%	
1/4/40	1/17/40	70%	0°	0°	45°	15.3cc	0mg	15mg	98%	
	1/30/40	200sq.cm.	0°	18°	44°	18cc	8.5mg	29mg	100%	
#30										
58- G.U.	6/15/39		26°	-	44°					
7/3/39	11/9/39		0°	0°	38°	11cc	0mg	15mg	90%	
#31										
53- G.U.	2/1/40		0°	0°	14°					
2/27/40	3/5/40	290sq.cm.	0°	0°	34°	12cc	0mg	15mg		

*Where actual measurements are not given, the amount resected is estimated from planimeter measurements of x-rays. This method is inaccurate not only because of differences in filling but also because the remaining stomach dilates.

TABLE V

EXTENSIVE GASTRIC RESECTION WITH ANTRAL EXCLUSION (FINSTERER OPERATION)

CASE NO. AGE DATE OF OPERATION	DATE	AMOUNT* RESECTED	FASTING FREE ACID	MAXIMUM FREE WITH HISTAMINE	MAXIMUM TOTAL WITH HISTAMINE	VOLUME OF SECRETION 1/2 hour	ACID OUTPUT mgm 1/2 hr. FREE	ACID OUTPUT mgm 1/2 hr. TOTAL	ACID NEUTRALIZ. % ACID LOSS	MOTILITY EMPTYING TIME Ba
#32										
38- D.U.	4/20/37		14°	-	22°					
5/13/37	6/6/39	60%	0°	0°	18°	10cc	0mg	6.5mg	88%	10 min.
	2/14/40		0°	0°	16°	35cc	0mg	20mg	85%	
#33										
36- G.U.	10/14/39	54°	-	58°						
	2/18/40	42°	-	63°						
2/26/40	3/7/40	195sq.cm.	0°	0°	44°	4.3cc	0mg	7mg		

*Where actual measurements are not given, the amount resected is estimated from planimeter measurements of x-rays. This method is inaccurate not only because of differences in filling but also because the remaining stomach dilates.

TABLE VI

PATIENTS HAVING THE SCHMILINSKY OPERATION IN STAGES

CASE NO. AGE DATE OF OPERATION	DATE	AMOUNT* RESECTED	FASTING FREE ACID	MAXIMUM FREE WITH HISTAMINE	MAXIMUM TOTAL WITH HISTAMINE	VOLUME OF SECRETION 1/2 hour	ACID OUTPUT mgm 1/2 hr. FREE	ACID OUTPUT mgm 1/2 hr. TOTAL	ACID NEUTRALIZ. % ACID LOSS	MOTILITY EMPTYING TIME Ba.
#34										
49- D.U.	4/19/35		16°	70°	82°	67cc	168mg	198mg		
G.E.-1918	12/31/36	43%	0°	0°	23°					
Ant.Res.- -4/20/35										
Schmilinsky 1/8/36	1/5/39		0°	9°	39°	12cc	5.9mg	26mg		
#26										
24- D.U.	12/1/38		37°	80°	100°					
G.E.- -2/14/39										
Ant.Res.- -2/21/39	5/8/39	150sq.cm.	31°	38°	50°	13cc	18mg	23mg		
Schmilinsky	6/28/39		24°	42°						
5/15/39	8/29/39		0°	26°	40°	15cc	14mg	22mg		
#35										
35- D.U.	9/5/35		69°	-	90°					
G.E.- -1/1938	4/14/39		89°	-	102°					
Fundusectomy	6/22/39	52%	0°	56°	60°	6cc	12mg	13mg		
4/14/39	7/15/39	336sq.cm.	0°	42°	96°	11cc	17mg	38mg		
Schmilinsky 8/18/39	9/28/39	70% 125 sq.cm.	5°	0°	21°					

*Where actual measurements are not given, the amount resected is estimated from planimeter measurements of x-rays. This method is inaccurate not only because of differences in filling but also because the remaining stomach dilates.

