An Internist's Illustrated Guide to Gastrointestinal Surgery

Edited by George Y. Wu, MD, PhD Khalid Aziz, MBBS, MRCP Giles F. Whalen, MD, FACS Illustrations by Lily H. Fiduccia









AN INTERNIST'S ILLUSTRATED GUIDE TO GASTROINTESTINAL SURGERY

CLINICAL GASTROENTEROLOGY

George Y. Wu, MD, PhD, Series Editor

- An Internist's Illustrated Guide to Gastrointestinal Surgery, edited by George Y. Wu, MD, PhD, Khalid Aziz, MBBs, and Giles F. Whalen, MD, 2003
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DEDICATION

This book is dedicated to my students, whose questions prompted the writing, my family, whose patience permitted its creation, to Sigmund and Jenny Walder, who have supported and encouraged us in all of our academic endeavors, and to Herman and Frances Lopata and their family, whose generosity toward our research has made available the time to devote to this book.

G. Y. W.

To the memory of my parents, whose guidance has provided me with inspiration for all of my accomplishments in life.

K.*A*.

To my teachers who have inspired me by their example, to my students who teach me still by their questions and curiosity, to my patients whose lessons I have tried to absorb, and to my family whose patience and tolerance of these endeavors make it all worthwhile.

G. F. W.

Foreword

Few clinical disciplines have been transformed so dramatically by advancements in science and technology as gastrointestinal surgery. To begin with, modern pharmacology has virtually eliminated some kinds of surgery altogether. If one were to take a peek at a typical operating room schedule in a busy hospital of the 1960s, gastrectomies of one kind or another would have constituted a large block of the major surgeries. The advent of effective H2-histamine receptor antagonists and, more recently, the H⁺,K⁺-ATPase (proton pump) inhibitors led to a precipitous decline in those procedures such that they are rarely performed today. Exciting new approaches to treating inflammatory bowel diseases and their complications—such as fistulas with anticytokine therapy may one day have a similarly profound effect on surgery for this condition as well.

Beyond pharmaceutics, advances in imaging techniques have greatly facilitated the identification and characterization of pathology in the gastrointestinal tract in a way that would have been unimaginable only a few years ago. Just to visualize the pancreas in some way was a horrendous task until abdominal ultrasound, magnetic resonance imaging, or computer tomography made it simple. The fact that the gut is a hollow organ that can be accessed through the mouth, anus, or even through the wall of the abdomen has been fully exploited with fiberoptic endoscopes that can bend around corners with ease and permit surgery to be conducted through them. Many physicians have earned their spurs in the operating room by laboriously hanging on to a Deaver retractor while a surgeon deftly removes a patient's gallbladder. Today, of course, laparoscopic surgery has virtually eliminated open cholecystectomy and threatens to make other complex surgeries, such as fundoplication or colectomy, obsolete. Other advanced technologies, such as transhepatic intravenous porta-systemic shunts, have practically converted dangerous and difficult operations to relieve portal pressure in liver disease to an outpatient procedure.

Despite these amazing advances, today's surgeon may still be called on to perform virtually all of the operations that have been performed for years, some even for centuries. Gastrectomies, cholecystectomies, fundoplications, colectomies, and porta-caval shunts all have to be performed on patients. The surgeon of today must be equally adept at performing traditional abdominal surgery as well as surgery through scopes, percutaneous wires, and the like.

The transformation that surgeons have had to make in the recent past has also necessitated change in the internist's practice. To begin with, the internist now has many options to choose from in treating patients with abdominal illnesses. It is important for the internist to understand the advantages and limitations of the different therapeutic approaches that might be taken. Thorough discussion and collaboration of an internist with the surgeon, both being well-informed on the approaches to therapy, will inevitably provide the best outcome for the patient. Beyond initial therapy, the internist almost certainly sees patients who have undergone various surgical procedures. It goes without saying that internists must be adept at handling the sequelae of surgery, some of which may have profound effects on normal physiological function.

An Internist's Illustrated Guide to Gastrointestinal Surgery by Wu, Aziz, and Whalen is directed at educating the internist on the common surgical approaches to gastrointestinal disorders. It is carefully written in language that would have meaning to an internist. In a logical way, each topic is approached from the standpoints of pathophysiology, diagnostic evaluation, treatment, and sequelae. Each chapter is accompanied by clear and simple diagrams that depict the essentials of the operation performed. The book covers both the "old surgery" of gastrectomies, colectomies, and cholecystectomies, as well as the "new surgery" of shunts, laparoscopic procedures, and TIPS. It is meant not only for the practicing internist but is equally appropriate for all students or other trainees in medicine who are bound to see patients who undergo surgery for gastrointestinal illness. An Internist's Illustrated Guide to Gastrointestinal Surgery should not only provide the reader with an understanding of the science and practice of gastrointestinal surgery, but also equip the reader with the tools to be a better physician.

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PREFACE

In general, primary care providers, family practitioners, and gastroenterologists have a limited knowledge of abdominal surgical operations, the medical aspects of these surgical procedures, and their immediate and late complications. In addition, these patients traditionally are not followed up by the surgeons, and thus the internist must become familiar with postsurgical problems in order to provide appropriate long-term care. A clear understanding of the concepts that underlie the surgery is crucial for proper management of these patients.

In addition, within the last 10 years, laparoscopic surgery has become increasingly commonplace, with new laparoscopic procedures being developed at a rapid pace. There are vast differences between traditional and laparoscopic surgery, not only in the way these procedures are performed, but also in their outcomes and complications. Many internists, as well as surgeons, have very limited understanding of these procedures. Therefore, the need exists for a book that can provide useful clinical information in an easy to access format, covering a variety of abdominal surgical procedures.

Almost all surgical books provide great detail about the technical aspects of surgical procedures and their surgical complications. However, the physician who needs to manage the patient who has undergone gastrointestinal (GI) surgery, currently must go through surgical texts to find the disease, and then the type of surgery the patient has undergone, wading through pages of details about the surgical procedure, without dealing with the issues relevant to the medical management of the patient. Thus, it is currently difficult for the nonsurgically trained physician to extract the relevant medical information. *An Internist's Illustrated Guide to Gastrointestinal Surgery* is a comprehensive textbook describing all of the surgical and laparoscopic procedures for the GI tract in a simple way, with artistic illustrations to educate the physician about surgery of the GI tract, and to provide not only clear descriptions of the changes in the anatomy and physiology, but also advice on medical management of the postsurgical patient.

An Internist's Illustrated Guide to Gastrointestinal Surgery describes in detail the indications, contraindications, anatomical alterations, and physiological alterations that result from various GI operations and procedures. Comparison between alternative operations, complications, medical management issues, and costs of these surgical procedures and operations are discussed. Clear, detailed, artist-rendered illustrations of the anatomy before and after surgery are included and, where appropriate, radiological images before and after surgery.

This is a unique textbook, written primarily for primary care physicians, general internists, and gastroenterologists to educate them about those aspects of GI surgery—including laparoscopic surgery—that are pertinent to an internist. It should also be a suitable textbook for medical students, residents, nurses and nurse practitioners, nutritionists, dietitians, and various subspecialists, who often take care of postsurgical patients.

The editors are indebted to the invaluable assistance of Jocelynn Albert. This project would not have been possible without her dedication and organizational skills.

George Y. Wu, MD, PhD Khalid Aziz, MBBS Giles F. Whalen, MD

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I ESOPHAGEAL SURGERY

Esophagectomy and Reconstruction

Michael Kent, MD, Jeffrey Port, MD, and Nasser Altorki, MD

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INTRODUCTION EPIDEMIOLOGY OF ESOPHAGEAL CANCER PREOPERATIVE EVALUATION TREATMENT OPTIONS FOR ESOPHAGEAL RECONSTRUCTION MANAGEMENT OF COMPLICATIONS COST OF SURGERY AND FUNCTIONAL OUTCOME SUMMARY REFERENCES

INTRODUCTION

Esophagectomy is one of the most formidable operations performed by the gastrointestinal (GI) surgeon. Esophageal resection carries a complication rate of more than 40%, and should only be performed in centers experienced with the management of these patients. Indeed, the mortality of esophagectomy has been shown to be significantly lower in larger volume centers (1).

Esophageal resection is most frequently performed for carcinoma of the esophagus. Although less common, several other benign conditions may necessitate esophageatomy. For example, severe caustic burns to the esophagus often require esophageal resection and reconstruction. Esophageal perforation, primary motility disorders such as achalasia and scleroderma, and unsuccessful antireflux operations are additional indications for esophagectomy. Usually, these diseases may be managed with esophageal-sparing surgery, such as fundoplication or myotomy. Esophagectomy often represents the final treatment of patients with a variety of benign conditions who have failed more conservative surgical management.

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EPIDEMIOLOGY OF ESOPHAGEAL CANCER

Although the prevalence of esophageal cancer reaches nearly epidemic levels in certain parts of Central and Southeast Asia, it remains a relatively uncommon disease in the United States. The American Cancer Society estimates that 13,000 patients have been diagnosed with esophageal cancer in 2001. Unfortunately, the majority of these patients will present with advanced disease not amenable to curative treatment. Despite the advent of novel chemotherapeutic agents and refinements in surgical technique, the overall 5-yr survival of patients with carcinoma of the esophagus remains in the range of 5-10%.

Esophageal cancer may develop as either a squamous cell or an adenocarcinoma. Although the clinical presentation is similar, the epidemiology and risk factors of these two histological subtypes differ markedly. Worldwide, squamous cell carcinoma is the more common. However, the incidence of squamous cell cancer exhibits a remarkable variability, with a "cancer belt" extending from northern Iran, through Central Asia, and into Northern China. Indeed, the disease accounts for almost 25% of all cancer deaths within the People's Republic of China (2). Outside these endemic areas, squamous cell carcinoma is far less common. However, clusters of high incidence have been identified in Northern France and Italy, as well as major metropolitan centers within the United States, such as New York, Los Angeles, and Washington, D.C. (3).

Several environmental factors have been clearly implicated in the development of squamous cell cancer of the esophagus. In the Western Hemisphere, alcohol and tobacco consumption are significant risk factors. The risk of both tobacco and alcohol use are strongly dose-related (4,5). The consumption of both seems to exert a synergistic rather than an additive effect. In part, this may owe to the ability of alcohol to improve the diffusion of tobacco-related carcinogens through the esophageal wall (6). Interestingly, in those locations where squamous cell cancer has its highest incidence, neither tobacco nor alcohol use seem to be significant risk factors. Instead, dietary components such as fermented fish or pickled corn that are rich in secondary amines have been implicated (7). The ingestion of hot beverages such as tea that are potentially caustic to the esophagus has also been postulated to predispose to squamous cell carcinoma (8). Finally, the observation that malignant cells may contain papillomavirus particles has suggested a possible infectious etiology (9).

Although squamous cell carcinoma had been the most common type of esophageal cancer in the United States 20 yr ago, adenocarcinoma is now the more prevalent. This change reflects an increase in the incidence of adenocarcinoma of almost 10% per year every year during the 1980s. This surge surpasses the increase in incidence of lung cancer, melanoma, and non-Hodgkin's lymphoma during the same period (10). Although the reason for this change is not known, it likely parallels the rise of cases of Barrett's esophagus, known to be a precursor to adenocarcinoma (11). It has been estimated that Barrett's esophagus increases the lifetime risk of developing adenocarcinoma retain residual Barrett's metaplasia (12). Given the likelihood that in other cases the metaplastic mucosa may have been completely overgrown with tumor, it appears that the majority of cases of adenocarcinoma are associated with Barrett's esophagus. The association between Barrett's esophagus and chronic gastroesophageal reflux has led to an intensive search for the responsible carcinogens. It appears that gastric and biliary reflux in com-

bination rather than either alone, which contributes to malignant transformation of the esophageal mucosa (13). It has been suggested that the increasing use of H_2 blockers has also contributed to the rise of Barrett's esophagus and adenocarcinoma. However, this hypothesis is solely observational and a causative relationship has been difficult to establish.

In addition to Barrett's esophagus, several less common conditions have been associated with the development of esophageal cancer. For instance, the risk of esophageal cancer has been estimated to be 30-fold higher in patients with achalasia compared with the general population (14). Typically, these patients develop large, squamous cell tumors located in the middle-third of the esophagus. Unfortunately, the majority of patients present with advanced, unresectable disease. This is in part owing to the fact that the symptoms of carcinoma are difficult to distinguish from those of achalasia itself. Other conditions, such as tylosis, Plummer-Vinson syndrome, and caustic strictures are also known to predispose to esophageal cancer.

