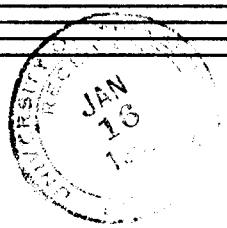

Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation

Tubular Gastric Resection: A Preliminary Evaluation

Volume XXIV | Friday, January 9, 1953 | Number 12

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UNIVERSITY OF MINNESOTA HOSPITALS
and
MINNESOTA MEDICAL FOUNDATION

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Published weekly during the school year, October to June, inclusive.

The Bulletin is sent to members of the Minnesota Medical Foundation.
Annual membership fee - $10.00.

Address communications to: Staff Bulletin, 3330 Powell Hall, University of Minnesota, Minneapolis 14, Minn.

I. TUBULAR GASTRIC RESECTION: A PRELIMINARY EVALUATION

Lloyd D. MacLean, M.D.

This report is a preliminary evaluation of tubular gastric resection as performed for peptic ulcer in this hospital over the last 12-month period. The results have been compared to the results obtained in 893 patients who underwent Billroth II type subtotal gastrectomy from 1940 through 1949[11], and to 90 patients who underwent segmental gastric resection from 1949 through 1951[12]. Fifty-one patients received tubular resection between January and November 1952.

Historical Background:

The foundation of modern gastric surgery was laid by Theodore Billroth in the 10-year period, 1874-1885. As early as 1874 Gussenbauer and von Winiwarter[1], working in Billroth's laboratory, had performed pylorectomy successfully on dogs. Careful planning and further experimentation culminated in the first successful partial gastrectomy in 1881 by Billroth[2]. Later the same year Woelfler, also an assistant of Billroth, performed the first gastroenterostomy in a patient who had an obstructing but unresectable carcinoma. Rydygier performed the first gastroenterostomy for an obstructing peptic ulcer in 1884. Billroth introduced gastric resection with gastrojejunostomy the following year in 1885.

Many modifications of these three basic procedures have been tried over the intervening years to overcome technical difficulties and to reduce the mortality rate and the incidence of recurrent ulceration.

Heineke and Mikulicz performed pyloroplasty for benign pyloric stricture in 1886[3].

Hofmeister in 1888, modified the Billroth II procedure, closing the upper portion of the cut end of the stomach using only the lower portion in establishing continuity with the jejunum following partial gastrectomy.

Braun and Jaboulay in 1892, and later Roux in 1898, devised procedures, to overcome bile regurgitation and afferent loop distention. Both operations, however, drained the alkaline duodenal secretion away from the anastomosis thus predisposing to jejunal ulcer.

When it became apparent that food left the stomach both via the enterostomy and the pylorus after gastroenterostomy, procedures were designed to ensure protection of the duodenum by excluding it. Von Eiselsberg in 1895 divided the stomach through the antrum, closed both ends and performed a gastroenterostomy. Devine's pyloric exclusion is very similar[5]. No stomach is resected in either operation. Both procedures resulted in high recurrence rates, probably due to hormonal stimulation of gastric secretion from the isolated antrum[5].

Schmilinski in 1918 suggested total intragastric regurgitation of the alkaline duodenal secretions after antral resection. Although this procedure is appealing, it produces ulcers rather than protects against recurrence. The high recurrence rate is probably due to interminable stimulation of the gastric phase of gastric secretion[4]. Previous procedures are illustrated in Figures I, II, and III.

Partial gastric resection as the only acceptable operation for the peptic ulcer diathesis was championed in Europe by Finsterer[6] and von Haberer[7] and in this country by Berg[8] and Lewisohn[9]. These surgeons were searching for an operation which would rid the patient of pain, hemorrhage and obstruction and provide assurance against recurrence. The three-quarter distal gastric resection meets these criteria.

In the past five years increasing emphasis has been placed on evaluating the side effects of gastrectomy which, while minor in most cases, have deterred certification of the procedure as ideal. With this in mind Wangensteen, in 1949, modified segmental resection, an operation designed to remove a maximal protion of