(V). Fundusectomy:

Since the antral portion of the stomach secretes little acid, and since the importance of this region as the site of formation of a secretory hormone for gastric secretion (as suggested by Edkins) is extremely doubtful, an excision of a portion of the fundus without removing the antrum has been done in an attempt to reduce gastric acidity. Animal experiments have been carried out by Connell, Watson, Seely and Zollinger, and Fauley and Ivy, in which either a V-shaped sector was removed, or the entire greater curvature side of the fundus was excised. There was usually an initial fall in gastric acidity followed by a gradual rise to normal or near normal levels after 4 to 8 months. Connell also applied fundusectomy to 14 human patients with 2 deaths, 1 recurrent ulcer, and 11 good results, in spite of the fact that the gastric size and secretion had returned to approximately normal levels within 6 months.

In 8 cases (Tables VII and VIII), Dr. Wangensteen has performed extensive fundic excision, leaving only a narrow tube along the lesser curvature in the corporic zone. Five of these patients also have had a gastrojejunal anastomosis (Table VII). Three of the patients with fundusectomy and gastrojejunostomy became achlorhydric to histamine after operation (two of them were last tested 10 months after the procedure), and the remaining 2 developed reduced acidity (Table VII). The three patients with fundusectomy without gastrojejunostomy (Table VIII) developed reduced acidity but not achlorhydria. In 4 of the 5 cases in which neutralization tests were performed the acid loss was rapid. After operation, the gastric emptying time was decreased to 6 minutes in one case, in another case the motility was approximately normal, and in one case emptying was delayed.

Subphrenic vagotomy has been done in addition to fundusectomy in 2 cases. Both of these still secrete free acid after histamine stimulation, but the level of acidity is greatly reduced. Wilhelmj et al have reported that in the experimental animal vagotomy plus partial gastrectomy produces a greater decrease in acid secretion than gastrectomy alone. Winkelstein and Berg have reported similar experiences in human patients. On the other hand, chronic gastric ulcers have been produced in the rabbit and in the dog by bilateral subdiaphragmatic vagotomy.

It is apparent that these groups of cases are too small to allow drawing any definite conclusions, but it appears that extensive gastric resection is the most effective operation for reducing gastric acidity. Fundusectomy is somewhat less effective but is technically more simple to perform. Judging from our cases, antral resection and the Schmilinsky operation are not suitable procedures for the relief of ulcer. Gastroenterostomy does at effectively reduce acidity, but it does promote more rapid emptying and increases regurgitation of intestinal fluids into the stomach. In well selected cases it is followed by a high percentage of good results and is still widely used, especially in cases with obstruction or in cases where more extensive procedures are contraindicated.

It is generally stated that it is more difficult to produce achlorhydria by operation in younger individuals than in elderly patients. Considering the age factor in all of our patients operated upon, the average age of the group with achlorhydria was 49.2 years, while the average age of those still showing free acid after operation was 44.9 years.

Summary

1. Much remains to be learned concerning the etiology of peptic ulcer.

2. Perforation and cicatricial obstruction require surgical treatment. Massive hemorrhage and intractability sometimes constitute indications for surgery and require the cooperative judgment of both internist and surgeon.

3. In 50 cases operated upon for perforated ulcer, the mortality was 34%. If the patients could be seen earlier this figure undoubtedly would be greatly reduced.