PREOPERATIVE EVALUATION

All patients considered for esophagectomy must undergo a thorough preoperative evaluation. The length of the procedure and high incidence of complications necessitate that elective surgery be performed only when comorbidities have been optimally managed. The majority of patients undergoing esophagectomy have coexisting pulmonary and cardiac disease and for this reason pulmonary function tests and cardiac stress studies are routinely obtained. Indeed, the FEV₁ is one of the most accurate predictors of postoperative mortality (15). Often, the incidence of postoperative complications can be greatly diminished by simple measures such as smoking cessation and a trial of antibiotics and inhaled bronchodilators.

In addition to a medical evaluation, patients with esophageal cancer must undergo preoperative staging prior to esophagectomy. Unfortunately, more than 50% of these patients will have unresectable disease at the time of their initial presentation. As in all fields of oncology, the main goal of staging is to ascertain which patients harbor locally advanced or metastatic disease, which would preclude curative surgery.

Several studies are routinely performed to stage esophageal cancer. A barium swallow is the initial study obtained in any patient who presents with dysphagia. This is customarily followed by esophagoscopy, which can provide vital information to the surgeon and oncologist. Most importantly, biopsy obtained during endoscopy will provide a tissue diagnosis. In addition, the length of esophagus involved by tumor, the presence of a hiatal hernia, and underlying Barrett's mucosa can all be determined at the time of endoscopy (Fig. 1). For tumors involving the upper- and middle-third of the esophagus, bronchoscopy is also necessary to exclude invasion of the trachea by tumor, which would imply unresectability. Computed tomography (CT) scanning is also routinely obtained in all patients with esophageal cancer. Although CT is not able to accurately determine nodal status and the depth of mural invasion, it is very sensitive in detecting the presence of distant disease, such as pulmonary or hepatic metastases.

Many other modalities to stage esophageal cancer have been reported and gained some degree of acceptance. Endoscopic ultrasound (EUS) is one modality that has become widely used in the past decade (16). EUS can accurately assess both the depth of invasion of the esophageal wall by tumor, as well as the presence of local lymphad-



Fig. 1. Endoscopic view of an esophageal tumor.



Fig. 2. Endoscopic ultrasound image of an esophageal tumor invading the muscular wall of the esophagus.

enopathy (Fig. 2). EUS can also allow for fine-needle aspiration of these lymph nodes. Finally, some groups have advocated more invasive methods of staging such as thora-

coscopy and laparoscopy (17). Although these procedures are clearly sensitive for detecting extra-esophageal disease, it is not clear how much additional information is provided compared with standard modalities such as EUS and CT scanning.

TREATMENT

Surgery, radiation therapy, and chemotherapy, either alone or in combination, have all been claimed as standard therapy of esophageal carcinoma. In part, this controversy stems from the generally poor outcome of any treatment modality. Although most surgical series studies report 5-yr survival rates of only 25%, esophagectomy is nonetheless considered to offer the best potential for cure. Recently, several randomized, controlled clinical trials have evaluated whether the addition of chemotherapy and radiation therapy to surgery offers any benefit. No study to date has supported the use of either of these modalities alone (18,19). However, the utility of combined induction chemoradiation is more controversial. Several small single-arm series has shown benefit for this approach compared with historical controls (20,21). However, three large, randomized trials have reported mixed results (Table 1) (22-24). Of these three, only one study demonstrated a statistically significant difference in survival with induction chemoradiation compared with surgery alone (24). This study has been criticized for the unusually poor survival rate (6%) in the surgical arm. To date, therefore, we consider surgical resection alone to be the standard of care for patients who are acceptable candidates.

As with nonoperative therapy, the surgical options for management of esophageal cancer are numerous. The two approaches most commonly used are the transthoracic (TTE) and the transhiatal esophagectomy (THE). The TTE exposes the esophagus through either a right or left thoracotomy, depending on the location of the tumor and the preference of the surgeon. In general, tumors of the distal third of the esophagus are best exposed through a left thoracotomy, those of the middle- and upper-third through a right thoracotomy. Regardless of the exposure, the principles of the operation do not differ: mobilization and resection of the involved esophagus with adequate margins, removal of adjacent lymph nodes, and the restoration of continuity of the GI tract. The esophagus must be completely mobilized from the diaphragmatic hiatus to the thoracic inlet to permit safe resection. Although tissue bearing lymph nodes is removed with the specimen, a meticulous lymph node dissection is not part of the standard esophagectomy. To restore continuity of the GI tract, a substitute for the esophagus must be found. Most commonly, the organ used for this purpose is the stomach. To do this, the stomach must be freed from its peritoneal attachments. If a left thoracotomy is used, the stomach may be exposed and mobilized through an incision in the diaphragm. If a right thoracotomy has been chosen, an additional upper abdominal incision will also be necessary. The greater curvature of the stomach is then freed from the omentum. A stapler is then fired across the lesser curve, in order to fashion the stomach into a tube appropriate for anastomosis with the remaining esophagus (Fig. 3A).

The vascular supply of this gastric tube is based on the right gastroepiploic artery, which must be preserved during mobilization of the stomach. Finally, the prepared gastric tube is then passed under the aortic arch and attached to the esophageal stump. Typically, the esophageal anastomosis is located within the mediastinum. However, a separate incision may be made in the neck to fashion a cervical anastomosis.

The transhiatal esophagectomy (THE) has become a popular alternative to a TTE, in part based on the belief that many potential complications are avoided by not entering

Author	No. of Patients	TR dose (GY)	Chemotherapy	Operative mortality (%)	Complete Pathologic Response	Mediam Survival Time (YR)	Survival rate (%)
Urba et al. (1997)	100	45	CDDP-BL-VBL	Surg-NS	NS	NS	33 (3 yr)
				CRT-NS	NS	NS	18 (3 yr)
Walsh et al. (1996)	113	40	CDDP-FU	Surg-3.6	_	11	32 (3 yr)
				CRT-8.6	25%	6	6 (3 yr)
Bosset et al. (1997)	297	18.5	CDDP	Surg-3.6	_	18.6	38 (3 yr)
				CRT 12.3	26%	18.6	38 (3 yr)

 Table 1

 Randomized Trials of Chemoradiotherapy Followed by Surgery Compared to Surgery

Abbreviations: CDDP = cis- platinum, FU = 5- fluorouracil, BL = bleomycin, VBL = binblastine, NS = not stated, Surg = surgical arm, CRT = chemotherapy radiotherapy plus surgery arm, CT = chemotheraphy, TR = total radiation.



Fig. 3. (A) Gastric pull-up. (B) Colonic transposition (Adapted from Shackelford's Surgery of the Alimentary Tract, Volume I, Fifth Edition, WB Saunders, 2002).

the chest. THE differs from TTE in two important respects. First, the thoracic esophagus is entirely mobilized through the hiatus of the diaphragm, without the need for a thoracotomy incision. Second, the tubularized stomach is brought up into the neck where a cervical anastomosis is preformed. Proponents of this approach report decreased pain and pulmonary complications by avoiding a thoracotomy. In addition, an anastomotic leak within the neck is much easier to manage. Usually, the incision can be opened at the bedside and the leak safely drained. In contrast, a mediastinal leak carries a 50% mortality and often requires operative reexploration and possible takedown of the anastomosis. Critics of THE note that the operation affords a less-complete lymphadenectomy. In addition, the leak rate from a THE may be slightly higher, because the stomach must be mobilized further and the anastomosis carried higher than for a TTE. However, in the hands of qualified esophageal surgeons, the operative approaches are essentially equivalent. The operative mortality, incidence of complications, and length of stay have never been shown to differ between these operations. Furthermore, and most importantly, the 5-yr survival following a standard esophagectomy is a consistent 25%, whether the approach be transthoracic or transhiatal (25,26).

Several modifications have been proposed to improve the disappointing cure rate of a standard esophagectomy. An *en bloc* esophagectomy offers to the esophageal surgeon what is a standard principle to other surgical oncologists: removal of the involved organ with an envelope of adjoining normal tissue. This envelope of normal tissue should include the posterior pericardium, both pleural surfaces where they abut the esophagus, and the lymphovascular tissue between the esophagus and the spine. The deep location of the esophagus within the mediastinum, however, makes this a more challenging operation.

The evolution of a more formal lymph node dissection represents a further refinement in esophageal surgery. The basis for this stems from the distribution of lymphatic drainage within the esophagus. Unlike other organs of the gastrointestinal tract, the abundant lymphatic channels of the esophagus course longitudinally within the submucosa of the esophagus for long distances before draining to adjacent lymph nodes. However, in a standard esophagectomy, little attempt is made to remove any lymphatic tissue distant from the primary tumor. Perhaps, this in part explains the disappointing local recurrence rates (20–60%) following the standard operation. In a "two-field lymphadenectomy," the standard operation is modified to include the systematic removal of middle and lower mediastinal nodes (periesophageal, parahiatal, subcarinal, and aortopulmonary) and upper abdominal nodes (those adjacent to the celiac axis, and splenic, left gastric, and common hepatic arteries). An overall disease-free survival of 40% was achieved at our center in esophageal cancer patients resected with a combined *en bloc*, two-field lymphadenectomy (Fig. 4).

A "three-field lymphadenectomy" extends the lymph node dissection to include the lymph nodes within superior mediastinum, located along the course of the left and right recurrent laryngeal nerves. The rationale for extension of the lymph node dissection is based on the finding that nearly one-third of patients with presumably localized esophageal cancer have occult metastases to these nodes. Recent reports both in our center and in Japan have confirmed this finding, particularly in patients with adenocarcinoma of the esophagus. In addition, we have shown that the procedure may be conducted with a mortality and morbidity comparable to the "two-field" lymphadenectomy. Significantly, our long-term survival with this approach demonstrates a significant survival advantage over the standard esophagectomy and two-field lymphadenectomy (27,28). Unfortunately, lack of familiarity with this approach has limited its performance to a few specialized centers in Japan and the United States.

For those patients who are not candidates for curative esophagectomy, other options for palliation may be offered. Primary chemoradiation has been shown to produce 5-yr survival rates as high as 10%, and should be considered for the majority of patients whose cancer is unresectable. Esophageal dilatation offers short-term palliation, although the risk of esophageal perforation is not insignificant. Stenting or laser fulguration may also offer symptomatic relief in patients with a limited life expectancy. It should be emphasized that although esophagectomy offers excellent palliation of symptoms, patients should not be offered surgery without curative intent.

OPTIONS FOR ESOPHAGEAL RECONSTRUCTION

Restoration of continuity of the GI tract is most commonly performed with a portion of tubularized stomach. However, other options for reconstruction are available to the esophageal surgeon. For instance, *colonic interposition* may be offered to patients undergoing esophagectomy for benign disease. Interposition of colon offers several potential benefits: an organ with potentially functional peristalsis and an epithelium



Fig. 4. Overall survival of patients treated with an *en bloc* esophagectomy at Weill-Cornell Medical Center.

relatively impervious to acid reflux, a conduit of nearly unlimited length, and the ability to place the conduit in a location other than the posterior mediastinum. In addition, the vascular supply to the colon is abundant and well described. For malignant disease, the gastric pull-up is the preferred method for reconstruction. The use of stomach is technically straightforward and requires only one anastomosis. However, in situations in which prior gastric surgery has rendered the stomach unsuitable, colon interposition is an acceptable alternative. Some centers routinely use colon interposition for reconstruction after esophagectomy for benign disease. This practice is based on the belief that the development of anastomotic stricture and acid reflux may be less after colon interposition. No long-term studies have demonstrated the superiority of colon interposition over gastric pull-up. Furthermore, the necessity of additional abdominal surgery and a second anastomosis increases the complexity of an already demanding operation. Nonetheless, several large series have demonstrated the safety of this procedure in experienced hands (29,30).

Colonoscopy is required for preoperative evaluation of patients undergoing colonic interposition. Occasionally, the findings of polyps or occult malignancy will preclude the use of colon. Although angiography had once been considered mandatory, it is currently reserved for patients with significant vascular disease or those with a history of prior colonic surgery. Although either the left or right colon may be used for reconstruction, the left colon is by far the better alternative for several reasons. First, the smaller diameter of the left colon provides for a technically easier anastomosis to the proximal esophagus. Also, the blood supply to the left colon is less variable than that of the right colon. Finally, the left colon may be placed in the thorax in an isoperistaltic direction.