Figure I

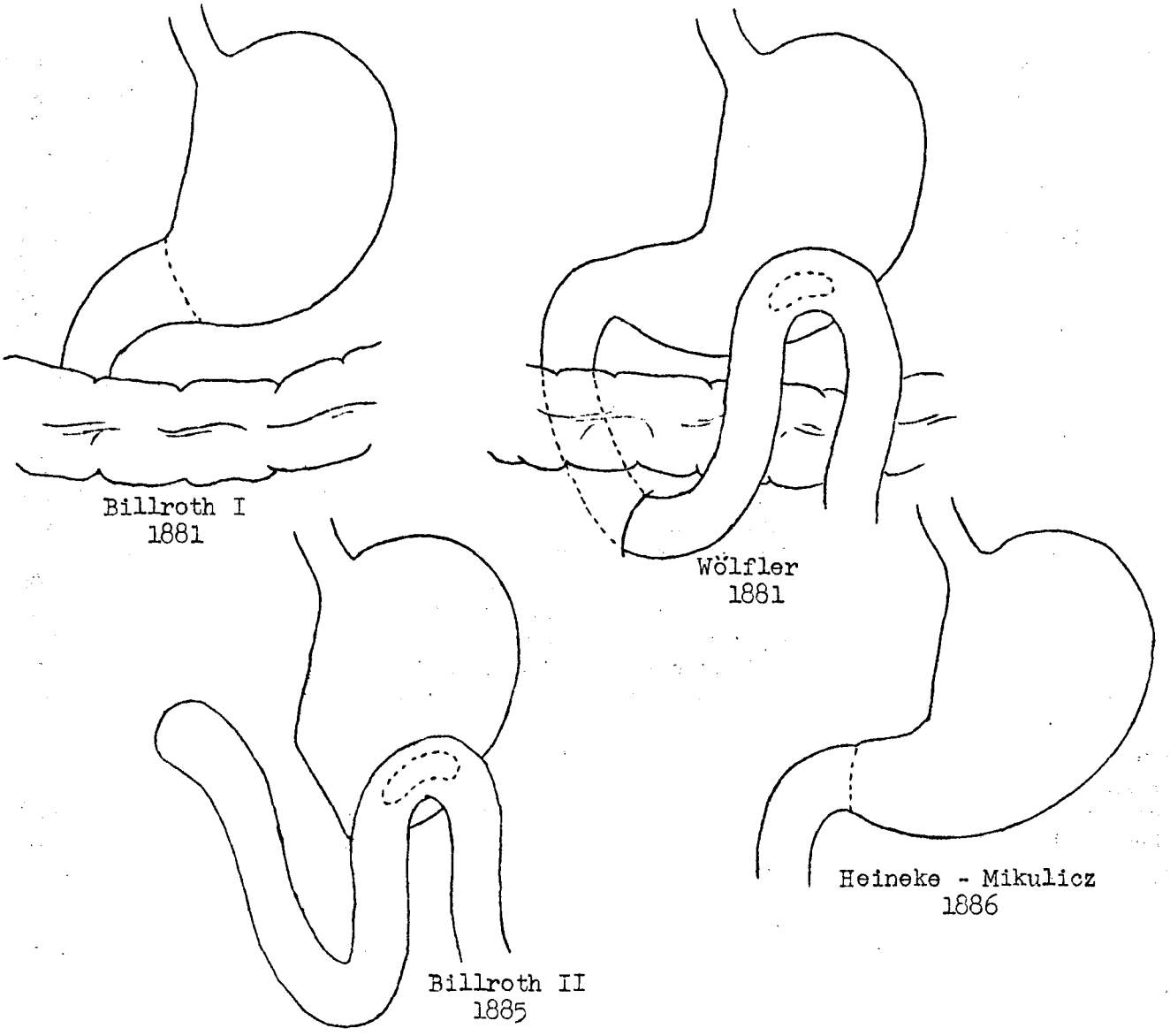

Billroth I
1881
Wölfler
1881
Billroth II
1885
Heineke - Mikulicz
1886

Figure II

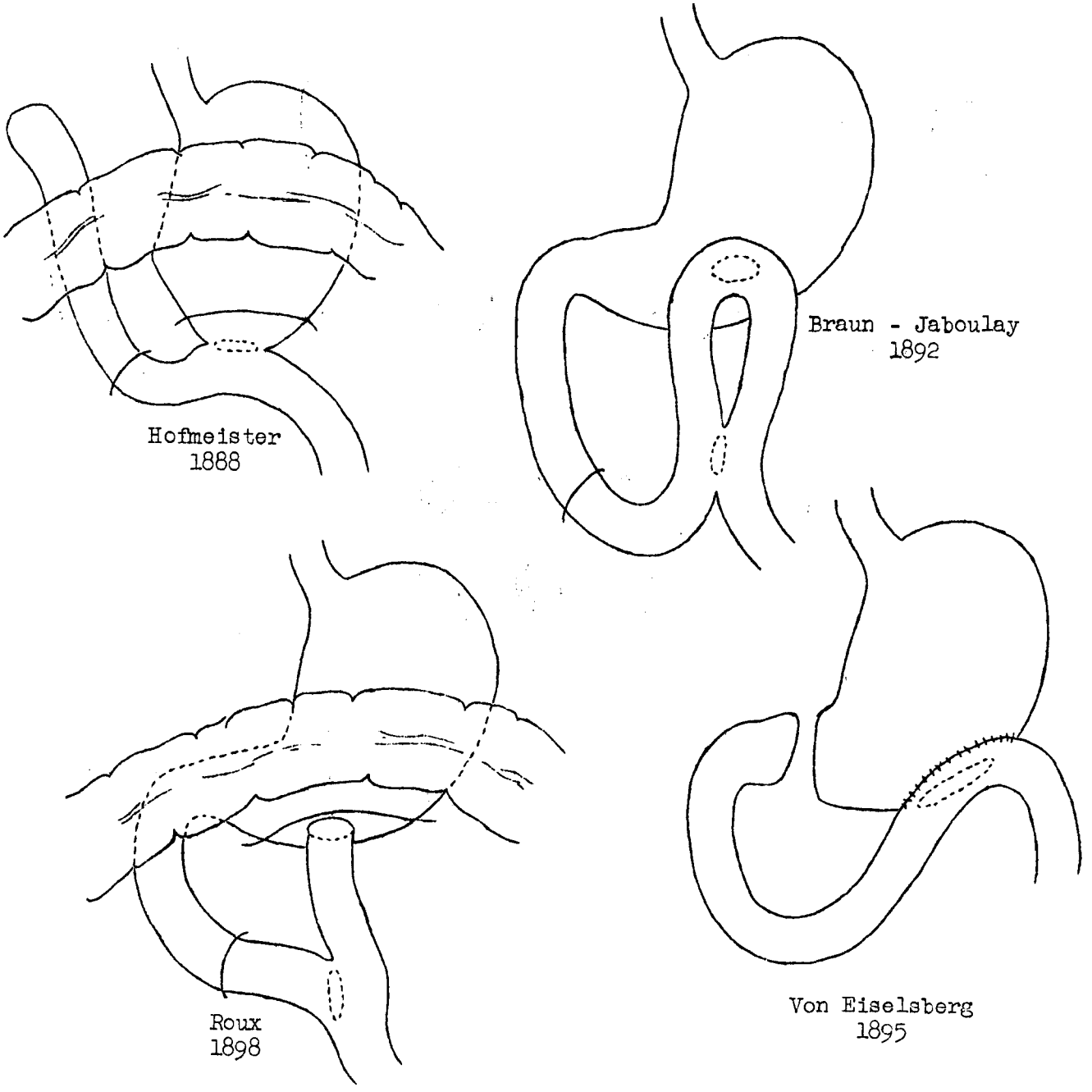
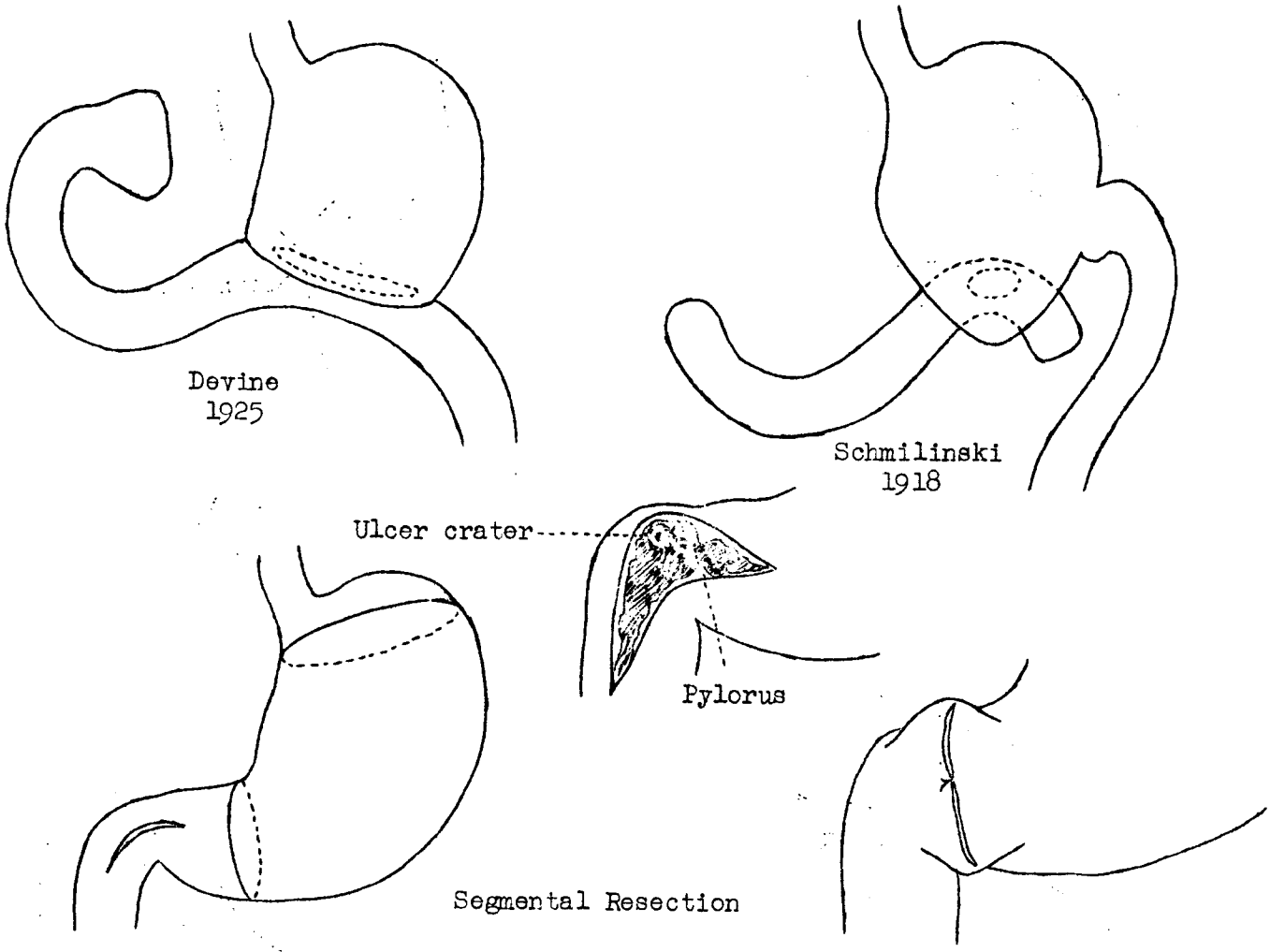

