David Fromm

Complications of Gastric Surgery

CLINICAL GASTROENTEROLOGY
MONOGRAPH SERIES

COMPLICATIONS OF GASTRIC SURGERY

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John M. Dietschy, M.D., *Series Editor*

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SERIES PREFACE

During the past decade remarkable progress has been made in our understanding of many basic physiological processes related to liver and gastrointestinal tract functions. Much of this information has led to significant improvements in our understanding of clinical diseases that alter normal hepatic and intestinal function and in the therapy of these diseases. Innumerable examples can be cited. For instance, the application of basic principles of physical chemistry has clarified considerably the manner in which cholesterol is solubilized in bile. Related studies have identified the causes of cholesterol gallstones in several large groups of patients, and specific forms of therapy for the prevention or the dissolution of such stones are now available. Other experimental work that relies heavily on basic techniques of immunology and electron microscopy has identified specific infectious agents affecting liver function. These studies, in turn, have provided considerable insight into the different clinical syndromes included under the general heading of viral hepatitis, raising the possibility that effective immunization against these organisms may soon be available. Equally impressive advances have been made in our understanding of the control gastric secretion and peptic ulcer disease, in the causes of intestinal malabsorption, and in radiographic and endoscopic methods for examining the liver and gastrointestinal tract.

This explosion of knowledge in gastroenterology poses a particularly difficult problem for those interested in the dissemination of new medical information to students, house officers and medical practitioners. Often advances have come so quickly that the information presented in standard textbooks is outdated before the books become available. Also, it is difficult to revise such texts rapidly because of the large number of authors involved and the long production time necessary for these books. Finally, the space available to authors for extensively reviewing both the basic physiological concepts and their clinical implications is limited in most texts and in more rapidly published medical journals.

This series of volumes published under the general title "Clinical Gastroenterology Monographs" was conceived and designed to overcome many of these difficulties and to bring to the medical practitioner the most current information on the pathophysiology and treatment of major areas of disease affecting the liver and gastrointestinal tract. Each volume covers an important group of related disorders and is sufficiently long to allow for extensive discussion of their basic pathophysiological, clinical, and therapeutic aspects.

New volumes will appear regularly, and a special effort will be made to identify areas for inclusion in the series in which there is a rapidly expanding body of information relevant to the care of patients with a particular gastrointestinal disorder. Existing volumes will be updated and republished frequently where continued advances in information justify such rapid revision.

It is hoped that this series will provide a continuously evolving and current reference source for the broad spectrum of physicians who deal with patients with diseases of the liver and gastrointestinal tract.

John M. Dietschy, M.D.

PREFACE

There is substantial uncertainty regarding the pathophysiology and treatment of many of the conditions occurring after gastric surgery. As a result, considerable controversy and even feelings of helplessness exist. Perhaps a manifestation of the latter is that there are more prospective controlled and retrospective studies on the incidence of symptoms occurring after surgery than there are on the treatment of such symptoms. The purpose of this monograph is to review several of the syndromes occurring after operations upon the stomach. While this volume is not intended as a substitute for other books or journals, it is hoped that it will be of use to those interested in the care of patients with symptoms attributable to their operation, the pathophysiology of such symptoms, and the development of more rational treatment.

A number of people have been instrumental in the completion of this monograph. I particularly want to thank Sharyn Brooks for her humor, patience, organizational ability, and many hours of help; Beverly Smith, Susan Dillon, and Virginia Geiss for their skilled help; Harriet Greenfield for her artwork; William Silen for his encouragement; the Countway Library staff; Fred Bernardi at John Wiley & Sons for his fortitude; and last, but not least, Barbara, Marc, Ken, and Kathy for their many hours of support and understanding. This work was supported in part by RCDA AM 00053 from the National Institute of Arthritis Metabolism and Digestive Diseases.

David Fromm, M.D.