To perform a left colon interposition, the descending and transverse colon are mobilized. This may be performed through either a laparotomy or an incision in the diaphragm if a left thoracotomy has already been performed. The vascular supply to the left colon is identified including the marginal artery of Drummond, the left and right branches of the middle colic artery and the ascending and descending branches of the left colic artery. Adequate blood supply is determined by transillumination of the mesentery and palpation of a pulse. Once the appropriate length of conduit has been determined, temporary vascular clamps are placed on the vessels to be ligated. The viability of the bowel is then reassessed by visual examination. On occasion, intravenous fluoroscein may be useful if the viability of the conduit is in question. For long segment interposition, the vascular supply is based on the left colic artery. The colon is then divided distal to the splenic flexure distally and at the mid-transverse colon proximally. If additional length is required, the colon may be transected near the hepatic flexure. The colon is then mobilized through the lesser sac behind the stomach and brought into the chest through the esophageal hiatus. Anastamoses are then constructed to the proximal stomach and posterior wall of the stomach (Fig. 3B). Great care must be exercised to ensure that the vascular pedicle is not disrupted during mobilization to the chest. Graft ischemia may readily occur if the anastomosis is placed under tension or if the pedicle is rotated. Venous drainage from the colon is as important as arterial supply and may be easily compromised if the pedicle has been rotated.

Interposition of jejunum may also be considered for short segment replacement of the esophagus. The variable blood supply to the jejunum mandates careful evaluation of the intestine prior to transfer. Congenital interruptions in the vascular arcade occur frequently and must be excluded before a segment of jejunum can be considered suitable. The dissection is usually begun at least 20 cm distal to the ligament of Treitz, at which point the vascular branches are longer and an appropriate pedicle may be identified more easily. Free jejunal transfer with construction of a microvascular anastomosis to the common carotid artery has been described for replacement of a short segment of the cervical esophagus (30).

MANAGEMENT OF COMPLICATIONS

Even in the most experienced hands, an esophagectomy is a complex procedure that carries a consistent mortality of 5% and a complication rate of 40%. Complications common to all lengthy operations, such as cardiac arrythmias, myocardial infarction, and pneumonia are frequent. However, several complications are unique to esophagectomy. An esophageal leak carries the highest mortality rate of any complication. An asymptomatic leak that is detected on a routine barium swallow and appears to drain back into the esophageal lumen will usually heal without intervention. However, larger, uncontained leaks require adequate drainage either by an interventional radiology catheter, chest tube, or open drainage. Signs of sepsis will appear in conjunction with a leak that is not adequately drained and indicate that thoracotomy with drainage of the chest and decortication of the lung will be required. Endoscopy is useful to determine the viability of the stomach and size of the leak. Small, well-drained leaks will often heal if the lung is well expanded and there is no local sepsis. However, if there is extensive necrosis, often the safest plan is resection of the conduit and creation of a cervical esophagostomy. Graft necrosis, caused by infarction of the gastric tube, is a very rare complication that may be fatal.

Other complications may not be apparent for several months postoperatively. An anastomotic stricture is often related to a prior leak or vascular insufficiency at the tip

of the gastric tube. Fortunately, the majority of patients respond well to periodic esophageal dilatation, and this is rarely required beyond the first postoperative year. Delayed gastric emptying is an uncommon complication that can usually be managed conservatively. Common causes of delayed gastric emptying include the lack of a pyloric drainage procedure, obstruction at a tight hiatus or a redundant intrathoracic stomach. Repeated endoscopy and balloon dilatation of the pylorus in conjunction with promotility agents such as metoclopramide and erythromycin are usually sufficient. Finally, reflux is a common problem after a gastric pull-up. It appears that the level of severity will vary inversely with the level of the anastomosis. Anastomoses above the azygous vein have a lower incidence of reflux than those below the vein. Symptoms of reflux are improved by smaller, more frequent feedings, avoidance of liquids with meals, and avoidance of recumbency after meals.

COST OF SURGERY AND FUNCTIONAL OUTCOME

As measured by both economic and psychological parameters, the cost of esophagectomy is high. Currently, an uncomplicated esophagectomy will require several hours of operating room time, and an average of 8 d spent in the hospital. The average cost incurred at our institution for this level of care is approx \$30,000–\$50,000. However, this figure may be easily doubled if complications ensue.

Few long-term studies on functional outcome following esophagectomy have been performed. In a longitudinal study evaluating more than 100 patients undergoing esophagectomy, more than 60% of patients experienced some form of gastroesophageal reflux and 25% of patients noted some degree of dysphagia. Despite this, the ability to work, perception of health, and resumption of daily activities were no different at long-term follow-up than the national norm (31). Although both physicians and patients must be aware that esophagectomy is a major undertaking, it may be performed safely and can provide excellent treatment for several disorders of the esophagus, as well as acceptable long-term quality of life.

SUMMARY

- 1. Esophagectomy is a formidable operation with a consistent mortality rate of 5% and morbidity rate of 40% whether it is done through the diaphragmatic hiatus with or without a thoracotomy.
- 2. The most common indication for this operation is potentially curable esophageal cancer, and the most common way that gastrointestinal continuity is restored is by pulling up a tube constructed out of the stomach. However, a segment of colon can be used if the stomach is not available, or the patient has benign disease and an expected long-term survival.
- 3. The most feared and lethal acute complication is a leak from the anastamosis; especially a leak into the chest and mediastinum which carries a 50% mortality.
- 4. Several postoperative complications following esophagectomy need medical therapy. These include strictures, which can be dilated, and gastric emptying problems and reflux symptoms.
- 5. Whereas extending the lymph node dissection during esophagectomy for cancer may increase survival in very experienced centers, it is also clear that esophagectomy is a poor palliative option for obviously incurable esophageal cancer.

REFERENCES

- 1. Dimick JB, Cattaneo SM, Lipsett PA. Hospital volume is related to clinical and economic outcomes of esophageal resection in Maryland. Ann Thorac Surg 2001;72:334–339.
- 2. Office of Research on Cancer Prevention and Treatment of the Ministry of Health: Atlas of Cancer Mortality and of The Peoples' Republic of China. Ministry of Health, China, Beijing, 1980.
- 3. Tuyns AJ, Masse G. Cancer of the esophagus: An incidence study in Ille-et-Vilaine. Int J Epidemiol 1975;4:55–59.
- 4. Bartsch H, Montesano R. Relevance of nitrosamines to human cancer. Carcinogenesis 1984;5:1381–1393.
- 5. Yu MC, Garabrant DH, Peters JM, et al. Tobacco, alcohol, diet, occupation and carcinoma of the esophagus: Cancer Res 1988;48:3843–3848.
- 6. Tuyns AJ, Pequignot G, Jensen OM. Le cancer de l'oesophage en Ille-et-Vilaine en fonction des nivaux de consommation d'alcohol et de tabac: des risques qui se multiplient. Bull Cancer 1977;64:45–60.
- 7. Yang J. Preliminary studies on the etiology and conditions of carcinogenesis of the esophagus in Linxian. In: *Experimental Research on Esophageal Cancer* Yang J Gao J, eds., Beijing, China: Renmin Weishberg, 1980, p. 82.
- 8. Ghavamzadeh A, Moussavi A, Jahani M, et al. Esophageal cancer in Iran. Semin Oncol 2000;28:153–157.
- 9. Hille JJ, Markowitz S, Margolius KA, et al. Human papillomavirus and carcinoma of the esophagus. N Engl J Med 1985;312:1707 (lett).
- 10. Blot WJ, Devesa SS, Kneller RW, et al. Rising incidence of adenocarcinoma of the esophagus and gastric cardia. JAMA 1991;265 (10):1287–1289.
- 11. Cameron AJ, Zinsmeister AR, Ballard DJ, et al. Prevalence of columnar-lined (Barrett's) esophagus. Gastroenterol 1990;99:918–922.
- 12. Hamilton SR, Smith RR, Cameron JL. Prevalence and characteristics of Barrett's esophagus in patients with adenocarcinoma of the esophagus or the esophagogastric junction. Human Pathol 1988;19:942–948.
- 13. DeMeester TR, Attwood SE, Smyrk TC, et al. Surgical therapy in Barrett's esophagus. Ann Surg 1990;212:528–540.
- 14. Meijssen MA, Tilanus HW, van Blankenstein M, et al. Achalasia complicated by oesophageal squamous cell carcinoma: A prospective study in 195 patients. Gut 1992;33:155–158.
- 15. Marmuse JP, Maillochaud JH. Respiratory morbidity and mortality following transhiatal esophagectomy in patients with severe chronic obstructive pulmonary disease. Ann Chir 1999;53:23–28.
- Lightdale CJ. Staging of esophageal cancer: I. Endoscopic ultrasonography. Semin Oncol 1994;21:438–446.
- 17. Krasna MJ. Role of thoracoscopic lymph node staging for lung and esophageal cancer. Oncology 1996;10:793–802.
- 18. Fok M, Sham JS, Choy D. Postoperative radiotherapy for carcinoma of the esophagus: A prospective, randomized controlled study. Surgery 1993;113:138–147.
- Schlag PM. Randomized trial of preoperative chemotherapy for squamous cell cancer of the esophagus. Arch Surg 1992;127:1446–1450.
- Schlag P, Herrmann R, Raeth V, et al. Preoperative chemotherapy in esophageal cancer: A phase II study. Acta Oncol 1988;27:811–814.
- Kelsen DP. Chemotherapy followed by operation versus operation alone in the treatment of patients with localized esophageal cancer: A preliminary report of intergroup study 113 (RTOG 89-11) (abstract). Meeting of the American Society of Clinical Oncology (ASCO) 1997.
- 22. Urba S. A randomized trial comparing surgery to preoperative concomitant chemoradiation plus surgery in patients with resectable esophageal cancer: Update analysis. Proc Am Soc Clin Oncol 1997;6:227.
- 23. Bosset JF. (1994) Randomized phase III clinical trials comparing surgery alone versus pre-operative combined radiochemotherapy (XRT-CT) in stage I-II epidermoid cancer of the esophagus. Preliminary analysis: A study of the FFCD (French group) no. 8805 and EORTC no. 40881. Proc Am Soc Clin Oncol 1994;13:197.
- 24. Walsh TN, Noonan N, Hollywood D. A comparison of multimodal therapy and surgery for esophageal adenocarcinoma. N Engl J Med 1996;35:462.
- 25. Chu KM, Law SY, Fok M, et al. A prospective randomized comparison of transhiatal and transthoracic resection for lower-third esophageal carcinoma. Am J Surg 1997;174:320.

- 26. Horstmann O, Verreet PR, Becker H, et al. Transhiatal esophgaectomy compared with transhoracic resection and systematic lymphadenectomy for the treatment of esophageal cancer. Eur J Surg 1995;161:557.
- 27. Isono K, Sato H, Nakayama K. Results of a nationwide study on the three-field lymph node dissection of esophageal cancer. Oncology 1991;48:411.
- 28. Altorki NA, Skinner D. Should en bloc esophagectomy be the standard of care for esophageal carcinoma? Ann Surg 2002;254:581.
- 29. Young M, Deschamps C, Trastek V, et al. Esophageal reconstruction for benign disease: Early morbidity, mortality and functional results. Ann Thorac Surg 2000;70:1651.
- 30. Mansour K, Bryan C, Carlson G. Bowel interposition for esophageal replacement: twenty-five-year experience. Ann Thorac Surg 1997;64:752.
- 31. McLarty A, Deschamps C, Trastek V, et al. Esophageal Resection for Cancer of the Esophagus: Long-Term Function and Quality of Life. Ann Thorac Surg: 1997;63:1568.

Zenker's Diverticulum

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INTRODUCTION

The Zenker's diverticulum is an out pouching of the hypopharynx arising between the fibers of the cricopharyngeus inferiorly and the inferior constrictor superiorly. This region of herniation is known as Killian's triangle. Patients often present with a longstanding history of gradually increasing dysphagia of both solids and liquids. Regurgitation of undigested food hours after a meal is a classic presentation. In addition, patients often complain of hoarseness, choking episodes, halitosis, and in severe cases, may have significant weight loss to the point of cachexia. Patients may also present with recurrent pneumonia.

Friederich von Zenker described the diverticulum and assigned his name in 1877 (1). The pathophysiology of the Zenker's diverticulum is thought to be chronic spasm or stricture of the cricopharyngeus muscle. Distal obstruction of the hypopharynx gradually causes proximal dilatation and eventual herniation. As time progresses, the herniation becomes large enough to produce a false passage to a blind sac (Fig. 1A,B). The same spasm or stricture that caused the initial herniation tends to divert ingested boluses into the sac and prevent transit into the esophagus (2).