Hofmeister
1888
Braun - Jaboulay
1892
Roux
1898
Von Eiselsberg
1895

Figure III


Devine
1925
Schmilinski
1918
Ulcer crater
Pylorus
Segmental Resection

the acid-secreting area of stomach, to alleviate or completely rid the patient of the undesirable sequelae of gastrectomy[10]. (Figure III) During 1952 Wangensteen has performed tubular gastric resection designed to leave the vagus nerve intact and reconstitute a smaller stomach by means of transverse gastroplasty.

Connell in 1929[54] suggested fundusectomy as a likely method to reduce acid production by the stomach. He excised a pie-shaped segment from corpus and fundus. The resection was however small, probably not over 40 per cent of the stomach. Between 1938 and 1940 Wangensteen performed a somewhat larger excision of the acid-producing area of the stomach by excising the greater curvature[55].

Tubular gastric resection, as performed at this time, removes a larger area of the acid-secreting mucosa of the stomach and experimentally protects the dog against histamine-induced ulcer[13].

The operation is performed through an extra-pleural sternotomy incision which extends out into the fourth left intercostal space. Exposure of the greater curvature to its uppermost limit is facilitated by this incision. Rubber-shod clamps are placed on the upper and lower ends of the stomach and a large segment of the acid-producing area excised. The stomach is reconstituted by transverse gastroplasty which is performed in an open fashion in two layers. The lesser curvature containing the vagus nerves becomes longer than the greater curvature after completion of the procedure. A complementary pyloroplasty is necessary only in those patients who have organic pyloric obstruction. The technic of tubular resection is illustrated in Figure IV.

The technical hazards of the difficult duodenum can be avoided by this procedure. Supraduodenal ulceration or large, indurated penetrating duodenal ulcers makes closure as in the Billroth II procedure hazardous and anastomosis as in the Billroth I procedure impossible.

Those who would criticize this principal of gastric resection state that recurrent ulcer is imminent because the gastrin-producing antrum is not removed[51]. Recurrence rates are excessively high after the von Eiselsberg procedure, the Devine operation and the Finsterer exclusion procedure. These operations all exclude the antrum and do not leave it in direct continuity with the stomach. The gastric antrum transplanted onto the colon in a dog causes an excessive production of acid by the stomach[52]. The fact remains there have been no recurrent ulcers after segmental gastric resection. It is therefore felt that if a substantial portion of the acid-secreting area of the stomach is excised, that retention of the antrum serves only the beneficial effect of increasing gastric capacity. It is separation of the antrum from the body of the stomach that invites ulcer[53].

The operative procedure removes a smaller total quantity of stomach but provides the same protection, an important consideration from the standpoint of unpleasant postoperative sequelae.

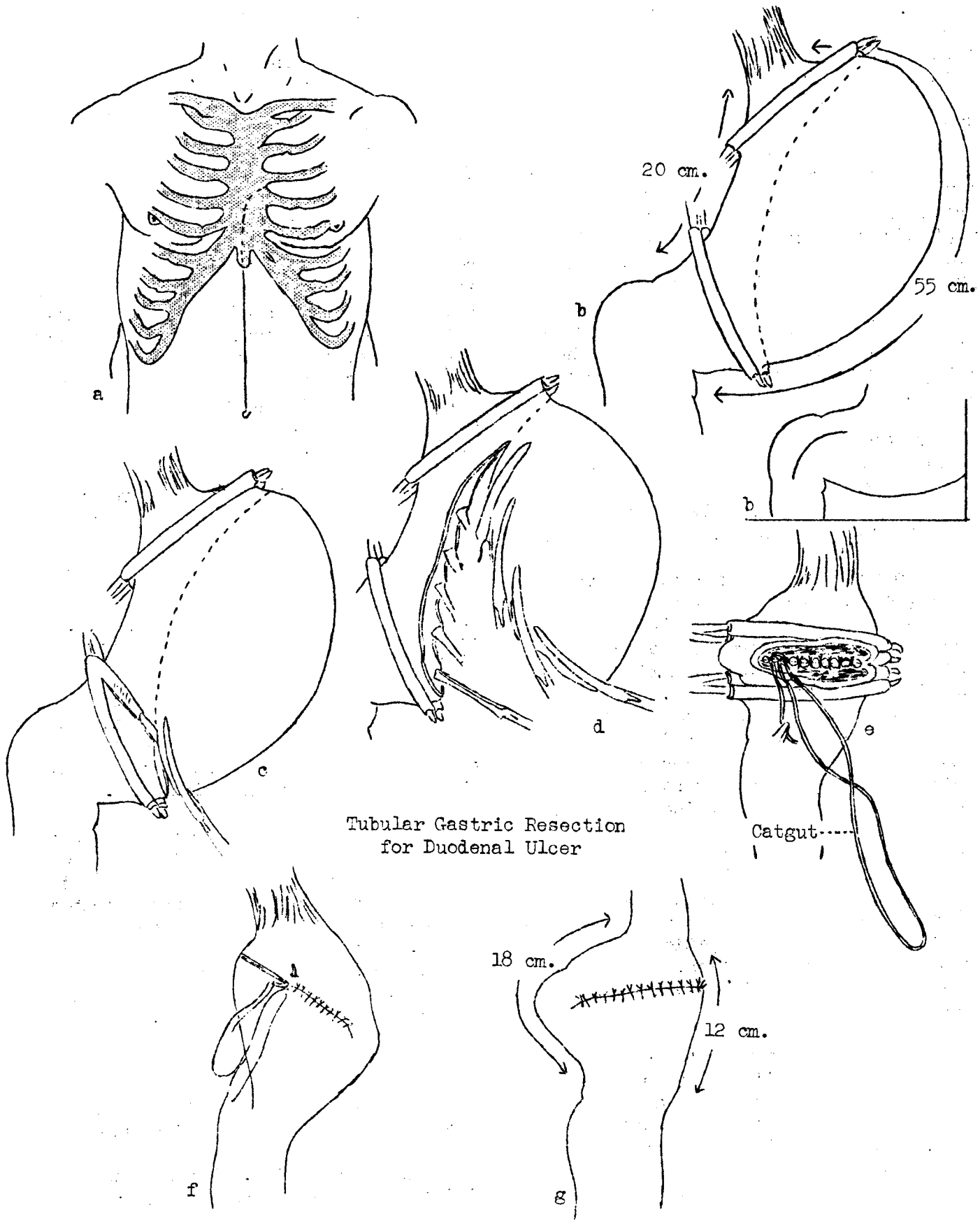
Patients Subjected to Tubular Gastric Resection:

Thirty-seven of the patients who underwent tubular gastrectomy were males, 14 were females. The average age of the group was 52.4 years. The youngest patient was 21 years, the oldest 85.