TABLE VII

PATIENTS HAVING FUNDUSECTOMY AND GASTROJEJUNOSTOMY

CASE NO. AGE DATE OF OPERATION	DATE	AMOUNT* RESECTED	FASTING FREE ACID	MAXIMUM FREE WITH HISTAMINE	MAXIMUM TOTAL WITH HISTAMINE	VOLUME OF SECRETION 1/2 hour	ACID OUTPUT mgm 1/2 hr. FREE	TOTAL	ACID NEUTRALIZ. % ACID LOSS	MOTILITY EMPTYING TIME Ba
#36										
43- G.U.	11/4/37		0°	36°	40°					
4/20/39	3/5/39		0°	30°	42°					
	5/1/39	47%	0°	0°	24°	7cc	0mg	6.1mg		
	8/10/39	252 sq.cm.	0°	0°	19°	10cc	0mg	6.9mg		
	2/20/40		0°	0°	30°	2cc	0mg	2.2mg		40min.
#37										
61- D.U.	9/14/39		41°	108°	116°	11cc	43mg	47mg		
9/19/39	9/29/39	253 sq.cm.	24°	30°	60°	18cc	20mg	39mg		
	10/5/39		22°	42°	64°	16.3cc	25mg	38mg	32%	
#38										
67- G.U.	5/10/39		35°	54°	94°	29cc	47mg	94mg		
5/11/39	5/18/39	53% 270 sq.cm.	0°	0°	17°	17cc	0mg	10mg		
#39										
63- D.U.	3/24/39		28°	-	36°					
3/24/39	4/3/39	52%	0°	10°	22°	7cc	2.5mg	5.6mg		
	2/13/40	180sq.cm.	0°	8°	16°	3.5cc	1mg	2mg	94%	3hr.10min.
#40										
31- D.U.	4/18/39		33°	62°	74°	18cc	22mg	49mg		
4/19/39	8/29/39	32%	0°	0°	14°	15cc	0mg	7.6mg		
	11/14/39	193sq.cm.	0°	0°	48°	3cc	0mg	5.2mg		
	2/6/40		0°	0°	48°	3.1cc	0mg	5.4mg		

*Where actual measurements are not given, the amount resected is estimated from planimeter measurements of x-rays. This method is inaccurate not only because of differences in filling but also because the remaining stomach dilates.

TABLE VIII

PATIENTS HAVING FUNDUSECTOMY WITHOUT GASTROENTEROSTOMY

CASE NO. AGE DATE OF OPERATION	DATE	AMOUNT* RESECTED	FASTING FREE ACID	MAXIMUM FREE WITH HISTAMINE	MAXIMUM TOTAL WITH HISTAMINE	VOLUME OF SECRETION 1/2 hour	ACID OUTPUT mgm 1/2 hr. FREE	ACID OUTPUT mgm 1/2 hr. TOTAL	ACID NEUTRALIZ. % ACID LOSS	MOTILITY EMPTYING TIME Ba
#41										
31- D.U.	1/17/40		34°	54°	94°	19cc	55mg	65mg	41.5%	
1/18/40	1/30/40	45% 300sq.cm.	0°	34°	52°	19cc	23mg	36mg	78%	1hr.50min.
#42										
31- D.U.	10/4/39		82°	114°	130°	62cc	257mg	293mg	7.7%	
10/9/39	10/24/39	45%	0°	20°	38°	46cc	33mg	63mg	91%	
	11/24/39	442sq.cm.	0°	49°	86°	14cc	25mg	44mg	90%	
	12/28/39		0°	60°	90°	17cc	37mg	55mg	85%	
#43										
29-D.U.	2/6/40		64°	74°	88°	26cc	70mg	84mg	77%	4 hr.
2/13/40	2/26/40	28% 375sq.cm.	0°	30°	74°	24cc	26mg	65mg		

*Where actual measurements are not given, the amount resected is estimated from planimeter measurements of x-rays. This method is inaccurate not only because of differences in filling but also because the remaining stomach dilates.

4. The mortality in 83 cases of massive hemorrhage was 10% in cases treated medically and 30% in cases treated surgically. However, only the most serious cases have been treated by operation so these figures cannot be compared. Earlier surgical intervention in selected cases might be advisable, but the indications for operation still constitute a moot question.

5. In 138 patients with gastrojejunostomy there were 14 anastomotic ulcers. This figure includes cases in which the anastomotic operation was done elsewhere. In our own operative group the incidence of stomal ulcer was 5%.

6. Gastroenterostomy is followed by increased intragastric regurgitation of duodenal juices, decreased gastric emptying time, and rapid acid loss. The effect on acidity is variable, but there is often a decreased acidity and, in some cases, even achlorhydria.

7. Extensive gastric resection is more effective than antral resection in producing achlorhydria. Both operations promote rapid emptying of the stomach.