There has been controversy over the years regarding the surgical treatment of this condition. Opinions have differed regarding the need for excision of the pouch and/or lysis of the cricopharyngeus muscle. Lysis of the muscle has been determined as the essential step in the treatment of the disorder and has prompted several treatment options ranging from chemo-denervation of the muscle to surgical lysis via either endoscopic or open approach.

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Fig. 1. (A) Normal anatomy. (B) Anatomical relationships of Zenker's diverticulum.

EVALUATION

Dysphagia is the presenting symptom for a large number of ailments of the upper aerodigestive tract. Diligent history taking and examination are required to elicit the correct diagnosis. Tumors of the hypopharynx, larynx, and esophagus may present with a similar spectrum of symptoms. Careful history-taking regarding the exact nature of symptoms, associated symptoms, comorbid conditions, and risk factors for carcinoma are vital. A thorough examination including indirect visualization of the oropharynx, hypopharynx, and larynx is needed to evaluate anatomy, as well as pathology. Pooling of secretions may be noted in the postcricoid region. A subtle fullness of the neck may be appreciated on palpation.

If no pathology is noted on physical exam, a barium esophagram is usually extremely helpful in determining the degree and area of obstruction. With Zenker's diverticulum there is often a blind pouch that fills with contrast (Fig. 2). Often there are filling defects within the pouch, which correlate with retained food particles. There is often a "cricopharyngeal bar" seen on the lateral view of the swallow, which is present as a result of persistent spasm of the cricopharyngeus (CP). Contrast will pass through the spasm and into the esophagus in variable amounts. One must be vigilant for other causes of obstruction and look for irregularities of the mucosa and filling defects. Computed tomography (CT) scan with contrast can help to rule out other causes of obstruction and can demonstrate the Zenker's as an air-filled sac.

TREATMENT

Pharmacological treatment of the CP muscle is now available for patients with significant CP spasm. Botulinum toxin, which when injected locally prevents release of acetylcholine from muscle nerve endings, has been successfully used to treat dystonia of the neck, face, and larynx. Injection into the CP muscle via transcutaneous route (done in an office setting) utilizing electromyogram (EMG) guidance or via direct esophagoscopy



Fig. 2. A barium swallow showing a Zenker's diverticulum filled with contrast.

in the operating room (OR) can provide temporary relief of CP spasm (3,4). The procedure is well tolerated and has a low complication rate, which can include recurrent laryngeal nerve paresis, infection, and local bleeding, all of which are usually minor and self-limited. Botulinum injection, if successful, will usually sustain an effect for a 4–14mo period. Reinjection is then necessary when symptoms recur. If the diverticular sac is large, treatment of the muscle alone may not be adequate to relieve the symptoms and the sac itself may need to be addressed either by suspension of excision.

Open surgical management of a Zenker's diverticulum is directed toward elimination of symptoms by transecting the stenotic cricopharyngeus muscle. Variations on the procedure include CP myotomy alone, CP myotomy with resection of the sac, or suspension of the sac. Elderly patients with significant comorbidities who are poor surgical candidates may be able to get relief from the symptoms with cricopharyngeal lysis alone. Some authors have recommended lysis of the CP muscle with suspension of the sac without excision (no mucosal incision). The open procedure allows for excellent visualization of the pathology and lysis of the CP muscle. The procedure does require an incision, can be time-consuming, and often requires retraction on the great vessels of the neck. Tension of the recurrent laryngeal nerve can cause vocal fold dysfunction, which can be permanent. Patients can also develop wound infections, hematomas, esophageal fistulae, and leaks at the site of the sac excision (5). Drains are typically placed postoperatively and removed when drainage is minimal. Barium swallow is often carried out prior to feeding the patient to assure the wound has closed.

In an attempt to decrease morbidity of treatment, as well as decrease operative and recovery time, direct endoscopic visualization and lysis of the cricopharyngeus was explored. Mosher first described endoscopic treatment of Zenker's diverticulum in 1917, but the first large series describing outcomes was put forth by Dohlman and Mattson (6). The procedure is now often referred to as the Dohlman procedure. The procedure has the advantages of no external incisions, generally shorter OR time, as well as generally shorter recovery time. The procedure does require general anesthesia and does have its own set of complications associated with it. Results of the Dohlman procedure in his series were excellent. They reported 90% improvement, and only a 7% incidence of residual sac. In this series, the esophagus and the party wall were divided by electrocautery. Modifications to this procedure have included section of the party wall with lasers, as well as a technique using a stapling device similar to that used in lung resections (7). Use of the stapler has the advantage of sealing the cut mucosa. Patients tend to recover more quickly and often can start a liquid diet on the day of surgery. Patients can be discharged home the next day if the postoperative course is uneventful.

Success of the endoscopic procedure is largely reliant on adequate visualization and access to the involved structure. The procedure is done through the open mouth and the patient's anatomy must be amenable to this type of exposure to ensure a successful outcome. Adequate visualization can be limited by patient anatomy including presence of teeth, a large neck, macroglossia, an anteriorly situated larynx, and redundant hypopharyngeal tissue. If adequate visualization is not possible, an attempt at endoscopic repair should be aborted and the open procedure performed. Preoperative counseling and informed consent should reflect this algorithm. The incidence of complications with the procedure increases significantly if visualization is difficult. Patients with cervical spine disorders or TMJ joint problems may not be suitable for the endoscopic approach.

PROCEDURE

A bivalve laryngoscope or specially designed upper esophagoscope is placed into the oral cavity and gently advanced into the oropharynx. Once the postcricoid region is in view, the scope is suspended. The jaws of the scope are then opened with the anterior part of the scope in the proximal esophagus and the posterior part of the scope in the diverticulum. This exposes the party wall. Once the true and false lumens have been sufficiently opened, the party wall must be secured and retracted toward the surgeon to allow for proper placement and firing of the stapler. This is usually accomplished by endoscopically passing one or two retracting sutures with an endoscopic needle passer. Once this accomplished, the stapler is carefully passed through the laryngoscope so that one jaw sits in the true lumen and one in the false lumen. When the location is confirmed, the stapler is fired in the standard fashion and then withdrawn. The resulting wound is then carefully examined and inspected to see that the staple lines are intact. Repeat stapling is sometimes required for larger diverticula. The distal end of the jaws of the stapler do not cut or staple and, as a result, the distal-most sac is often intact. This does not seem to cause a problem as long as 1 cm or less remains. Some surgeons

advocate lysis of the distal-most sac with bovie or laser following stapling. Once adequate lysis of the party wall has taken place, the stapler and then the scope are removed. Patients are watched carefully postoperatively and broad-spectrum antibiotics are continued. The patient is maintained on iv fluids and is kept strictly NPO. Particular attention is paid to temperature, respiratory rate, and pulse. The neck and superior chest are carefully monitored for erythema or tenderness, which could suggest a leak. Any of the above signs or symptoms warrants aggressive management with imaging studies to rule out a leak and appropriate management of a leak if it is found. If the postoperative period is uneventful, the patient is started on a liquid diet postoperative day 2 or 3. The diet is usually advanced as tolerated and the patient discharged shortly thereafter if a diet is tolerated. Some surgeons obtain a barium swallow prior to initiating oral intake regardless of postoperative course. Patients should be treated for reflux with a proton pump inhibitor as acid reflux onto freshly cut tissues may result in excessive scar formation (7).

COMPLICATIONS

Acute minor complications can include damage to teeth or alveolar ridge, scrapes of the oral mucosa, and pressure on the tongue causing transient pain or numbness. These problems usually resolve with conservative management and observation. An avulsed tooth may necessitate a dental consult. Recurrent laryngeal nerve dysfunction has been reported and is likely as result of pressure from the laryngoscope (8).

More severe complications include lacerations of the pharyngeal mucosa by the scope, mediastinitis from a leak at the transection site, and anesthesia-related morbidity and mortality. A large perforation of the pharynx may be noted intraoperatively and may require conversion to an open procedure if there is concern of a significant leak. A leak resulting from the procedure may not be suspected until many hours postoperatively. Patients may complain of increasing neck pain, odynophagia, and chest pain. Temperature curves will trend upward and erythema may be noted on the neck and superior chest. A barium swallow may show extravasation of contrast from the pharyngeal lumen into the mediastinum. CT scan may be needed for diagnosis and to fully assess extent of spread. If a collection is seen in the mediastinum, it must be drained either via open techniques or with the assistance of interventional radiology. The patient should be kept NPO and broad-spectrum antibiotics maintained. A feeding tube may need to be passed under fluoroscopic guidance to feed the patient. The mortality of this complication has been reported to be as high as 30% (9). Patients who have this complication may have persistent morbidity as a result of intense scarring including prolonged severe dysphagia requiring long-term nutritional support by feeding tube.

Chronic complications are rare. Recurrence of the diverticulum has been reported. This is thought to be caused by incomplete lysis of the pathologic cricopharyngeus muscle. Direct visualization of the muscle is not possible with the endoscopic approach and cricopharyngeal fibers may be preserved. This may lead to eventual relapse (10). Postoperative barium swallows have shown small residual pouches following the endoscopic procedure even in asymptomatic patients. Other long-term complications are exceedingly rare.

Long-term follow-up of patients undergoing the Dohlman procedure have been very promising. The majority of patients is satisfied with the result and can resume a nearly normal diet. Cook et al. reviewed a series of 74 patients. Sixty-eight of these patients
underwent endoscopic repair. Of these patients, 74% reported complete resolution of symptoms and 96% reported improvement. Average hospital stay was 1.3 d with only two patients staying in the hospital more than 1 d.

COST

The cost for the excision of a diverticulum is approx \$1500 (surgeon's fee) and the cost for Botox injection including esophagoscopy is approx \$1000.

SUMMARY

- 1. Zenker's diverticulum is an uncommon condition caused by out pouching of hypopharynx between cricopharnygeus muscle and inferior constrictor.
- 2. The exact etiology is unknown, but is thought to be caused by spasm or stricture of the cricopharyngeus muscle.
- 3. Several treatment options are available in the symptomatic patients. These include pharmacological therapy with botulinum toxin injection either transcutaneously or via esophagoscopy, endoscopic therapy, or open cricopharyngeus myotomy with or without resection of the hernia sac.
- 4. Surgical therapy is highly successful with very few immediate or late complications.

REFERENCES

- 1. Zenker FA, von Ziemessen H. Krankheiten des oesophagus. In: *Handbuch der specciellen Pathologie und Therapie*. (Ziemessen H, ed.), Leipzig: FC Vogel, 1877, p. 187.
- McConnell FMS, Hood D, Jackson K, et al. Analysis of intrabolus forces in patients with Zenkers diverticulum. Laryngoscope 1994;104:571–581.
- 3. Blitzer A, Brin MF. Use of botulinum toxin for diagnosis and management of cricopharyngeal achalasia. Otolaryn. Head and Neck Surg 1997;116:328–330.
- 4. Schneider I, Thumfart WF, Pototschnig C, et al. Treatment of dysfunction of the cricopharyngeal muscle with botulinum a toxin: introduction of a new, non invasive method. Ann Otol Rhino Laryng 1994;103:31–35.
- 5. Aggerholm K, Illum P. Surgical treatment of Zenkers diverticulum. J Laryngol Otol 1990;104:312-314.
- 6. Dohlman G, Mattsson O. The endoscopic operation for hypopharyngeal diverticula. Arch Otolaryngol 1960;71:744–752.
- 7. Cook C, Huang P, Richstmeier W, et al. Endoscopic staple assisted esophagodiverticulostomy for Zenker's diverticulum. Laryngoscope 2000;110:2020–2025.
- 8. Scher R, Richtsmeier W. Long-term experience with endoscopic staple assisted esophagodiverticulostomy for Zenkers diverticulum. Laryngoscope 1998;108:200–205.
- 9. Papalia E, Rena O, Oliaro A, et al. Descending necrotizing mediastinitis: surgical management. Eur J Cardiothoracic Surg 2001;4:739–742.
- 10. Welch AR, Stafford F. Comparison of endoscopic diathermy and resection in the surgical management of pharyngeal diverticula. J Laryngol Otol 1985;99:179–182.

Esophagectomy for Achalasia

Laparoscopic Heller Myotomy and Dor Fundoplication

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INTRODUCTION

At length the Disease having overcome all remedies, he was brought into that condition, that growing hungry he would eat until Oesophagus was filled up to the Throat, in the mean time nothing sliding down into the Ventricle, he cast up raw (or crude) whatsoever he had taken in: when that no Medicines could help and he languished away for hunger, and every Day was in Danger of Death. I prepared an instrument for him like a Rod, of a whale Bone, with a little round Button of Sponge fixed to the top

From: Clinical Gastroenterology: An Internist's Illustrated Guide to Gastrointestinal Surgery Edited by: George Y. Wu, Khalid Aziz, and Giles F. Whalen © Humana Press Inc., Totowa, NJ of it; the sick Man having taken down meat and drink into his Throat, presently putting this down in the Oesophagus, he did thrust down into the Ventricle, its Orifice being opened, the Food which otherwise would have come back again... (1).