The indications for surgery were predominantly the conventional three -- pain, hemorrhage and obstruction as outlined in Table I. One patient only was operated upon as an emergency while bleeding profusely. Previous surgery had been performed for peptic ulcer in nine of the patients. Perforated peptic ulcers had been closed in seven patients. One patient had undergone gastroenterostomy and one vagotomy.

Twenty-six of the 51 patients had received supervised therapy with Banthine which was either ineffective or effective for only a short time. Banthine was of limited value as a substitute for surgery

Figure IV



Tubular Gastric Resection
for Duodenal Ulcer

TABLE I

INDICATIONS FOR SURGERY

Intractable pain	14
Pain and Hemorrhage	11
Hemorrhage	8
Pain and Obstruction	5
Hemorrhage and Obstruction	4
Pain, Hemorrhage and Obstruction	3
Esophagitis and Esophageal Stricture	3
Obstruction	2
Possible malignancy	1

in those patients who developed a relapse soon after discontinuing the drug or while still on it. All patients had received medical treatment prior to surgery.

The average duration of symptoms for the entire group of patients was 13.7 years which is a reflection of the chronicity and recurrent nature of duodenal ulceration.

The lesions encountered at surgery are illustrated in Table II.

TABLE II

LESIONS ENCOUNTERED AT SURGERY

Duodenal Ulcer	41
Gastric Ulcer	3
Esophagitis and Esophageal Stricture	3
Gastric and Duodenal Ulcer	2
Stomal Ulcer	1
Post-vagotomy obstruction	1

This procedure has a wide field of usefulness but is probably of greatest value in the treatment of duodenal ulcer. The patient with post-vagotomy obstruction had elevated free gastric acid on histamine stimulation. A partial resection was therefore performed in addition to pyloroplasty. Both patients with combined gastric and duodenal ulcers had symptoms for many years and previously demonstrated duodenal ulcers. Partial pyloric obstruction developed and subsequently gastric ulcers occurred in both. This is not an uncommon occurrence.

Weights of Resected Specimens:

For the past twelve years at this hospital all resected stomachs have been weighed, in an attempt to establish, in a more accurate manner than the surgeon's estimate, the extent of resection. This measurement shows least variation when the stomach has been resected for non-obstructing duodenal ulcer. Peptic ulcer of the stomach and pyloric obstruction increase the weight of the specimen and variation in the weight is greater.

A comparison of specimen weights after Billroth II, segmental and tubular resections is illustrated in Table III.

The amount of stomach removed utilizing tubular gastric resection is significantly less than with the other procedures. This fact may be an advantage when we consider postoperative results.

Kelly[13] et al have shown this procedure to be adequate in protecting dogs

TABLE III

WEIGHT OF RESECTED SPECIMENS

	Duodenal Ulcer	Non-obstructed	Obstructed
1.	Billroth II - 75% resection	176 Gm.	276 Gm.
2.	Segmental Resection	163 Gm.	224 Gm.
3.	Tubular Resection	137 Gm.	210 Gm.
	Gastric Ulcer		
1.	Billroth II - 75% resection	183 Gm.	289 Gm.
2.	Segmental Resection	153 Gm.	283 Gm.

against histamine-induced ulcers, which in the past has been an excellent criterion of the adequacy of an operation for peptic ulcer.

Acid Values:

The preoperative and postoperative gastric acid values are available in the great majority of patients. A triple histamine test is routinely done. The average preoperative free acid value for patients with duodenal ulcer was 83°. The preoperative free gastric acid values of 90 patients who underwent segmental resection for duodenal ulcer was 88°. (Table IV).

TABLE IV

FREE GASTRIC ACID BEFORE AND AFTER OPERATION

	Segmental	Tubular
Before Operation	88°	83°
After Operation	1°	13°
Highest Postoperative free acid	30°	49°

The free gastric acid in patients with gastric ulcer is 50°.

All patients but 5 after segmental gastric resection were achlorhydric as might be expected following extensive resection of the acid-producing area of the stomach. Over one-half of the tubular resection patients have free acid after operation.

Postoperative X-rays:

A gastro-intestinal series is obtained within three months following surgery. To date, all duodenal ulcers have healed at this interval. Although there is immediate passage of barium into the duodenum, the stomach is not usually empty before 30 minutes. There is no essential difference in gastric emptying time as measured by this method in the 12 patients who did not have pyloroplasty. The gastric emptying time is slower than usually seen after the Billroth II type gastric resection.

Mortality:

There have been two deaths to date after a total of 60 resections. The mortality rate therefore is 3.3 per cent. The first patient died on the eighth postoperative day of liver failure. At postmortem examination, thrombosis of the portal vein and necrosis of the right lobe of the liver was found. Thrombosis in this patient was probably the result of simultaneous splenectomy. Routine platelet counts are now done after splenectomy even in the absence of hematological disorder. The second death occurred in an 85-year old man admitted with a bleeding duodenal ulcer. The bleeding did not stop but became more severe after 24 hours hospitalization. Surgery was advised but refused. At the end of 48 hours in the hospital, the patient had received 5000 cc. of blood without complete replacement. A tubular resection was done at that time and the bleeding halted utilizing a pylorotomy to find the point of hemorrhage. He received an additional 5000 cc. of blood on the day of surgery. The patient died on the tenth postoperative day with cerebral thrombosis.

Postoperative Evaluation and Discussion:

The postoperative evaluation will be considered from the following standpoints: early postprandial distress, hypoglycemic symptoms, food intolerance, maintenance of nutrition and weight change, recurrent ulceration, maintenance of occupation and degree of endurance, anemia and the patient's opinion regarding the benefits obtained.

(I) EARLY POSTPRANDIAL DISTRESS SYNDROME

Discomfort after eating in a patient who had undergone gastric surgery was first described by Denechaux[14] in 1907. In 1913, Hertz[15] described a similar syndrome in patients who had undergone gastroenterostomy for duodenal ulcer, and attributed the difficulty to too rapid gastric evacuation. Mix[16] in 1922, first affixed the title "dumping" to the syndrome after noting rapid gastric emptying on roentgen examination in a patient who had a gastroenterostomy. The

symptoms disappeared after normal bowel continuity was re-established.

We have defined early postprandial distress or the dumping syndrome as one or more of the following symptoms coming on near the conclusion of a meal or shortly thereafter: profound weakness, generalized feeling of warmth, sudden appearance of perspiration, either of a localized area such as the forehead or generalized over the entire body, drowsiness, dizziness, palpitation, nausea and perhaps crampy pain with subsequent passage of a watery stool.

Early postprandial distress occurs after total gastrectomy, subtotal and partial gastrectomy, gastroenterostomy and following vagotomy alone[17,18].