8. Fundusectomy is somewhat less effective than high gastric resection for the purpose of reducing gastric acidity. It has the advantage, however, of being technically easier.

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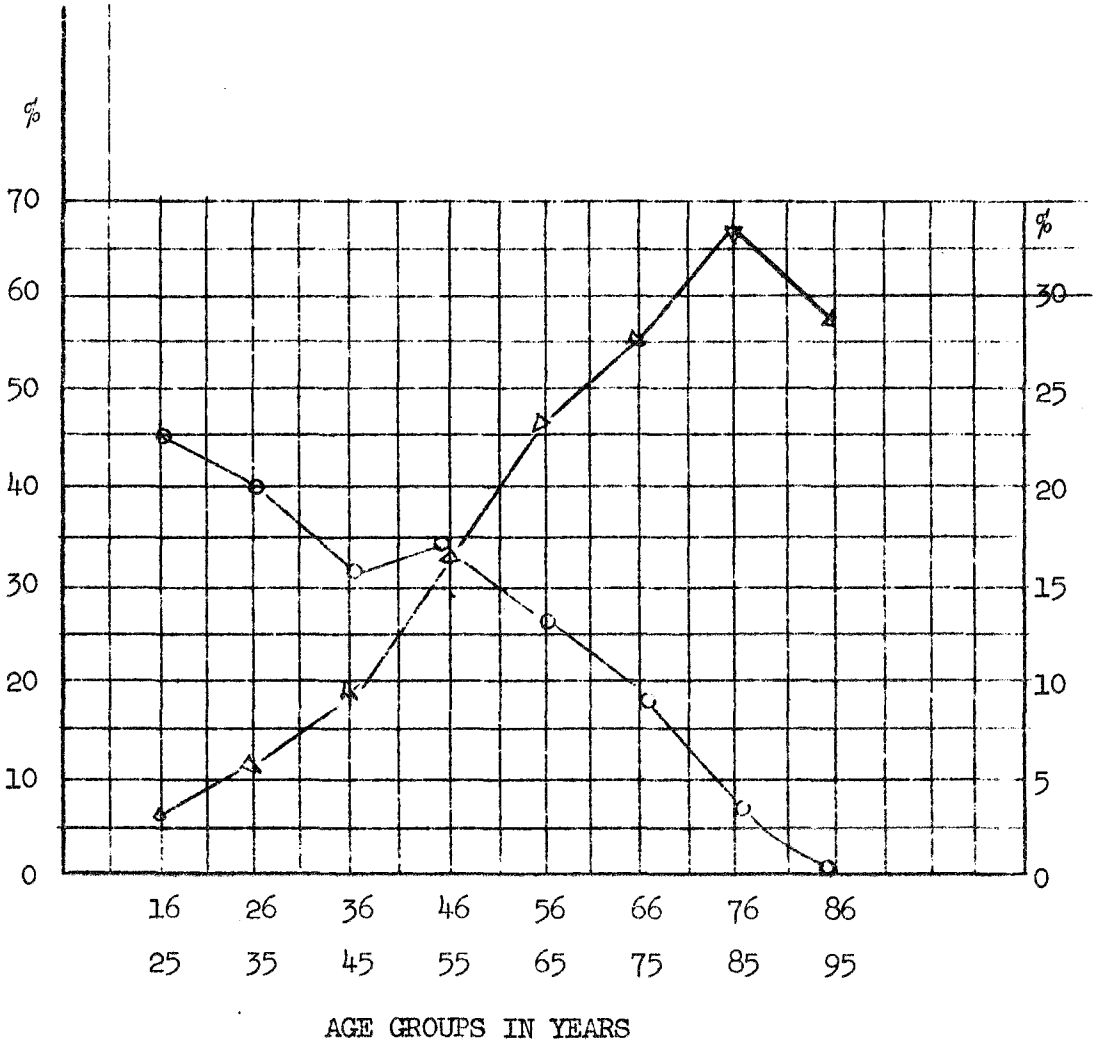
V. ADDITION - TO LAST WEEK'S ABSTRACT

"Fluoroscopy of the Chest"

TOTAL - 7,114 Cases at University of Minnesota Hospitals

△——△ Shows percentage with Fluoroscopic findings in each age group (Lt. % scale)

o——o Shows percentage of total Patients in each age group (Rt. % scale).