CONTENTS

1 INTRODUCTION

The ideal operation for peptic ulcer disease is one that is free of mortality, recurrent ulcer, and morbidity. Such an operation does not yet exist. In fact, there is no single operative procedure for peptic ulcer disease that has been universally accepted. There have been waves of enthusiasm claiming better results for one operation over another, but many such claims have been based on clinical impressions or incomplete data. In recent years, however, well-controlled clinical trials comparing various ulcer operations have been published. These studies form a basis upon which one can make a more rational judgment as to the choice of operation for peptic ulcer disease. For malignant conditions requiring operations on the stomach, there are generally fewer surgical options. In the pages that follow, no conscientious effort is made to advocate one operative procedure over another for the treatment of peptic ulcer disease, nor is there any attempt to describe all of the operative procedures currently in use. The fact remains that only about 50 to 60% of patients (and in some series even less) after gastric resection or vagotomy with drainage are without symptoms resulting from their operation. The patient acceptance rate, however, is much higher, but this situation is a tradeoff, accepting new symptoms in place of older ones. From the patient's point of view it makes little difference which operation was performed if there are significant symptoms after recovering from the acute effects of the operation.

This mongraph deals with many of the complications that may occur after a patient has recovered from the acute effects of gastric surgery. It is relatively easy to classify postoperative symptoms, but it may be very difficult to categorize the etiology of such symptoms. In large part, this is due to similarity of symptoms found in the various syndromes occurring after gastric surgery. The hazard of not trying to determine the precise cause of a patient's symptoms is that inappropriate treatment may result. Those physicians who have become cynical will maintain that even though the etiology of a patient's symptoms has been determined, the current methods for treatment frequently are limited and not always successful. To some extent and in select instances this is true, but fortunately, success can be achieved in many instances. Failures of treatment often are attributed to psychological factors that appear to occur in some patients, but Jordan stated another aspect of this problem clearly: "It is true that the psychoneurotic patient who experiences any of the [postgastrectomy] syndromes . . . is more likely to be incapacitated than the stoic person who seldom complains" (1). Other problems are that the definition of successful treatment of the physiological

aberrations created by surgical procedures varies, and the failures of treatment frequently are not critically analyzed. Although the syndromes that may occur after operation are staggering in number, there is no need to take the attitude that a patient must have suffered for years with ulcer symptoms before surgery is advised. Even though many patients after operation will have symptoms, these vary in extent and severity and the majority do not require extensive diagnostic procedures or even treatment. These patients do, however, require careful follow-up, for certain of the problems that they may encounter are insidious in onset or are unfortunately accepted as inevitable.

In order to understand the anatomical and physiological basis of many of the postgastric surgery sequelae, there must be a limited knowledge of the major operative procedures used. What immediately follows is a brief description for those unfamiliar with the various commonly employed operations.

Basically, there are three operative concepts used to treat peptic ulcer disease: 1) interruption of the parasympathetic nervous system influence on gastric secretion, 2) reduction of the parietal cell mass, and 3) excision of the antral gastrin cell-bearing area. In the case of gastric ulcer proximal to the distal antrum, an additional concept is used: removal of the ulcer-bearing area. Frequently the above concepts are combined, since a mixture may be more effective in preventing recurrence of ulceration.

VAGOTOMY (2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12)

The distribution of the vagal innervation to the abdominal viscera has three components: 1) the two vagal trunks, 2) the esophageal plexus and 3) the four truncal divisions. The esophageal plexus begins below the root of the lung, and is formed by a number (usually three to four) of communicating branches from the right and left vagus nerves. Near the lower end of the thorax, the vagi tend to form single trunks, with the left trunk passing along the anterior surface of the esophagus and the right on the posterior surface. In about 80% of people, the vagi pass through the esophageal hiatus as single trunks. About 10 to 30% of the time, there is at least one additional branch running a parallel course. The vagal system has no anatomical relationship with the diaphragm, so the relationship between plexus or trunks or truncal divisions and the terminal esophagus may be variable.

The four divisions of the vagi are: hepatic, coeliac, anterior and posterior gastric. The left vagal trunk gives a variable number of branches forming the hepatic division, which traverses the lesser omentum, sending branches to the biliary tract, liver, pylorus, proximal duodenum, and head of the pancreas. The right vagal trunk forms the coeliac division, which innervates the body and tail of the pancreas, entire small intestine, and colon to the splenic flexure. The anterior and posterior gastric divisions run in the lesser omentum parallel to the lesser curvature of the stomach, supplying the anterior and posterior aspects of the stomach respectively. The gastric divisions (frequently referred to as the anterior and posterior nerves of Latarjet) terminate in the antrum, where they fan out into branches looking like the digits of a crow's foot.