In an effort to establish whether the syndrome occurs after any other operative procedure, sixty patients were questioned who had undergone the following operations: appendectomy, cholecystectomy, partial and total colectomy, combined abdominoperineal resection, radical and super-radical mastectomy and extensive small bowel resection. Two patients of the entire group had symptoms suggesting mild postprandial distress, both had undergone cholecystectomy.

The incidence of the syndrome after partial gastrectomy has been quoted at from 2 to 75 per cent[19,20,21,50]. The discrepancy lies in the difference in definition of the syndrome and the care used in follow-up appraisal.

All patients who have postprandial distress emphasize the fact that it is the size of the meal that determines the occurrence of difficulty. Almost all patients are relieved by lying down. Sweet foods, milk and ice cream are more likely to precipitate the syndrome than any other foods. The symptoms are most severe immediately following surgery and ameliorate steadily with the passage of time even though ingestion of food usually increases.

The etiology of early postprandial distress is unknown. The most prominent theories are as follows:

1. Hyperglycemic Shock - was proposed by Glaessner[22] in 1940 based on the rapid rise of blood sugar to hyperglycemic levels known to follow carbohydrate ingestion in gastrectomized patients. Experimentally, however, the symptoms usually occur before a significant rise in blood glucose had developed and intravenously induced hyperglycemia does not reproduce the syndrome[23].

2. Shock - was suggested by Perman[24] since the signs and symptoms of the dumping syndrome do at times suggest a shock-like state. There is, however, no drop but usually a slight elevation in blood pressure during the distress[21].

3. Gastritis was considered the cause by Schindler[25] who observed these changes in the gastric stump on gastroscopy. Hebbel[26] however, has shown gastritis to be an almost constant accompaniment of duodenal ulcer, the changes being present microscopically at the time of surgery. The syndrome also occurs after total gastrectomy.

4. Hypoglycemia was proposed by Gilbert and Dunlop[27] mainly on the symptomatology. This explanation does not seem possible since the symptoms occur within 10 minutes of the meal and hypoglycemia does not occur until one to three hours later.

5. Jejunal Distention - as a result of rapid gastric emptying was first suggested by Hertz[2]. Adlersberg and Hammerschlag[28] agree but believe the disseminated symptoms, which must be mediated through a nervous or hormonal mechanism, are the result of vagus stimulation. The syndrome however, has been reported when vagotomy alone has been performed for peptic ulcer[17,18].

Irving[29] also agrees that jejunal distention is the cause but thinks the syndrome is mediated through the sympathetic nervous system. We are at present assessing the value of hexamethonium bromide, a ganglion blocking agent in the therapy of the dumping syndrome.

Machella[30] suggests that jejunal distention is the result of ingestion of hy-

pertonic, especially glucose, solutions which are diluted in the jejunum. This undoubtedly occurs since we have reduplicated the syndrome exactly, in individuals given oral glucose for a tolerance test, however, it is not the only mechanism. We have precipitated the syndrome in patients given milk only, preceded for 12 hours by no other food.

6. Gastric stretch - is thought to be the cause by Capper and Butler[31] who did not encounter the postprandial distress syndrome in patients who had undergone total gastrectomy, therefore discrediting jejunal distention as a cause. They were able, in addition, to produce dumping by placing a mercury-weighted bag in the gastric pouch with the patient in the erect position. We have carefully interviewed 12 patients who have had total gastrectomy performed over the last two-year period and find early postprandial distress definitely present, however in a mild form and almost entirely confined to the "bowel symptoms" of nausea, abdominal cramps and diarrhea. "Vasomotor symptoms", palpitation, sweating, dizziness and weakness were present in only one patient of the twelve after total gastrectomy. This will later be referred to in Table V.

7. Temporary Hypokalemia has been suggested by Hamilton Smith[18] on the thesis that unprepared hypertonic carbohydrate food repidly enters the jejunum, is rapidly absorbed as glucose and converted to glycogen with resultant utilization of a more than usual amount of extracellular potassium and production of electrocardiographic changes consistent with hypokalemia. We investigated six patients and were able to produce typical postprandial distress in four patients. The patients were given a meal, milk or 100 Gm. of glucose solution to produce the dumping syndrome. Electrocardiographic changes consistent with hypokalemia were produced in some patients. Serum potassium values were obtained before the meal and at the height of distress. As illustrated in Table V there was no consistent change in serum potassium. The electrocardiograms of these patients were seen by Doctor Ernst Simonson, of the Department of Physiological Hygiene, who noted non-specific abnormalities, especially of the T waves and ST segments. The electrocardio-

TABLE V

SERUM POTASSIUM AND E.C.G. CHANGES DURING DUMPING SYNDROME

NAME	MEAL	REACTION	SERUM POTASSIUM BEFORE	SERUM POTASSIUM AFTER	E.C.G.
H.M.	Breakfast	Sweating Weakness	4.8 MEG	4.4 MEG	Abnormal
A.K.	Milk	Palpitation Weakness	4.8 "	4.8 "	Abnormal
I.N.	Milk	Palpitation Weakness	5.0 "	5.3 "	Tachycardia
D.M	Milk	Palpitation Weakness Sweating	4.4 "	5.1 "	Tachycardia
J.F.	Dinner	No reaction	4.6 "	4.5 "	Abnormal
M.W.	Glucose	No reaction	-	-	Tachycardia

graphic changes were not consistently typical of hypokalemia, and in absence of supporting data, could not be considered due to abnormality of the serum potassium. The changes in serum potassium noted by Smith were one to two hours after the meal when postprandial distress symptoms have disappeared. We believe that transient hypokalemia is not the explanation of early postprandial distress after gastrectomy.

Although we are better prepared to say what does not cause the dumping syndrome than what causes it, certain facts stand out:

1. The bulk or size of a meal is the single most important precipitating factor, which suggests a mechanical cause. This outlook is supported by the fact that very cold or very hot foods precipitate postprandial distress in some patients.

2. Symptoms definitely decrease in severity or disappear over the first few months after operation suggesting an adaptation on the part of the jejunum or duodenum to the new conditions.

3. The increased tolerance of patients to the vasomotor symptoms of postprandial distress after total gastrectomy may be related to the achlorhydria present prior to operation.

4. The symptoms are almost always relieved in a short time by lying down.

5. Fewer patients are troubled after tubular resection with diarrhea which may be attributable to the continuity of the vagus nerve. Diarrhea was quite common after segmental resection where the vagi are divided high on the lesser curvature of the stomach and it is a well known complication of vagotomy for peptic ulcer. Diarrhea occurs after Billroth I and Billroth II resections also.

A comparison of severity and frequency of early postprandial distress is illustrated in Table VI.

It must be borne in mind that all tubular gastric resections were performed over the last 12-month period and will undoubtedly improve. There were no patients with incapacitating dumping after segmental or tubular gastric resection. All patients were asked if they considered their difficulty severe enough to take medication for. Most patients classified as B did not think medicine was required. Many others in Class B could avoid the symptoms entirely by eating more often and eating less

TABLE VI

FREQUENCY AND SEVERITY OF EARLY POSTPRANDIAL DISTRESS

	Billroth II (%)	Segmental (%)	Tubular (%)	Total (%)
A-Asymptomatic	54	49	53	50
B-Mild symptoms five times weekly or with overindulgence in food	26	37	43	50
C-Moderately severe symptoms one-three times daily	17	14	4	0
D-Symptoms once daily of an incapacitating nature	3	0	0	0

at each meal.