This observation made by Thomas Willis in 1674 was the first description of a clinical entity that would later be coined "achalasia" by Sir Arthur Hurst in 1913. Translated from the Greek, achalasia means, "lack of relaxation" and today refers to a disease of the esophagus in which the lower esophageal sphincter fails to relax in the setting of a dilated, aperistaltic, esophageal body. In 1913, Earnest Heller performed the first esophagomyotomy. The Heller myotomy, with its subsequent modifications, has become the gold standard for the treatment of achalasia. This chapter will examine the pathophysiology of achalasia, key elements in the diagnostic assessment, the medical treatment options, and a review of the surgical therapy for achalasia.

EPIDEMIOLOGY

Achalasia affects patients of all age groups. Mean ages range between 30 and 60 years of age, with a peak incidence in the 40s. It is uncommon during the first two decades of life and has an incidence of 0.4 to 0.6 per 100,000 with a prevalence of 8-13 persons per 100,000 population (2).

PATHOPHYSIOLOGY

Achalasia is characterized by a hypertensive, nonrelaxing lower esophageal sphincter and a dilated, aperistaltic esophageal body. Pathologically, the esophagus demonstrates only minimal dilation early in the course of the disease course but later can become as large as 16 cm. Histologically, the major abnormality is the loss of ganglion cells in the myenteric plexus of the distal esophagus. Several other neuropathic lesions are also observed. These include: a) inflammation or fibrosis of the myenteric plexus early in the disease course; b) decrease in varicose nerve fibers of myenteric plexus; c) degeneration of the vagus nerves; d) changes in the dorsal motor nucleus of the vagus; e) decreases in the number and histology of small intramuscular nerve fibers; and f) occasional intracytoplasmic inclusions in the dorsal motor nucleus of the vagus and myenteric plexus. It is unknown where the initial neurological injury occurs (2).

ETIOLOGY

Three basic theories regarding the etiology of achalasia exist: familial, autoimmune, and infectious. Less than 1% of cases of achalasia are familial, displaying an autosomal recessive inheritance pattern. Many of the familial cases are associated with consanguineous union. The presence of T cells in the ganglion cells of the esophagus suggests an autoimmune etiology to the disease. There is an association between achalasia and class II histocompatibility antigen Dqwl. The similarity between achalasia and Chagas' disease caused by Trypanosoma cruzi suggests an infectious etiology. Furthermore, there is an increased incidence of varicella-zoster virus (VZV) antibodies in the serum of patients with achalasia as well as the presence of VZV by *in situ* DNA hybridization in tissue removed at esophagomyotomy (2).

CLINICAL FEATURES

The presentation of achalasia depends upon the duration of the disease process. Most patients are between 20 and 40 years of age with a ratio of men to women of 2:1. Solid food dysphagia is the most common presenting symptom. Patients describe fullness of the chest during a meal and a "sticking" in the lower substernal area. Early in the disease process, the sensation is intermittent but invariably becomes constant. Food sometimes passes easier when it is warm and the amount of dysphagia can vary daily. Various maneuvers appear to aid in the passage of food. These include: a) a head back position in the upright position associated with a Valsalva maneuver; b) drinking carbonated beverages; c) belching; d) drinking alcoholic or warmed beverages; e) and smoking marijuana.

Regurgitation is the second most common complaint and occurs in approx 70% of cases. The regurgitated food is described as undigested, nonbilious and nonacidic, and frequently awakens the patient from sleep (1).

Other symptoms include chest pain and heartburn occurring in approx 40% of patients. The pain is described as substernal or epigastric, radiating to the neck, arms, jaws, and back. Depending of the severity of the symptoms, weight loss is a common feature. Displacement of mediastinal structures, esophageal ulcerations and perforation, and aspiration of esophageal contents may also occur (1).

PATIENT EVALUATION

The evaluation of patients with achalasia involves three basic studies: the barium swallow, upper endoscopy, and esophageal manometry.

The diagnosis of achalasia is often first considered with a barium swallow (Fig. 1), which classically demonstrates a dilated esophagus and a distal "bird's-beak" narrowing. This finding, present in 90% of cases, may not be present early in the disease course. Videofluoroscopy can improve the sensitivity of this study by noting abnormal or absent esophageal contractions.

Endoscopy should be performed in all patients with achalasia, especially those who have risk factors for cancer including a greater than 20-lb. weight loss and age greater than 60 yr. A malignancy of the gastroesophageal junction may present with symptoms mimicking achalasia, thus described as pseudoachalasia.

Esophageal manometry is the definitive test for achalasia. Patients with achalasia demonstrate poor relaxation of lower esophageal sphincter on swallowing, lack of peristalsis in the distal esophagus, simultaneous, low-amplitude, single-peaked, widened peristaltic contractions, and a positive gastroesophageal pressure gradient.

Computed tomography (CT) scan of the chest, 24-h pH study, and nuclear scintigraphy are occasionally utilized. A CT scan of the chest and upper abdomen may reveal an extrinsic mass or other cause of a pseudoachalasia. The 24-h pH study is used to diagnose gastroesophageal reflux disease, which is uncommon among patients with achalasia unless they have received prior dilation or surgical intervention. Esophageal transit studies using nuclear scintigraphy can be used to assess esophageal motility. This test is used to assess esophageal emptying after myotomy or dilation.



Fig. 1. Barium swallow demonstrating "bird-beak" narrowing of esophagus typical of achalasia.

TREATMENT OPTIONS

It is impossible to restore normal peristaltic function of esophagus. The treatment of achalasia focuses on relieving the distal esophageal obstruction at the lower esophageal sphincter (LES). The most common methods include balloon dilation, botulinum toxin (Botox) injection, and Heller myotomy.

Pneumatic balloon dilation is performed endoscopically with intravenous (iv) sedation. The muscle fibers of the distal esophagus are disrupted without causing perforation of the mucosa. A volume-limited, pressure-controlled (Gruntzig-type) catheter is placed across the gastroesophageal junction. The esophagus is then forcefully dilated to a pressure of 300 Torr for 15 s. A contrast swallow is performed immediately following to confirm the absence of a perforation

Most people report some symptomatic relief from pneumatic dilation. Approximately 60% of patients have relief of dysphagia and an additional 10% respond to a second dilation. There is recurrence of dysphagia over time in 10% to 70% of patients requiring redilation. The incidence of esophageal perforation following dilation is approx 4% with a mortality of 0.5%. Gastroesophageal reflux occurs in 20 to 40% of patients (3).

Intrasphincteric injection of the LES with Botox through the flexible endoscope represents a newer modality for treating achalasia. The toxin blocks release of acetylcholine from the presynaptic parasympathetic nerve endings in the smooth muscle producing a denervation of the LES. The immediate results are excellent, with 70% to 100% of patients experiencing relief within the first month. However, favorable results are reported by 60% of patients by 6 mo, and by only 3 to 36% of patients at 1 yr. Repeat treatments offer transient improvement, but beyond 6 mo, the results are negligible. Although there are relatively few immediate complications with Botox injection, these injections induce scarring and inflammation around the esophagus, making subsequent surgical intervention more difficult (4).

SURGICAL MANAGEMENT

Heller esophagomyotomy is the optimal treatment of achalasia. This procedure allows for the precise division of the longitudinal and circular muscles of the lower esophagus, thus relieving the functional obstruction of distal esophagus. Although the Heller myotomy was first performed transthoracically, the development of video-assisted minimally invasive techniques has led to the development of a laparoscopic approach that is equally effective but with minimal morbidity.

INDICATIONS

All patients who can tolerate general anesthesia and laparoscopy should be candidates for surgery. In particular, patients under 40 yr of age have worse results with pneumatic dilation, whereas Heller myotomy offers a 90% long-term success rate (5). Patients who have failed other forms of therapy such as Botox injection or pneumatic dilation are surgical candidates. These patients may have scarring in the distal esophagus increasing the difficulty of the myotomy and increasing the mucosal perforation rate, but they have equivalent outcomes with little additional morbidity (6).

CONTRAINDICATIONS

The surgery is contraindicated in patients with severe cardiopulmonary disease or other morbidities that will put them at a higher risk for general anesthesia. These patients may be treated with dilation or Botox injection. Patients with overwhelming cardiopulmonary risk may be treated with percutaneous endoscopically placed gastrostomy tube for alimentation.

SURGICAL TECHNIQUE

The traditional approach to Heller myotomy is through a left thoracotomy in the seventh intercostal space. The distal esophagus and proximal stomach are mobilized. The longitudinal and circular muscles of the esophagus are incised from the inferior pulmonary vein across the gastroesophageal junction completing the myotomy a variable distance onto the stomach. The muscle is dissected away from the mucosa allowing the strong mucosal layer to protrude. A longer myotomy allows complete disruption of the lower esophageal sphincter, relieving dysphagia but increasing the risk of reflux. To optimize results, many surgeons add a partial fundoplication to a long myotomy. The chest is closed with placement of chest tubes. Patients are hospitalized for 4–7 d.

Laparoscopic Heller myotomy is the optimal procedure performed today, with excellent results and minimal morbidity. The procedure should be performed by surgeons with advanced laparoscopic skills who have experience with this relatively unusual



Fig. 2. Schematic of Heller esophagomyotomy. Longitudinal and circular esophageal muscles are divided from distal esophagus and incision extended to proximal part of stomach and mucosal layer is exposed.



Fig. 3. Partial fundoplication after myotomy.

disease. The surgery is performed under general anesthesia. Five laparoscopic trocars are placed. The peritoneum overlying the distal esophagus is divided and the anterior esophagus is exposed after inducing pneumoperitoneum. The anterior vagus nerve is identified and protected. With laparoscopic magnification, the longitudinal and circular muscles are carefully divided, exposing the mucosal layer (Fig. 2). The myotomy is now extended proximally 6 cm from the G–E junction and distally 1 cm onto the proximal stomach. The muscle is



Fig. 4. Postoperative barium swallow.

dissected from the mucosa allowing the mucosa to protrude. Intraoperative flexible endoscopy is then performed to be certain there is no further distal obstruction. The myotomy can be easily extended if necessary until the lower esophageal sphincter is ablated. Air is insufflated into the esophagus and the distended mucosa is assessed for evidence of perforation.

Once the myotomy is completed, an antireflux procedure is added. A 360° fundoplication (Nissen) will cause dysphasia. Therefore, a partial fundoplication is added. Some surgeons completely mobilize the G–E junction and perform a posterior 270° partial Toupet fundoplication. We favor an anterior 180° Dor fundoplication that protects against reflux, yet does not require disruption of all the phrenoesophageal attachments (Fig. 3). In the Dor fundoplication, the proximal fundus is sutured to the hiatus and the divided esophageal musculature (Fig. 4).

The instruments and trocars are removed. The 0.5-cm to 1-cm incisions are closed with absorbable sutures and Band-Aids[®]. Nasogastric tubes are not necessary. The patient begins a liquid diet that evening and is discharged the following day. Dysphagia is immediately improved. The postoperative pain, recovery, and return to work are similar to that seen in elective laparoscopic cholecystectomy.

COMPLICATIONS

Complications are uncommon with this procedure. Mucosal perforation occurs in approx 4.5% of cases. If identified at the time of surgery, it is easily managed with simple repair of the mucosa. Death is extremely uncommon, reported at 0.1%. Early complications occur in approx 5% of cases and include pneumonia, deep venous thrombosis, urinary tract infection, paraesophageal hernia, subphrenic abscess, pleural effusion, esophageal ulcer, and peptic ulcer.

Gastroesophageal reflux can occur after Heller myotomy. Pathologic reflux can be subclinical in 50% of patients, but it can be shown on ambulatory pH testing. When myotomy is performed without an antireflux procedure, reflux occurs in at least 25% of patients, but it occurs in less than 10% of patients who have a concurrent antireflux procedure. Reflux should be treated even if subclinical with acid-suppressive therapy to avoid peptic ulceration and stricture.

Recurrent obstruction may occur as a result of several causes. The patient may have had an inadequate myotomy or a fundoplication causing obstruction. The patient may develop a peptic stricture if subclinical reflux occurs. The nature of the obstruction can be investigated with barium swallow. Forceful dilation or reoperation may improve these patients. In a few cases, esophagectomy may provide definitive management.