There are three types of vagotomy that may be performed (Figure 1). A

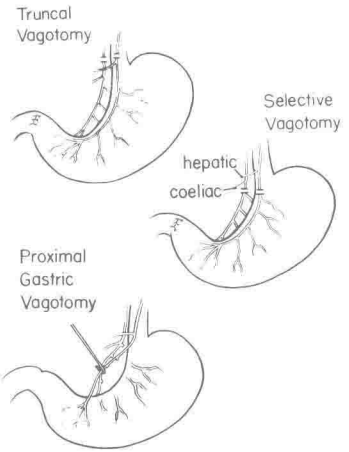

FIGURE 1
Types of vagotomy.

truncal vagotomy involves division of all vagal structures on or about the lower esophagus. Most surgeons prefer to do this operation subdiaphragmatically, but whether it is done from within the abdomen or the chest, the result is parasympathetic denervation of the entire stomach in addition to other abdominal viscera. A selective vagotomy involves division of the vagal structures below the origin of the hepatic and coeliac divisions. Thus, the vagal supply of the entire stomach is interrupted, but the remaining visceral supply is preserved. The third form of vagotomy involves denervation of the parietal cell mass by division of the branches of the anterior and posterior gastric divisions; the antral innervation of the nerves of Latarjet are left intact. This operation has been called by a variety of names: highly selective vagotomy, ultra selective vagotomy, parietal cell vagotomy, or selective proximal vagotomy. However, the preferred term for this type of vagotomy is *proximal gastric vagotomy.* Following truncal or selective vagotomy, the total gastric denervation results in a slowing of the process of emptying of solids; this must be overcome by some type of permanent gastric drainage procedure. A drainage procedure is not required with proximal gastric vagotomy, since the innervation to the antrum is intact and, therefore, its motility preserved.

DRAINAGE PROCEDURES

There are two basic concepts used to provide a means of drainage of the stomach after vagotomy without gastric resection: 1) enlargement, or destruction, of the pylorus, which is referred to as *pyloroplasty,* and 2) bypassing the pylorus (Figure 2). The two most commonly used pyloroplasties are the Heineke-Mikulicz and the Finney. The Heineke-Mikulicz pyloroplasty is performed by incising the pyloric ring and adjacent stomach and duodenum longitudinally. The incision is then closed transversely. The Finney pyloroplasty is a more extensive procedure in which a longer incision is made through the pylorus and adjacent walls of the

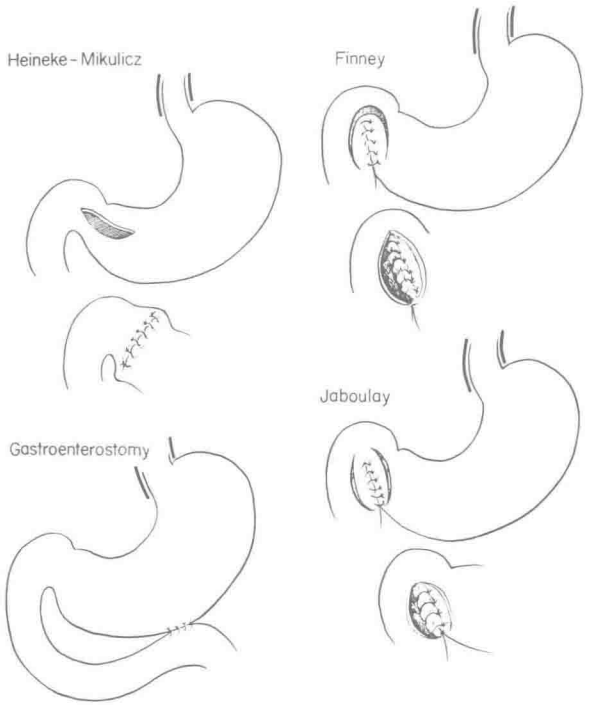

FIGURE 2
Drainage procedures.

stomach and duodenum. An inverted U-shaped anastomosis is then made between the stomach and duodenum. Technical considerations may favor the Finney pyloroplasty, particularly in the presence of extensive scarring of the duodenum. The two most commonly used drainage methods for bypassing the pylorus are a gastrojejunostomy or a gastroduodenostomy (Jaboulay), as shown in Figure 2.