(II) HYPOGLYCEMIC SYMPTOMS

The hypoglycemic symptoms which occur after gastrectomy are dizziness, weakness, sweating, palpitation and inability to accommodate which occur one to three hours after eating. Troubled individuals often volunteer that, eating or drinking something sweet relieves them immediately. These individuals exhibit a characteristic glucose tolerance curve, the lag phase curve. There is a rapid rise of blood glucose to hyperglycemic levels, accompanied by glycosuria, followed by a rapid fall of blood glucose in one to three hours to hypoglycemic levels. The symptoms occur during the hypoglycemic phase.

The hyperglycemia is due to rapid absorption of glucose from the small bowel after rapid gastric emptying. The mechanism of the subsequent hypoglycemia is not understood. It may be due to an excessive production of insulin or to a sensitivity to insulin. The hypoglycemia does not occur when the glucose is given intravenously.

Hypoglycemia symptoms occurred in 19 per cent of the Billroth II patients but in less than five per cent of patients who have undergone segmental and tubular gastric resection. The difference, we believe is due to slower gastric emptying in the latter two procedures and therefore more gradual intestinal absorption.

(III) FOOD INTOLERANCE

All patients were asked if there were any foods they could not eat since the operation. Most patients were very pleased with the variety of food they could eat; however, one-third of the patients no longer enjoyed milk or ice cream. A dislike for sweets was expressed by an additional smaller group. Milk, ice cream and sweets occasionally precipitate the dumping syndrome or make the patient feel uncomfortably full.

(IV) DEGREE OF MAINTENANCE OF NORMAL NUTRITION AND WEIGHT CHANGE

A major concern of patients after gastrectomy is inability to maintain or gain weight. This is more pronounced during the first postoperative year and will be accentuated in the tubular gastric resection patients. As a basis for normal, the average weight over the five years preceding surgery was used thus eliminating abnormal changes due to obstruction or diet immediately before operation. On this basis 80 per cent of the patients lost weight; however, only 25 per cent were below their ideal weight for age and body build. Many of those who were underweight at this time were also underweight five years prior to the operation. The operation per se can be indicted for an underweight or malnourished state in very few patients.

Why do these patients lose weight? There are two plausible explanations: (1) a decreased intake, (2) increased loss of calories especially in the form of fat in the stool.

Increased fat loss after partial gastrectomy has been emphasized by Wollaeger [33,37,36], Gordon-Taylor[34] and others[35]. In an attempt to evaluate this after tubular gastric resection, five patients were hospitalized for one week at a period three to six months after operation. These patients were placed on a weighed diet consisting of 80 grams of fat, 80 grams of protein, and 145 grams of carbohydrate daily. The dietary intake was charted as for a diabetic so the true intake was determined. Stools were marked with carmine. A three day stool was collected and total fat and nitrogen calculated. Fat loss in the stool was expressed in per cent of intake and nitrogen loss in grams per day. (Table VII)

The normal fat excretion on an 80 gram of fat per day diet is five per cent of intake, never over ten per cent. The fat excretion in all cases is within normal limits. One patient (L.L.) is excreting a slightly elevated amount of nitrogen.

Four of five of these patients have lost an excessive amount of weight; one is underweight. Fat loss in the stool is not a causal factor; however, decreased food intake is closely related to weight

TABLE VII

POSTGASTRECTOMY NUTRITION

NAME	WEIGHT LOSS	FAT LOSS PER DAY	PER CENT OF INTAKE	NITROGEN LOSS PER DAY	PER CENT NORMAL FOOD INTAKE PRE- OP. INTAKE = 100 PER CENT
W.H.	20	3.4 GM.	4.0	1.3 GM.	60-75
L.L.	30	5.6 GM.	6.0	2.6 GM.	75
Y.R.	10	3.4 GM.	4.3	1.3 GM.	90
G.CE.	30	4.0 GM.	5.0	0.5 GM.	50-60
G.C.	25	3.6 GM.	4.5	0.4 GM.	50-60

loss and would seem to be the factor of importance. Every patient who underwent tubular resection and weighed the same as he did five years ago was eating 100 per cent of what he did formerly. The average weight loss after segmental resection was 18 pounds, after Billroth II - 15.3 pounds and after tabular resection 16 pounds.

(V) RECURRENT ULCERATION

One patient gives a history of dark stools on one occasion since tubular gastric resection. There was no accompanying anemia and no stomal ulcer demonstrated on x-ray; however, this patient must be viewed as possible recurrence. The follow-up period is too short to give the final result of this operation in regard to recurrence. The recurrence rate after Billroth II resection is 1.2 per cent. There have been no recurrences after segmental resection.

(VI) MAINTENANCE OF OCCUPATION

The average time from operative procedure to return to occupation was 7.5 weeks. The patients who had segmental resection returned to work in ten weeks, and the Billroth II resection patients operated upon in 1949 returned to work in 13 weeks. Many gastrectomy patients notice a decrease in stamina for at least a year following operation. This was not as common after other operations of equal magnitude e.g., colectomy.

(VII) ANEMIA

Anemia after gastrectomy is an exceedingly interesting subject which provides, a not fully utilized source of investigation into the physiology of iron and extrinsic factor absorption.

Seventeen per cent of the patients who underwent Billroth II resection developed an iron deficiency anemia. The anemia is more common in females and usually responds to oral iron over a long period of time but occasionally parenteral iron is required.

Iron deficiency has not occurred to date after segmental or tubular gastric resection. If freedom from anemia persists in these patients it may be attributable to slower gastric emptying time and/or gastroduodenal continuity since most ingested iron is absorbed from the upper gastrointestinal tract, especially the duodenum[39].

Latest reports discount the necessity of acid for iron absorption[38].

Table VIII illustrates the incidence of anemia after partial gastric resection. This review of the literature would suggest that by-passing the duodenum predisposes to iron deficiency anemia, however clinical reports of the Billroth I procedure in the literature with follow-up blood examination are exceedingly few. Further evidence, however, that gastro-

TABLE VIII

IRON-DEFICIENCY ANEMIA AFTER GASTRECTOMY

Author & Date	Type of Operation	Number of Pts.	Criterion of Anemia	Per Cent Anemia Men	Women	Both
Gordon-Taylor, 1929	Polya	52				44
Morley and Roberts 1928	Polya Schoemaker	21 68	70%			57 0
Lublin 1936	Billroth II Gastro-enterostomy	46	Males 80% Females 70%			69
Dedichen 1934	Billroth II	254	80%	27	80	
Drablos 1951	Billroth II	492	Males 13 GMS Females 11.5 GMS	26.6	41.2	
Hartfall 1934	Polya	120	85%	16	50	
Holst-Larson, 1934	Polya Gastro-enterostomy	86 65	Males 90% Females 80%	27 25	49 35	
Rauch 1952	Billroth II	893	11 GMS			17
Bohmanson 1950	Billroth I	1200				0
Johnsson 1951	Billroth II	80	Males 80% Females 70%	33	33	
Ramberg 1948	Polya	667				20
Lyngar 1950	Polya Billroth II	146	Males 87% Females 80%	15	43	

duodenal continuity prevents postgastrectomy iron deficiency anemia is cited by Kelly[40]. He has gastrectomized two groups of rats -- one by the Billroth I method, the other by the Billroth II procedure. The incidence of anemia is much higher in the latter group.