COST OF PROCEDURE

The cost for this procedure is approx \$8000. This includes hospital charges for the operating room, one night of hospitalization, and professional fees. There are few studies comparing cost between pneumatic dilation, Botox, and Heller myotomy. These studies are limited by their lack of extended follow-up, absence of quality-of-life assessment; and changes in the hospitalization pattern for pneumatic dilation (fewer overnight admissions). However, for a 5–7-yr period, laparoscopic Heller myotomy is the most expensive option and the pneumatic dilation the least. Botox injection, in these studies, is similar in cost to pneumatic dilation (7).

RESULTS OF HELLER MYOTOMY

Although there are no randomized prospective trials comparing surgical therapy with medical therapy, there is data on the outcome of patients undergoing laparoscopic esophageal myotomy. Several excellent series have been published. Dysphagia was relieved in more than 90% of patients with a follow-up of 2 yr (8). The largest published series of 133 patients by Patti et al. reported excellent results in 90% of patients with a mean follow-up of 28 mo (9).

SUMMARY

- 1. Achalasia is a neurological disease of the esophagus characterized by an aperistaltic body and poor relaxation of LES.
- 2. Dysphagia and regurgitation with eventual weight loss are usual presenting complaints.
- 3. The diagnosis may be made with a barium swallow, but should be followed with upper endoscopy and manometric studies.
- 4. Laparoscopic Heller myotomy with partial fundoplication is the optimal treatment for patients with acceptable surgical risk. Ninety percent of patients report excellent results with this minimally invasive procedure.

5. Pneumatic dilation and botulinum toxin injection are alternatives for patients who have unacceptable surgical risk factors. In patients who are surgical candidates, these nonsurgical interventions should be avoided as first-line therapies because they increase the risk of esophageal perforation if surgery is performed.

REFERENCES

- 1. Ellis FH, Olsen AM. Achalasia of the Esophagus. Major Problems in Clinical Surgery, Volume IX. W.B. Saunders, Philadelphia, 1969.
- Wong KH, Maydonovitch CL. Achalasia. In: *The Esophagus*. (Castell DO, Richter JE, eds.) Lippincott Williams & Williams, Philadelphia, 1999, pp. 185–213.
- Katz PO, Gilbert J, Castell DO. Pneumatic dilation is effective long-term treatment for achalasia. Dig Dis Sci 1998;43:1973–1977.
- 4. Pasricha PJ, Ravich WJ, Hendrix TR, et al. Intrasphicteric botulinum toxin for the treatment of achalasia. N Engl J Med 1995;332:774–778.
- 5. Spiess AE, Kahrilas PJ. Treating achalasia: from whalebone to laparoscope. JAMA 1998;280:638-642.
- 6. Hunter JG, Richardson WS. Surgical management of achalasia. Surg Clin N Am 1997;77:993–1015.
- 7. Richter JE. Comparison and cost analysis of different treatment strategies in achalasia. Gastrointest Endosc Clin N Am 2001;11:359–370.
- Zaninotto G, Costantini M, Molena D, et al. Treatment of esophageal achalasia with laparoscopic Heller myotomy and Dor partial fundoplication: Prospective evaluation of 100 consecutive patients. J Gastrointest Surg 2000;4:282–289.
- 9. Patti MG, Pellgrini CA, Horgan S, et al. Minimally Invasive Surgery for Achalasia: An 8-year experience with 168 patients. Ann Surg 1999;230:587–594.

Surgery for Gastroesophageal Reflux Disease

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INTRODUCTION

Gastroesophageal reflux disease (GERD) is one of the most common problems seen in medical practice. Approximately 10% of the U.S. population experiences heartburn daily, and 40% of the population has heartburn monthly. Seven percent of the population (40 million individuals) use over-the-counter antacids, H-2 receptor antagonists, or proton pump inhibitors at least twice weekly to relieve GERD symptoms. Surgical management of GERD is an effective alternative to medical management of GERD, and it is being more commonly employed (1).

Antireflux surgery was first performed in the 1950s. Diagnostic modalities and technical details evolved during the ensuing 30 yr, yielding superb results from antireflux procedures. However, these procedures, which necessitated thoracotomy or laparotomy, were usually only employed in the most severe cases refractory to medical management. The advent of minimally invasive videoscopic surgery has revolutionized the surgical management of GERD. The transabdominal Nissen fundoplication, which has a greater than 90% effectiveness in treating GERD, became a laparoscopic procedure with equiva-

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lent results to the open Nissen, but with minimal postoperative pain and a rapid return to normal activities. The minimally invasive laparoscopic Nissen fundoplication (LNF) is now increasingly utilized in treating GERD (1,2).

LNF was initially performed only at referral centers. As experience with this procedure has grown, surgeons who perform advanced laparoscopy are routinely performing LNF in community hospitals. As with many laparoscopic procedures, there is a learning curve of 30 to 50 operations. When this curve is surmounted, operative times and complications decrease and long-term successful antireflux repair is achieved (2).

This chapter will discuss the pathophysiology of GERD, treatment options, indications for surgery, necessary preoperative evaluations, a description of LNF, alternative antireflux procedures, and LNF's results, complications, and costs.

PATHOPHYSIOLOGY OF GERD

Gastroesophageal reflux is multifactorial in etiology. The three major determinants of GERD include transient lower esophageal sphincter (LES) relaxation with normal resting LES pressure, anatomical disruption of gastroesophageal junction associated with hiatal hernia, and hypotensive LES. The LES is not a discrete anatomic structure; rather, it is a high-pressure zone that exists because of the anatomic relationships of the distal esophagus, stomach, and diaphragm. The factors that contribute to the LES are as follows: intrinsic esophageal and gastric musculature, relationship of the esophagus to the gastric fundus, and relationship of the distal esophagus to the diaphragm (2,3).

The distal esophageal musculature is contracted in the resting state, but it completely relaxes on swallowing. The orientation of the musculature of the cardia of the stomach contributes to the LES. The relationship of the distal esophagus to the gastric fundus (which compresses the distal esophagus when the stomach is distended) also contributes to this high-pressure zone (4).

The relationship of the distal esophagus to the diaphragm stops reflux (Figs. 1–3). Normally, the distal esophagus rests within the abdomen. As the esophagus traverses the hiatus, the crura of the diaphragm compress the esophagus, increasing LES pressure. This compression is maximal during inspiration, when intrathoracic pressure decreases and risk of reflux is greatest. The intraabdominal pressure is also greater than that of the thorax. This high-pressure zone is transmitted to the distal intraabdominal esophagus, thus contributing to the LES pressure (4,5).

Pathologic reflux occurs if the elements contributing to the LES are dysfunctional. In the absence of a primary esophageal motility disorder, the most common cause of reflux and a low LES pressure is a Type I or *sliding* hiatal hernia (Fig. 4). A sliding hiatal hernia develops when there is a laxity of the phrenoesophageal attachments. High intraabdominal and negative intrathoracic pressures cause the distal esophagus and gastric cardia to migrate into the chest, lowering the LES pressure and allowing reflux to occur (2,4,5).

Antireflux procedures augment the LES pressure by returning the distal esophagus to abdomen. The relationship of the esophagus to the diaphragm and fundus is restored by repairing the hiatus and performing a fundoplication.

SYMPTOMS OF GERD

GERD presents with symptoms related to exposure of gastric contents to the esophagus, pharynx, and lungs. Heartburn is the most common presenting symptom of GERD,



Fig. 1. Normal anatomy of esophageal hiatus: coronal section.



Fig. 2. Normal anatomy of esophageal hiatus: overview.



Fig. 3. Normal anatomy of esophageal hiatus: upper gastrointestinal contrast study.

occurring in 80% of patients. Chronic acid reflux can lead to esophagitis. In severe cases of esophagitis, stricture may develop leading to dysphasia. Belching and regurgitation occur in 50% of patients with GERD. Thirty percent of patients present with abdominal pain. Occasionally, patients present with minimal heartburn but with severe extra-esophageal manifestations of GERD. Chronic respiratory symptoms, such as chronic cough, recurrent pneumonias, episodes of nocturnal choking, and asthma may occur. Chest pain may be an atypical symptom of GERD. Fifty percent of patients in whom a cardiac cause of the chest pain has been excluded will have increased acid exposure as the etiology (1,2).



Fig. 4. Type I hiatal or sliding hiatal hernia: coronal section.

INDICATIONS FOR SURGERY

The majority of patients with heartburn can be managed through modification of lifestyle and through medical management. These should be optimized prior to consideration of surgery.

Caffeine, tobacco, and alcohol all decrease the LES pressure and cause reflux. Large meals late at night often results in nocturnal reflux symptoms. Their elimination will often improve GERD. Obesity increases intrabdominal pressure. Weight loss will often effectively decrease reflux. H2-blockers and proton pump inhibitors potently neutralize gastric secretions stopping heartburn and healing esophagitis (1).

When lifestyle modifications and medications are ineffective or poorly tolerated, surgery should be considered. In addition, in patients who are good surgical risks, LNF is an excellent alternative to lifelong medication (6).

Patients with esophageal injury because of acid reflux (including esophagitis, ulceration, stricture, and Barrett's metaplasia) should be considered for surgery. Although these complications can be controlled with medication, cessation of treatment often leads to recurrence. Regurgitation despite acid suppression is a clear indication for surgery (1,6).

Healthy patients are able to tolerate general anesthesia and laparoscopy, and they should be considered candidates for surgery. In particular, patients less than 50 yr old should consider surgery as an alternative to lifetime medication. Elderly patients are usually best treated medically (1,5,6).

CONTRAINDICATIONS TO SURGERY

There are few absolute contraindications to LNF except those precluding laparoscopy or general anesthesia. Although LNF has been done successfully in patients older than 70 yr of age, the risk of surgery will often outweigh the benefit of avoiding medication. There are several relative contraindications to surgery. Obesity increases the technical difficulty of LNF, and is often a cause for conversion to an open procedure. Obesity will also increase the risk of long-term failure of the fundoplication with recurrence of symptoms. Morbid obesity is better treated with medical management or with gastric bypass surgery. Previous upper abdominal or gastric surgery increases the difficulty of LNF necessitating an open approach (1-3,5,6).

PREOPERATIVE EVALUATION

Prior to surgery, the patient should undergo ambulatory esophageal pH testing, esophageal motility testing, and upper gastrointestinal (GI) endoscopy. Most patients will have an upper GI series (Fig. 5).

The success of LNF in eradicating GERD is dependent on the presence of acid reflux. Ambulatory 24-h pH testing will accurately characterize the severity of GERD, and allows the correlation of symptoms with acid reflux. The absence of acid reflux or poor correlation between symptoms and reflux is a predictor of poor outcome of surgery (2,5,6).

Esophageal motility testing is essential to rule out esophageal motility disorder as causing symptoms. An LNF done in the presence of a motility disorder can lead to severe dysphagia. Patients with poor motility may benefit from a partial fundoplication (2,5,6).

Upper GI endoscopy should be performed to document the presence of esophagitis or Barrett's esophagus both visually and through biopsies. Barrett's esophagus is a potentially premalignant columnar metaplasia of the distal esophagus that can progress to adenocarcinoma. Patients with Barrett's esophagus need lifetime surveillance endoscopy to identify potential progression to severe dysplasia, which is an indication for esophagectomy (2,3).

LAPAROSCOPIC FUNDOPLICATION: CONDUCT OF OPERATION

Selection of the antireflux procedure and approach is based on an assessment of esophageal contractility and length. A transabdominal approach is used for patients with normal esophageal contractility and length. Patients who present with long-standing disease associated with poor esophageal function, a short esophagus, or stricture should undergo an open antireflux procedure tailored to their underlying anatomic and physiologic abnormalities. Those with weak esophageal contractions may be treated with a partial 270° fundoplication such as the transabdominal Toupet (Fig. 6) or transthoracic Belsey IV fundoplication in order to avoid the increased outflow resistance associated with a 360° Nissen fundoplication. Patients with poor contractility or questionable esophageal length can be approached transthoracically. If the esophagus is too short after it is mobilized from diaphragm to aortic arch, a Collis gastroplasty is done to provide additional esophageal length and to avoid placing the repair under tension. Finally, if the disease has resulted in esophageal body failure, Barrett's metaplasia with high grade dysplasia, or esophageal adenocarcinoma, an esophagectomy is required (2,3,5).



Fig. 5. Type I hiatal or sliding hiatal hernia: upper GI contrast study.