GASTRECTOMY

The amount of stomach removed at operation varies and depends upon the indications for operation and in some instances whether o not vagotomy is performed. The majority of present-day gastric resections encompass the pylorus. There are two ways to re-establish gastrointestinal continuity: anastomosis of the remaining stomach to the duodenum (referred to as a Billroth I procedure, or anastomosis, or gastrectomy) or to the jejunum (referred to as a Billroth II procedure, or anastomosis, or gastrectomy) (Figure 3). In the latter instance the proximal duodenum is closed and the jejunum in the vicinity of the ligament of Treitz is anastomosed to the stomach. Although many surgeons prefer the Billroth I procedure, technical considerations at the time of operation may preclude safe anastomosis of the stomach to the duodenum. When performing a Billroth II gastrectomy, many surgeons prefer to anastomose the entire transected end of the remaining stomach to the side of the jejunum (frequently referred to as a Polya gastrectomy) rather than closing the

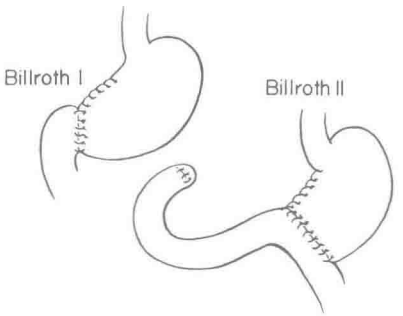

FIGURE 3
Billroth I gastrectomy and Billroth II gastrectomy.

lesser curvature side and anastomosing only a portion of the transected greater curvature side (Hofmeister gastrectomy). Although the former method (Polya) results in a longer anastomosis, it does not carry any implication as to the rate of gastric emptying. The rate-limiting factor is the transverse diameter of the jejunum (13).

TOTAL GASTRECTOMY

The commonest indications for total gastrectomy are the Zollinger-Ellison syndrome and carcinoma of the stomach. Gastrointestinal continuity may be reestablished by anastomosing the end of the esophagus to the duodenum or jejunum (Figure 4). There also are various procedures for making a jejunal pouch to act as a reservoir for food (see Figure 4, this chapter, and Figure 7, Chapter 2).

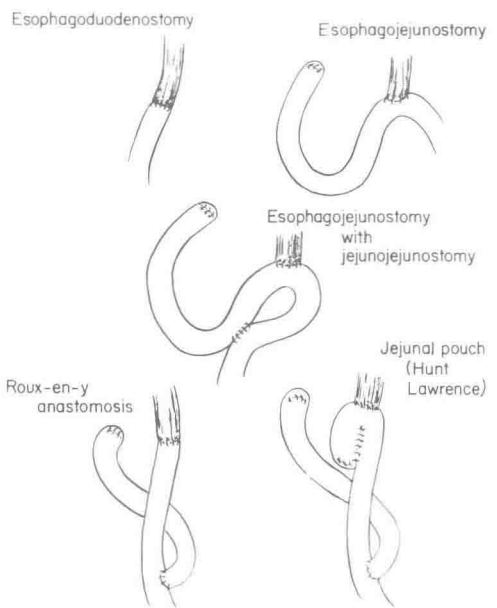

FIGURE 4
Methods for reconstruction of esophagointestinal continuity after total gastrectomy.

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DUMPING SYNDROMES

DEFINITION, INCIDENCE, SYMPTOMS

There are very few syndromes that have generated so much controversy and confusion as the dumping syndrome. Experimentally, the syndrome is hard to study because of difficulty in defining objective symptoms in animals. Clinically, few have really defined what they mean by dumping. It is generally agreed that in its full form, the dumping syndrome consists of both gastrointestinal and cardiovascular, or vasomotor, symptoms. The intestinal components include epigastric distress, bloating, fullness, cramping abdominal pain, nausea, vomiting, and diarrhea. The cardiovascular manifestations include weakness, dizziness, pallor, blurred vision, vertigo, palpitations, sweating, tachycardia, and increased peripheral blood flow. It has been emphasized by many that dumping symptoms occur in various forms with any or all meals. Several investigators writing on this subject have been willing to attribute to dumping any of the above-mentioned symptoms, yet others (1) feel that symptoms of vasomotor imbalance are dominant.