(VIII) PATIENTS' OPINION OF OPERATION

All patients who underwent tubular gastric resection felt they were helped by the operation. Some were quite outspoken about their particular benefits. Some of these patients, of course, experienced decreased endurance, postprandial distress and weight loss but considered them subordinate to the relief obtained from the ulcer pain and complications.

To arrive at an overall evaluation, all the above factors were taken into consideration and the criteria listed in Table IX were used to classify the patients as excellent, satisfactory or poor.

TABLE IX

BASIS FOR OVERALL EVALUATION

Excellent

1. No early postprandial distress
2. Loss of less than 12 pounds
3. Patients affirmative statement of success.
4. Absence of recurrent ulceration.
5. Ability to ingest as much as before.
6. Maintenance of full-time occupation.

Poor

1. Recurrence of ulceration.
2. Marked weight loss, improper nutrition.
3. Frequent and incapacitating dumping.
4. Inability to maintain occupation.
5. Patient's statement that operation has failed.

Satisfactory - Lies between these extremes.

RESULTS OF OVERALL EVALUATION

	Billroth II	Segmental	Tubular
Excellent	26%	25%	35%
Satisfactory	63%	66%	61%
Poor	11%	9%	4%

The Billroth II resections represent a stable group who will not change appreciably at this time.

Improvement can be expected in both the segmental and tubular resection patients. The two patients classified as poor results after tubular gastric resection both consider the operation successful. One patient has the suspected recurrence previously mentioned, the other patient has lost an excessive amount of weight. Many patients are excluded from the excellent classification because they fail to satisfy one only of the criteria.

DISCUSSION AND CONCLUSIONS

The preliminary evaluation of tubular gastric resection, based on observations in 51 patients, is presented.

The operation designed to remove a major portion of the acid-producing area of the stomach, is performed utilizing a sternotomy incision and gastric continuity is re-established by transverse gastroplasty. Complementary pyloroplasty is necessary only when organic pyloric obstruction exists. This confirms the fact that vagus nerve continuity is preserved since it is well recognized that pyloroplasty or enterostomy is necessary after vagotomy to ensure gastric emptying.

Tubular resection has been used primarily for ducdenal ulcer since by this method the difficulties of closure or anastomosis in an area of ulceration can be avoided. The operation also has been used successfully in the treatment of esophagitis and lower esophageal stricture.

A smaller segment of stomach is removed than is usual after Billroth II or segmental gastric resection which may account for lower incidence and milder variety of postprandial distress.

Diarrhea is less frequent after tubular resection which may be attributable to the fact that the vagus nerve is not interrupted.

Recurrent ulceration is a possibility in one patient. The final evaluation in this regard cannot be made at this time.

Free hydrochloric acid is present in 50 per cent of patients after tubular resection and averages 13°.

Hypochromic, iron-deficiency anemia has not occurred. Gastroduodenal continuity is probably a factor of importance in iron absorption and the prevention of postgastrectomy anemia.

The incidence, etiology and clinical features of the dumping syndrome have been reviewed. It is suggested that its origin is mechanical due to irritation, distention or hyperperistalsis of the upper small bowel.

Hypoglycemia is an unimportant cause of postcibal distress if gastric emptying is not too rapid.

No abnormal fat excretion was found in five patients studied by intake-output balance methods. These patients had lost an excessive amount of weight which was closely related to decreased oral intake. On the whole, weight loss was not excessive, especially when one considers all patients are under one year postoperative. Eighty per cent of the patients lost weight, only 25 per cent were under weight and over one-half of the latter were under weight five years prior to operation. The operation could be indicted for an underweight condition in very few patients.

Almost all patients were agreed that health had been immeasurably improved. The operation has been successful in removing a patent source of misery and a danger to life.

Tubular gastric resection on overall evaluation at this early date compares very favorably with operations performed in this hospital for the peptic ulcer diathesis over the last 12-year period.

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II. MEDICAL SCHOOL NEWS

Coming Events

January 8-10 Continuation Course in Anesthesiology for General Physicians
January 15 Continuation Course in Management of the Polio Patient
January 19-23 Continuation Course in Ophthalmology for Specialists
January 26-31 Continuation Course in Pediatric Neurology for Pediatricians, Neurologists, and General Physicians
February 2-4 Continuation Course in Clinical Chemistry for General Physicians
February 5-7 Continuation Course in Cancer Detection for General Physicians
February 12-14 Continuation Course in Cardiovascular Diseases for General Physicians
February 16-18 Continuation Course in Recent Advances in Diagnosis for Internists
February 17 Phi Delta Epsilon Lecture; "Iron Metabolism and Iron Deficiency Anemia"; Carl V. Moore, Professor, Department of Medicine, Washington University School of Medicine, St. Louis; Owre Amphitheater; 8:00 p.m.

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Continuation Course

The University of Minnesota will present a continuation course in Ophthalmology for Specialists at the Center for Continuation Study from January 19 to 23, 1953. Emphasis will be placed throughout the session on therapy of a variety of ocular disorders. The visiting faculty for the course will include Dr. Kenneth C. Swan, Professor and Head, Department of Ophthalmology, University of Oregon Medical School, Portland; and Dr. Harold G. Scheie, Associate Professor, Department of Ophthalmology, University of Pennsylvania Medical School, Philadelphia. The course will be presented under the direction of Dr. Erling W. Hansen, Professor and Director, Division of Ophthalmology, and the remainder of the faculty will include members of the staff of the University of Minnesota Medical School and the Mayo Foundation.

Senior Student Receives Mariette Award

James Jude, a member of the Senior Class, was recently presented with the E. S. Mariette Award by the Hennepin County Tuberculosis Association. This award, which carries with it a $500 scholarship, is awarded annually to a student of outstanding ability who has manifested an interest in chest diseases. The faculty joins in extending congratulations to Mr. Jude and our best wishes for future success.

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Faculty News

Dr. Wesley W. Spink, Professor, Department of Medicine, was selected by the Editorial Board of Modern Medicine as one of ten physicians who made outstanding contributions to medical progress during 1952. Announcement of his selection was recently made by Dr. Walter Alvarez, Editor of the publication. Dr. Spink will receive a scroll honoring him for his research work in brucellosis.

A featured speaker at the recent meeting of the American Association for the Advancement of Science in St. Louis, Missouri, was Dr. Maurice B. Visscher, Professor and Head, Department of Physiology. On December 28 Dr. Visscher discussed "The Importance of Public Education in Physiology" and on December 30 he spoke on "The Proper Role of Scientists in a Schizophrenic World."

Six members of the faculty of the Medical School took part in the scientific program of the meeting of the Hennepin County Medical Society on Monday, January 5. Joining in the discussion of "Practical Aspects of Cardiac Surgery" were Dr. Richard V. Ebert, Professor, Department of Medicine, Dr. James Dawson, Professor and Head, Department of Pathology, and Doctors Richard L. Varco, Ivan D. Baronofsky, F. John Lewis, and C. Walton Lillehei of the Department of Surgery.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

January 12 - 17, 1953

Monday, January 12

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:30 - Tumor Conference; Doctors Kremen, Moore, and Stenstrom; Todd Amphitheater, U. H.