Laparoscopic Nissen fundoplication (Fig. 7) is the procedure of choice in the majority of patients presenting with GERD. General anesthesia is required. The patient is placed in a low lithotomy position. Pneumoperitoneum and five laparoscopic trocars are placed (Fig. 8). The Nissen fundoplication (laparoscopic or open) is performed in what can be summarized as four major steps:

- 1. Crural Dissection: Crura of the diaphragm are circumferentially dissected from the distal esophagus and stomach by dividing the phrenoesophageal attachments. The lower esophagus is completely mobilized, returning the distal esophagus to the abdomen without tension. The vagus nerves are preserved.
- 2. Fundic mobilization: The gastric fundus is completely mobilized by division of the short gastric vessels and retrogastric attachments.
- 3. Crural closure: The crura of the diaphragm are loosely approximated posteriorly.
- 4. Fundoplication: A short, loose 360° fundoplication is created by wrapping the anterior and posterior walls of the fundus around the distal esophagus and vagus nerves. This loose wrap is 1.5 to 2 cm in length (2,5,6).



Fig. 6. 270° (Toupet) fundoplication.



Fig. 7. 360° (Nissen) fundoplication.

In the presence of altered esophageal motility, where the propulsive force of the esophagus is not sufficient to overcome the outflow obstruction of a complete fundoplication, a partial 270° Toupet fundoplication can be performed. This procedure is identical to the



Fig. 8. Incision locations for Belsey IV and open fundoplications, trocar positions for laparoscopic fundoplication.

Nissen fingoplication except that the stomach is sutured to the esophagus and crura, leaving the anterior esophagus uncovered and able to fully distend (4).

Operative time is 2-3 h. The patient begins liquids that night and is discharged the following morning. Most patients return to sedentary work in 2 wk. Patients are advised to avoid heavy lifting and straining for 6-8 wk to decrease the risk of herniation of the fundoplication into the chest. Patients are given a diet progressing to solid foods over 2 wk (5).

RESULTS AND COMPLICATIONS

Nissen fundoplication is extremely effective in treating GERD. Typical symptoms of GERD (heartburn, regurgitation, and dysphagia) are alleviated in 90 to 95% of patients. With the open technique, 90% of patients have no recurrence of symptoms at 10 yr. The first LNF was done in 1991; therefore, 10-yr data is not available. However, the LNF is identical to the open procedure, and 5-yr data for LNF is similar to that seen in the open Nissen fundoplication (3,5).

Recurrence of symptoms is reported to occur in 3.4% after 3 yr in a meta-analysis by Perdikis. Recurrent reflux may be caused by inadequate technical repair, shortening of the esophagus or inadequate esophageal mobilization leading to excessive tension and retraction of the fundoplication into the chest, or weakening of the musculofascial structures by aging, atrophy, or obesity. Recurrent symptoms can usually be treated medically (2,6).

Less than 1% of patients require further surgical intervention for recurrent reflux. This subgroup consists mainly of patients who had severe esophagitis, esophageal stricture, and ulceration prior to surgery, and whose fundoplication failed. Failed fundoplication can take several forms: disrupted wrap, "slipped" fundoplication onto the proximal stomach, and recurrent hiatal hernia with intrathoracic migration of the fundoplication (Fig. 9) (5,6).

Conversion rate to open surgery is approx 2%, and early reoperation is necessary in 0.5%. Morbidity following LNF averages 3–10%. Pulmonary complications are more



Fig. 9. Intrathoracic migration of fundoplication.

common and morbidity is higher after a thoracic operation than after transabdominal surgery. Pitfalls unique to the laparoscopic approach include pneumothorax and surgical emphysema, occurring in 1 to 2% of patients. Death is rare, whether the procedure is open or closed. In a recent collective review, 4 of 2453 (0.2%) patients died. Deaths that have been reported were caused by visceral perforation, superior mesenteric vessel thrombosis, and myocardial infarction (5,6).

The most serious operative complication is instrumental perforation of a hollow viscus. Perforations can also occur in the stomach or distal esophagus caused by passage of the bougie used to size the fundoplication. If recognized at the time of surgery, a perforation can be repaired without added morbidity. Unrecognized perforations will be manifested by postoperative toxicity (fever, oliguria, hypoxia, tachycarida, and peritoneal signs). Suspicion of a perforation should necessitate radiological examination or reexploration in a timely fashion (5,6). Postoperative hemorrhage is unusual, but can arise from the liver, short gastric vessels, or spleen. Splenic injury necessitating splenectomy has been virtually eliminated with the advent of laparoscopic fundoplication (5,6).

Dysphagia is the most common postoperative complaint occurring in 30% of patients. Dysphasia is usually worse with solids, is self-limited, and is caused by postoperative edema at the fundoplication. Persistent postoperative dysphagia occurs in approx 9% of patients after laparoscopic repair and in 3% after open. The majority of patients are asymptomatic by 8 wk. An esophagram and endoscopy may help to define the underlying problem. Gentle dilation of the fundoplication will usually alleviate symptoms (5,6).

Gastric distension ("Gas bloat") was common after the early variation of the open Nissen fundoplication. It is relatively uncommon today because of routine creation of a short "floppy" fundoplication that allows the patients to belch to a limited degree. Patients undergoing antireflux surgery habitually swallow air to clear the esophagus of reflux contents, and this habit continues after antireflux surgery. Gas-binding agents and prokinetics may be helpful when patients complain of bloating and increased flatulence (5,6).

Diarrhea and nausea occur in up to 8%. Most of these symptoms disappear after several weeks, and medical therapy is usually not required. Postoperative diarrhea is thought to be caused by rapid gastric emptying, change of diet, or incidental vagotomy. Severely affected patients may be treated with antidiarrheals (1,4).

ALTERNATIVE PROCEDURE

Lapraoscopic vs Open Fundoplication

LNF has been compared to the open Nissen fundoplication as well as to the Belsey IV. LNF has equivalent control of symptoms to open Nissen, and superior results to the Belsey IV. LNF has less perioperative morbidity and a shorter recovery time. Convalescence is faster after laparoscopy: return to normal life being 14 vs 31 d and return to work being 21 vs 44 d (1,2).

Partial vs Complete Fundoplication (Table 1)

In contrast to the 360° fundoplication typical of a Nissen fundoplication, antireflux protection also occurs when the fundus is incompletely wrapped around the lower esophagus. Table 1 lists some of the common antireflux procedures and a description of their conformation. Partial fundoplication results in less postoperative bloating and dysphagia. However, partial fundoplication has a higher incidence of recurrent reflux and, therefore, is not routinely used. Partial fundoplication is typically reserved for patients with abnormal esophageal motility, such as scleroderma and achalasia. Partial fundoplication has been linked to a greater overall level of patient satisfaction 6 mo after surgery. Fewer patients had difficulty swallowing, inability to belch, or had excessive flatus in the partial fundoplication patients. These benefits may be offset by recurrent GERD. In addition, the prevalence of these symptoms in patients following Nissen fundoplication is highly dependent on the technical aspects of the procedure that can be minimized given ideal technique (4).

Angelchik Procedure

The Angelchik procedure is no longer performed. It involves placing a doughnut shaped silicone prosthesis around the intraabdominal esophagus. After it is tied in place,

Partial Fundoplications			
Туре	Eponym	Year Described	Description
Total	Nissen	1956	360° wrap
	"Floppy" Nissen	1977	360° short (< 2 cm) wrap
	Rosettil	1965	360° with short gastric vessels not divided
Partial	Belsey Mark IV	1967	270° transthoracic
	Toupet	1963	180° posterior wrap
	Dor/Watson	1962	180°/120° anterior wrap
	Modified Toupet	1982	270° posterior wrap
	Lind	1965	270° posterior with crural closure
	Guarner	1975	270° posterior with gastropexy
	Thal	1964	90° anterior wrap
Other	Allison	1951	Hiatal closure with esophagogastropexy
	Hill	1967	Esophagogastropexy with 180° anterior wrap

Table 1 Partial Fundoplications

the prosthesis prevents the hiatal hernia from recurring and mildly constricts the lower esophagus with increase in the sphincter pressure. Although insertion of the prosthesis is easier than fundoplication, patients often require reoperation to remove the prosthesis because of migration, esophageal compression, ulceration, or erosion. More than 100,000 of these procedures have been performed (5).

COST

Although medical therapy and surgery both control GERD, the cost of LNF is a single initial expense, whereas the cost of medical therapy is lifelong. Charges for surgery include hospital charges of approx \$5000 and professional fees of \$2000. When one compares the cost of proton-pump inhibitors with surgery, open Nissen fundoplication becomes a cost-effective treatment option compared with medical treatment in patients with refractory GERD if treatment continues more than 4 yr. LNF shifts this so-called break point toward 1.4 yr, mainly because of a shorter hospital stay (7-9).

SUMMARY

- 1. GERD is a common condition and majority of patients are managed effectively by medical therapy.
- 2. Antireflux surgery restores the mechanically defective esophageal sphincter and is an effective treatment in patients suffering from severe GERD, which is unresponsive to medical therapy.
- 3. Laparoscopic Nissen fundoplication is the superior antireflux procedure for the majority of patients requiring surgery for GERD, but it is technically demanding and should be performed by properly trained and experienced surgeons.
- 4. The outcome of laparoscopic Nissen fundoplication is excellent and morbidity is minimal. It offers a cost effective alternative to medical therapy in healthy patients with refractory symptoms.

REFERENCES

- 1. Katz PO. Treatment of gastroesophageal reflux disease: use of algorithms to aid in management. Am J Gastroenterol 1999;94:11. Suppl.
- Soper N. Laparoscopic management of hiatal hernia and gastroesophageal reflux. Curr Probl Surg 1999;36:765–838.
- 3. Perdikis G, Hinder RA, Lund RJ, et al. Laparoscopic Nissen fundoplication: where do we stand? Surg Laparosc Endosc 1997;7:17–21.
- 4. Swanstrom LL. Partial fundoplication for gastroesophageal reflux disease: indications and current status. J Clin Gastroenterol 1999;29:127–132.
- 5. Bowrey DJ, Peters JH. Laparoscopic esophageal surgery. Surg Clin N Am 2000;80:1213–1242.
- 6. Klingler PJ, Bammer T, Wetscher GJ, et al. Minimally invasive surgical techniques for the treatment of gastroesophageal reflux disease. Digest Dis 1999;17:23–36.
- Heikkinen TJ, Haukipuro K, Koivulkangas P, et al. Comparison of costs between laparoscopic and open Nissen fundoplication: a prospective randomized study with a 3-mo follow-up. J Am College Surg 1999;188:368–376.
- 8. Narain PK, Moss JM, DeMaria EJ. Feasibility of 23-h hospitalization after laparoscopic fundoplication. J Laparoendoscop Adv Surg Tech 2000;10:5–11.
- 9. Van Den Boom G, Go PM, Hameeteman W, et al. Cost effectiveness of medical versus surgical treatment in patients with severe or refractory gastroesophageal reflux disease in the Netherlands. Scand J Gastroenterol 1996;31:1–9.

Hiatal Hernia Repair

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CONTENTS

INTRODUCTIOIN INDICATIONS FOR PROCEDURE CONTRAINDICATIONS REPAIR OF PARAESOPHAGEAL HERNIA COMPLICATIONS RESULTS ALTERNATIVE PROCEDURES COST SUMMARY REFERENCES

INTRODUCTION

The history of surgery for hiatal hernia and gastroesophageal reflux disease (GERD) has paralleled our gradual understanding of the physiological features of the esophagus. The association between GERD and esophagitis was not established until the 1940s, and much controversy arose concerning the relationship between hiatal hernia and GERD. Initial attempts at simply reducing the hernia by closing the crura proved to have unacceptably high failure rates. The Allison repair, introduced in 1951, involved mobilization of the distal esophagus with placement of the gastroesophageal junction within the abdomen and repair of the crura. This operation had a high recurrence rate, and subsequently several attempts were made at both fixing the gastroesophageal junction within the abdomen and wrapping the gastric fundus around the distal esophagus (fundoplication) to create an antireflux valve (1).

The most commonly performed hiatal hernia repair is the Nissen fundoplication. This was first performed in 1937 in a patient with a perforated ulcer of the gastric cardia in an effort to protect the repair. Because this patient subsequently had no evident reflux, Nissen performed this operation purposefully in patients with GERD. Other fundoplications that have become eponymic were subsequently developed, applied, and reported; but modifications of the Nissen fundoplication are currently the most widely used operations for GERD and hiatal hernias, (*see* Chapter 4, Table 1) (1,2).

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Fig. 1. Type II hiatal or pure paraesophageal hernia: coronal section. Gastroesophageal junction is in normal intrabdominal position.