VI. GOSSIP

Dental Hygienist Marjorie Gormican has a unique collection of toothbrushes on display today. The large tree in the meeting room is decorated with a collection of brushes brought by our patients. The Admissions Department writes to all in-coming patients urging them to bring a toothbrush and bed clothing. The 200 brushes hanging on the tree include eyebrow brushes, brushes for gas stoves, pipe cleaners, milk bottle brushes and nail brushes. In addition, many of them are the "family" toothbrush; others are brushes which are used otherwise for cleaning the false teeth. The notes with these brushes include many interesting items. This display was first shown at the annual meeting of the Minnesota State Dental Association in St. Paul February 27-29... Medical nurse Nan Fleming was surprised on her birthday, March 13, by a large noon party in the special dining room. Miss Fleming was so overcome when her friends brought her in and presented her with a cake with the customary 16 candles on it that for once she was at loss for words. There was the suggestion that the whole hospital should have participated in the party for our favorite one. A move was made to have someone sing over the loud speaker system "Happy Birthday to You", but was abandoned because of possible action by Western Union. Many more of them!....Ophthalmologist Edward S. Murphy and Health Service physician Meredith Hesdorffer of Missoula, Montana have organized a Staff Meeting Club. They take our Bulletin, abstract the material and present it at their meetings. There is a group of loyal Minnesotans and other followers of our group out there who keep up with us and our doings. Congratulations for a splendid idea to Drs. Murphy and Hesdorffer....Dr. Churchill, our distinguished guest today, is another one of those small-town boys who make good in the big city. He came from Chenoa, Illinois, the second town away from ours, and my aunt gave him music lessons. We had quite a fanning-bee talking over old times back in Illinois....At the Minneapolis Marches On broadcast the other evening on WTCN, the speakers represented medicine, dentistry, nursing, public health, and the hospitals. An amazing array of information concerning the health possibilities of Minneapolis was presented. Yours Truly conducted the various interviews. My opportunity came at the end when they asked me what I thought medicine had in store for Minneapolis during the next ten years. I was forced to admit that I thought the Center for Continuation Study would play a very important part in medical progress....The Minnesota State Medical Association will meet in Rochester April 22, 23, and 24. It will be held in the new Mayo Civic Auditorium and there will be plenty of room for various demonstrations and meetings. One of the highlights will be the series of round tables which will be conducted during the noon hours of the three days. The first day's program will be exclusively presented by the Mayo Clinic.. ..Harold J. Dvorak, one time graduate student at the University of Minnesota, is a member of the Continuation Course in Surgery group this week. After leaving Minnesota, Dr. Dvorak spent some time in study in Prague and other European countries. He is now located in Milwaukee, his former home.....The surgical colloquia have been an outstanding success this week. Dr. Churchill proved to be an ideal leader of such discussions. It is probable that we will have many more such informal presentations...Surgeon Charles Rea is back after a whirl at private practice in New Ulm.. Charlie took a little more time off and went to Hot Springs. Down in Illinois the towns people used to call it going down to get boiled out....A recent report on College Health Service practices in 549 colleges by Drs. Diehl and Charles E. Shepard showed that over half of the entrance examinations did not include a simple test of vision. Only 29 institutions tested hearing with the audiometer and only two gave Wasserman tests to incoming students. The report was published in April 1939...Health Service in industrial plants is becoming very popular. Many of our well trained graduates are participating in these programs. The examinations are made before employment and at periodic intervals afterward. Patients go to private physicians for care and the companies and the employees band together for funds to pay medical and hospital bills. In the Minneapolis Public and Parochial school systems every child receives a dental examination once a year. Reference to a dentist is made....