11:30 - 12:30 Physical Medicine Staff Seminar; Heart Hospital Auditorium.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - 1:30 Physiology Seminar; Metabolism Butyrate Mammalian Myocardium; Victor Lorber; 214 Millard Hall.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Pediatric Seminar; Thrombocytopenia Associated with Hemangioma; Thomas Good; Sixth Floor West, U. H.

4:00 - 5:30 Seminar on Fluid and Electrolyte Balance; Gerald T. Evans; Todd Amphitheater, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

4:30 - Public Health Seminar; 15 Owre Hall.

4:30 - 6:00 Physiology 114A and Cancer Biology 140 -- Research Conference on Cancer, Nutrition, and Endocrinology; Drs. Visscher, Bittner, and King; 129 Millard Hall.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Eldon Berglund; Newborn Nursery, Station C.

10:30 - 12:00 Tuberculosis and Contagion Rounds; Thomas Lowry; Station M.

11:00 - Pediatric Rounds; Erling Platou; Station K.

12:30 - Surgery Grand Rounds; Dr. Zierold; Sta. A.

1:00 - X-ray Conference; Classroom, 4th Floor.

2:00 - Pediatric Rounds; Robert A. Ulstrom; Stations I and J.

Monday, January 12 (Cont.)

Ancker Hospital

8:30 - 10:00 Chest Disease Conference

1:00 - 2:00 Medical Grand Rounds.

Veterans Administration Hospital

8:00 - 9:00 Neuroradiology Conference; J. Jorgens, R. C. Gray; 2nd Floor Annex.

9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

11:30 - X-ray Conference; J. Jorgens; Conference Room, Bldg. I.

2:00 - Psychosomatic Rounds; Bldg. 5.

Tuesday, January 13

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

12:30 - 1:30 Physiology 114D -- Current Literature Seminar; 129 Millard Hall.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

4:30 - EOG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 6:00 X-ray Conference; Presentation of Cases by Veterans Hospital Staff; Eustis Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Medical-Roentgenology Conference; Auditorium.

1:00 - 2:30 X-ray - Surgery Conference; Auditorium.

Minneapolis General Hospital

10:00 - Pediatric Rounds; Spencer F. Brown; Stations I and J.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station F.

12:30 - Grand Rounds; Fractures; Sta. A; Willard White, et al.

12:30 - Neuroroentgenology Conference; O. Lipschultz, J. C. Michael and Staff.

12:30 - EKG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

1:00 - Tumor Clinic; Drs. Eder, Cal, and Lipschultz.

1:00 - Neurology Grand Rounds; J. C. Michael and Staff.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

Tuesday, January 13 (Cont.)

Veterans Administration Hospital (Cont.)

8:30 - Infectious Disease Rounds; Dr. Hall.

8:45 - Surgery Journal Club; Conference Room, Bldg. I.

9:00 - Liver Rounds; Drs. Nesbitt and MacDonald.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery Tumor Conference; L. J. Hay, J. Jorgens; Conference Room, Bldg. I.

1:00 - Chest Surgery Conference; Drs. Kinsella and Tucker; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

3:30 - 4:20 Clinical Pathological Conference; Conference Room, Bldg. I.

Wednesday, January 14

Medical School and University Hospitals

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler, Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Surgery Case; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

12:30 - 1:30 Radioisotope Seminar; 110 Botany Building.

1:30 - 3:00 Physiology 114B -- Circulatory and Renal System Problems Seminar; Dr. M. B. Visscher, et al; 214 Millard Hall.

4:00 - 5:30 Physiology 114C -- Permeability and Metabolism Seminar; Nathan Lifson; 214 Millard Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater, U. H.

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; R. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

2:00 - 4:00 Medical Ward Rounds;

3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Max Seham; Stations I and J.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

11:00 - Pediatric Seminar; Arnold Anderson; Classroom, Station I.

Wednesday, January 14 (Cont.)

Minneapolis General Hospital (Cont.)

11:00 - Pediatric Rounds; Erling S. Platou; Station K.

12:00 - Surgery-Physiology Conference; Dr. Zierold, Dr. E. B. Brown; Classroom.

12:30 - Pediatrics Staff Meeting; Classroom, Station I.

1:30 - Visiting Pediatric Staff Case Presentation; Station I, Classroom.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Conference Room, Bldg. I.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

2:00 - 4:00 Infectious Disease Rounds; Main Conference Room, Bldg. I.

4:00 - 5:00 Infectious Disease Conference; Wesley W. Spink; Conference Room, Bldg. I.

4:00 - Combined Medical-Surgical Conference; Conference Room, Bldg. I.

7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, January 15

Medical School and University Hospitals

8:00 - 9:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Heart Hospital Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

12:30 - Physiological Chemistry Seminar; 214 Millard Hall.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 6:00 X-ray Seminar; Report of Meeting of Radiological Society of North America; Eustis Amphitheater, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Ancker Hospital

4:00 - Medical-Pathological Conference; Auditorium.

Thursday, January 15 (Cont.)

Minneapolis General Hospital

9:30 - Neurology Rounds; Heinz Bruhl; Station I.

10:00 - Pediatric Rounds; Spencer F. Brown; Station K.

10:00 - Psychiatry Grand Rounds; J. C. Michael and Staff; Sta. H.

1:00 - Fracture - X-ray Conference; Dr. Zierold; Classroom.

1:00 - House Staff Conference; Station I.

2:00 - 4:00 Infectious Disease Rounds; Classroom.

4:00 - 5:00 Infectious Disease Conference; Wesley W. Spink; Classroom.

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

8:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

11:00 - Surgery-Roentgen Conference; J. Jorgens; Conference Room, Bldg. I.

Friday, January 16

Medical School and University Hospitals

8:00 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; Audiology; Frank M. Lassman; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold, O. Peterson and Staff; Todd Amphitheater, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

4:00 - 5:00 Physiology 124 -- Seminar in Neurophysiology; Ernst Gelhorn; 113 Owre Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Wallace Lueck; Station J.

10:30 - Pediatric Surgery Conference; Oswald Wyatt; Tague Chisholm; Station I. Classroom.

Friday, January 16 (Cont.)

Minneapolis General Hospital (Cont.)

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

1:00 - 3:00 Clinical Medical Conference; Thomas Lowry; Classroom, Station M.

1:15 - X-ray Conference; Oscar Lipschultz; Classroom, Main Bldg.

2:00 - Pediatric Rounds; Robert Ulstrom; Stations I and J.

Veterans Administration Hospital

1:00 - Pathology Slide Conference; E. T. Bell; Conference Room, Bldg. I.

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

Saturday, January 17

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 10:00 Infertility Conference; Louis L. Friedman, David I. Seibel, and Obstetrics Staff; Station 54.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; L. G. Rigler, J. Friedman, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:30 - Anatomy Seminar; Autogenous Transfusions of Lymphocytes Labelled with 3, 6 Diamino -10- Methylacridinum Chloride; Roland B. Meader; 226 Institute of Anatomy.

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

Minneapolis General Hospital

11:00 - 12:00 Medical - X-ray Conference; O. Lipschultz, Thomas Lowry, and Staff; Main Classroom.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III

8:30 - 11:15 Hematology Rounds; Drs. Hagen, Goldish, and Aufderheide.
11:15 - 12:00 Morphology . . . . . Dr. Aufderheide.