The reported incidence of the dumping syndrome ranges from zero to 100% of patients who have had surgery for peptic ulcer disease (2,3,4,5,6,7,8,9). This great disparity in incidence most likely is related to the problem of definition. Thus, the true incidence of dumping is difficult to evaluate. For example, if patients who complain of mild epigastric distress with eating are considered to have dumping, then the incidence of the syndrome will be quite high. On the other hand, if one insists that a history of vasomotor phenomena is required for the diagnosis, the incidence will be lower. Those who are willing to make the diagnosis of dumping on the basis of a single or a variety of gastrointestinal symptom(s) alone may miss other syndromes that may follow gastric operations. The confusion is further compounded by the erroneous labeling of other postgastrectomy disorders as part of the dumping syndrome. It is also unfortunate that the dumping syndrome has been classified into an early and late form. This distinction does not refer to the time interval following operation. What has been referred to as late dumping consists of symptoms of hypoglycemia occurring an hour or more after a meal rather than the gastrointestinal and/or vasomotor symptoms of "early" dumping, which bear a reasonably close time relationship to a meal.

Any or all of the symptoms of dumping typically begin during a meal or within 5 to 45 minutes after completion of a meal and in general subside within an hour. Classically, the dumping syndrome is precipitated by a "heavy" meal or

one that contains a large amount of carbohydrate. However, the symptoms of dumping may occur in various forms with any or all meals. Fortunately, most patients with the dumping syndrome have mild, transitory symptoms. Many of those with symptomatic vasomotor phenomena intuitively learn that assumption of the recumbent position during the onset of symptoms will afford partial or complete relief. Those with significant symptoms quickly realize that eating precipitates their discomfort, and therefore may lose weight as a result of restricted dietary intake. Others, by trial and error, know that certain types of foods willl provoke symptoms.

Several reports have suggested that the incidence of the dumping syndrome is higher in women than men, but this has been disputed by other studies which indicate that the incidence or severity of symptoms does not depend upon the sex of the patient (3,7,9,21). It is believed by many that younger patients, age 20 to 30, and in particular those with a short history of ulcer disease and minimal symptoms prior to operation have a higher incidence of dumping, but other studies with careful follow-up suggest that the age of the patient does not play a significant role in the incidence of the dumping syndrome (3,7). The duration of ulcer symptoms before operation has been shown by several authors to influence neither the severity nor the incidence of the dumping syndrome (3,27,28). Some authors have indicated that the site of the initial ulcer plays a role in determining the appearance of the dumping syndrome, but this too has been disputed in patients followed carefully after operation (3,20).

OPERATIVE PROCEDURES AND INCIDENCE

It is generally thought that the severity of dumping symptoms is directly proportional to the extent of the gastric resection (5,10,11,12,13,14), but this is not uniformly agreed upon (15,16,17). Several authors have felt that the incidence of dumping following a Billroth I gastrectomy is singificantly less than that occurring after a Billroth II gastrectomy, but there are other series (11,18,19) in which no significant difference has been found in the incidence of dumping between these two operations. It is also believed by some that the severe form of the syndrome seems to be more common after a gastrojejunostomy (3,20). In the prospective Leeds/York controlled trial of elective surgery for duodenal ulcer (21), the incidence of dumping syndrome was approximately 13 to 18% following vagotomy and gastroenterostomy, approximately 9% following vagotomy and antrectomy with gastrojejunal anastomosis, and approximately 22% following subtotal gastrectomy. The reason for the much lower incidence following an antrectomy is not clear (21). Following vagotomy and pyloroplasty, the Leeds/York trial found the incidence of dumping to be approximately 12%. The different incidence in dumping after vagotomy and pyloroplasty compared to vagotomy and gastroenterostomy was not significant (22). In another double-blind, randomized control trial of selective vagotomy with pyloroplasty or gastrojejunostomy, the incidence of dumping, either mild or severe, was not significantly different for the two operations (23). In fact, the incidence of mild and severe dumping was less following gastrojejunostomy. A vasomotor component, in addition to gastrointestinal symptoms, was included in the definition of