The term hiatal hernia refers to the protrusion of any structure other than the esophagus through the esophageal hiatus of the diaphragm and generally refers to a sliding hiatal hernia or a paraesophageal hiatal hernia. The most generally accepted nomenclature of hiatal hernia includes four principal categories. Type I, or sliding hiatal hernia, accounts for more than 90% of all hiatal hernias (see Chapter 4, Fig. 4). The esophagogastric junction is displaced through the hiatus into the mediastinum because of circumferential weakening of the phrenoesophageal membrane. Eighty percent of patients with GERD have type I hernia. Type II, or paraesophageal hernia, accounts for less than 5% of hiatal hernias (Figs. 1 and 2). Type II hernia occur more commonly in an older population than do sliding hiatal hernia. The esophagogastric junction remains fixed below the diaphragm, and the gastric fundus herniates through the defect into the mediastinum. Type III is a combination of both types I and II hernia (Fig. 3). Components of both sliding and paraesophageal hernia are present. It is seen in more than 5% of patients. Finally, Type IV is comprised of anatomically complex hiatal hernias. In addition to sliding and paraesophageal components, Type IV hernia contain other viscera such as the colon, omentum, small intestine, pancreas or spleen (Figs. 4 and 5) (2,3).

Other rare hernias may occur in the hiatal region, including parahiatal hernias that can be differentiated from types I through IV in that there is a separate extra-hiatal diaphragmatic defect in which intervening normal crural muscle tissue is present. They are rarely seen and may be associated with previous trauma. Additionally, congenital diaphragmatic hernias (Bochdalek—posterolateral or Morgagni—retrosternal) are more likely to present in childhood but may not be found until much later in life (2-4).



Fig. 2. Type II hiatal or pure paraesophageal hernia: upper gastrointestinal contrast study.

INDICATIONS FOR PROCEDURE

The majority of sliding hiatal hernias are asymptomatic. Only when surgical intervention is indicated for GERD should Type I (sliding hiatal hernia) be repaired (*see* **Chapter 3**) (2).

The presence of Type II paraesophageal hiatal hernias has traditionally been considered an indication for surgery in a patient who is otherwise fit for surgery. Paraesophageal hiatal hernias have been associated with the risk of strangulation of incarcerated viscera and the potential need for emergency operations. In their classic article from 1967, Skinner and Belsey found that 6 of 21 patients under review for paraesophageal hernias developed gastric volvulus and died of the catastrophic complications of strangulation, perforation, exsanguinating hemorrhage, or acute dilatation of the herniated intrathoracic stomach. Although other series reported similar findings to that of Skinner and Belsey, more recent large series suggest that symptoms associated with paraesophageal



Fig. 3. Type III or mixed sliding and paraesophageal hernias: coronal section.



Fig. 4. Type IV hiatal hernia: combination of type III with herniation of other viscus (colon).

hernia may develop more gradually. Allen et al. followed 23 patients for a median of 78 mo and found only three cases of gastric strangulation in 735 patient years of follow-up (5).

Although the true incidence of gastric volvulus presenting with strangulation is controversial (ranging from 3-30%), the elective repair of paraesophageal hernia is generally recommended because emergency surgery for acute complications carries a high mortality rate (5,6).

There are two patterns to gastric volvulus, organoaxial and mesenteroaxial (Figs. 6 and 7). Organoaxial volvulus occurs when 180° torsion occurs about the stomach's longitudinal axis. Mesenteroaxial volvulus is less common and occurs with torsion about



Fig. 5. Type IV hiatal hernia: barium enema study demonstrates herniated colon.



Fig. 6. Paraesophageal hernia: gastric volvulus.

the vertical axis. Thirty percent of patients with paraesophageal hiatal hernia present with hematemesis or exhibit anemia, which is likely caused by mucosal hemorrhage



Fig. 7. Paraesophageal hernia: gastric volvulus. Upper gastrointestinal contrast study.

from venous congestion located at the neck of the hernia. They may also complain of dysphagia, early satiety, referred diaphragmatic pain with postprandial gastric distention, and weight loss (7).

About 30% of patients with paraesophageal hernia have Type 3 hernia and have symptoms of GERD. Many patients with paraesophageal hernia have no serious symptoms or complications of their condition for years. As the hernia progresses, varying degrees of complaints and severity of symptoms will be directly attributable to the configuration of the hiatal hernia (2).

Gastric volvulus presenting with infarction occurs when the stomach dilates and gastric ischemia occurs. Progression of ischemia can lead to perforation. Symptoms of epigastric pain, the inability to vomit, and gastric obstruction on contrast study are indication for emergency intervention (2).

When patients with paraesophageal hiatal hernias are considered for operative repair, diagnostic tests should include upper endoscopy to exclude other significant esophageal mucosal disease, upper gastrointestinal contrast radiographs to classify the type of hiatal hernia and give an indication of the degree of esophageal shortening, and esophageal manometry to assess the adequacy of esophageal peristalsis (2,6).

CONTRAINDICATIONS

There are few absolute contraindications for an operation, including inability to tolerate a general anesthetic or an uncorrectable coagulopathy (2,4,6).

REPAIR OF PARAESOPHAGEAL HERNIA

The repair can be performed transthoracally, transabdominally, or laparoscopically. Factors including patient age, medical conditions, elective vs emergency procedure, presence of esophageal shortening, prior surgery, obesity, and body habitus all influence choice of surgical approach and procedure performed (2,4,6).

The thoracic approach is favored in patients who are obese, who have had extensive upper abdominal surgery, and who have a type III hernia with severe esophageal shortening necessitating extensive esophageal mobilization or a lengthening procedure.

The majority of cases are best approached transabdominally. Through this approach the volvulus is readily reduced, gastropexy can be performed, and if esophageal mobilization is not adequate, a lengthening procedure can also be performed.

Laparoscopic repair of paraesophageal hernia has been reported in numerous series. Although the technique is similar to that of the standard laparoscopic Nissen fundoplication, the technical difficulty of repair of paraesophageal hiatal hernias is generally much greater than that for antireflux surgery alone. The inherent difficulties of this operation include the underlying physical status and frequent comorbidities of the patient, the often compromised nature of the gastric wall, which has been incarcerated chronically in a mediastinal hernia sac, the necessity of excising the hernia sac without damaging critical structures, and the problem of closing the enlarged hiatus adequately. Therefore, unlike the standard laparoscopic Nissen done for reflux, laparoscopic repair of paraesophageal type II, III, and IV hernia should only be done in tertiary care centers by surgeons with extensive experience in laparoscopic antireflux surgery (2,4,6).

The repair of a paraesophageal hernia is performed transabdominally through an upper midline incision, or laparoscopically with five to seven trocars under general anesthesia. The critical steps of the procedure are as follows:

- 1. Mobilization of gastric fundus. The short gastric vessels are divided and the left crus is identified.
- 2. Reduction of stomach into peritoneal cavity. Mobilization of the hernia sac from the mediastinum facilitates reduction of the gastric fundus. The hernia sac is either completely resected or at least circumferentially transected at the hiatus.
- 3. Identification and mobilization of the esophagus and vagus nerves. The esophagus must be fully mobilized distally to allow reduction into the peritoneal cavity without tension.
- 4. Closure of hiatus. The crura are approximated. Nonabsorbable mesh is occasionally employed to close the defect.
- 5. Fundoplication. A loose 360° nissen fundoplication is created over a bougie.
- 6. Gastropexy. The body of the stomach is sutured to the abdominal wall with placement of gastrostomy tube (1,6,7).

There are several controversies regarding repair of paraesophageal hiatal hernias. These include the necessity of excising the hernia sac, the best technique for closing the diaphragm, the requirement of an antireflux procedure, and the need to perform a gastropexy. There are few definitive studies to answer these questions. Most authors recommend routinely performing a fundoplication. First, two-thirds of patients with paraesophageal hernia have objective evidence of GERD. Second, even though GERD may not be a complaint before the operation, the extensive periesophageal dissection performed during this operation may lead to significant postoperative GERD. Finally, the presence of the fundoplication helps to fix the wrapped fundus in the abdomen, because its diameter is greater than that of the esophagus by itself (1,4,7).

COMPLICATIONS

Morbidity of surgery for paraesophageal hernia repair is significantly greater than that for antireflux surgery alone. The poorer outcomes are both a result of the nature of the patient population and the difficulty of the operative approach. Patients with paraesophageal hiatal hernias are significantly older with more comorbidities than the usual patient undergoing an antireflux operation. The average age of patients with a paraesophageal hernia is 70–80 yr. These patients commonly have significant preexisting conditions. Complications, such as atelectasis, pneumonia, deep venous thrombosis, pulmonary embolism, myocardial infarction, and arrhythmias, may occur (1,8).

Complications are more frequent than those reported with antireflux surgery. Traumatic visceral injury or vagus nerve injury occurred in 10-15%. Pneumothorax occurred in 5-10% because of the more extensive mediastinal dissection. Subcutaneous emphysema occurred in 50% of laparoscopic cases, but resolved spontaneously as carbon monoxide is absorbed.

In most series, 10% of patients have dysphagia, poor gastric emptying, recurrent hernia, and GERD leading to suboptimal outcome after paraesophageal hernia repair. Recurrence of the paraesophageal hiatal hernia can occur and be asymptomatic. This may be caused by an inability to close the hiatus in a tension-free fashion. Several authors have recommended placement of a prosthesis at the hiatus. However, cicatricial involvement of the esophagus by a piece of mesh can lead to significant complications (3,6).

Postoperative complications are encountered 20–30% of patients, and reoperations are necessary in nearly 10% of patients. Mortality following elective repair is less than 5%, and in centers with a large volume of esophageal surgery, mortality should be less than 1% (5,8).

RESULTS

Most studies report relief of symptoms in more than 90% of patients with follow-up for more than 5 yr. These results are true of laparoscopy, laparotomy, and thoracotomy. Long-term results of open repairs suggest that 83-100% of patients remain symptom free after a mean follow-up of 6 yr. Laparoscopic repair is as successful and safe as open. Laparoscopic repair, in expert hands, has a shorter hospital stay, a lower hospital cost, and increased patient satisfaction. However, the long-term durability of laparoscopic repairs is as yet unknown. Some retrospective studies suggest that transthoracic approach for large paraesophageal hernia might be associated with the highest likelihood of a durable repair (5,8).

ALTERNATIVE PROCEDURES

Patients who are debilitated and are not candidates for formal repair of paraesophageal hernia may be treated with gastropexy performed with gastrostomy. Although this will

not allow complete reduction of the fundus from the hernia sac, gastropexy will eliminate the risk of volvulus and strangulation. This is rapidly performed either laparoscopically or through a small laparotomy (5,6).

COST

Cost of repair of paraesophageal hernia varies and depends on the size and complexity of the hernia. Overall, it can be as low as \$5000 for hospital cost and \$7000 for total cost for laparoscopic repair of a type II paraesophageal hernia, followed by an antireflux procedure. However, cost can be significantly higher for repair of large, long-standing, complex hernias in old debilitated patients who may have prolonged, complicated hospital courses (9).

SUMMARY

- 1. Sliding (type I) hiatal hernias are only repaired when associated with significant symptoms of GERD. Laparoscopic Nissen fundoplication is the usual repair performed.
- 2. Paraesophageal (type II, III, and IV) hernias are relatively uncommon. Most occur as mixed-type hernias in association with a sliding hiatal hernia.
- 3. Paraesophageal hernia are present in an elderly population with symptoms of early satiety, dysphagia, dyspnea, and chest pain.
- 4. Paraesophageal hernia can present as a gastric volvulus. This can occur with strangulation necessitating emergency surgery with high morbidity and mortality. Good surgical risk patients should be offered elective hernia repair.
- 5. The surgical management of patients with paraesophageal hernias can be complicated. The anatomic derangements are complex and variable.
- 6. Laparoscopic paraesophageal hernia repairs require advanced skills and a thorough knowledge of the hiatal area and include hernia reduction, crural closure, fundoplication, and gastropexy.

REFERENCES

- 1. Bowrey DJ. Laparoscopic esophageal surgery. Surg Clin N Am 2000;80:1213-1241.
- 2. Oddsdottir M. Paraesophageal Hernia. Surg Clin N Am 2000;80:1243-1253.
- 3. Williamson WA, Ellis FHJ, Streitz JMJ, et al. Paraesophageal hiatal hernia: is an anti-reflux procedure necessary? Ann Thorac Surg 1993;56:447-451.
- 4. Skinner DB, Belsey RH. Surgical management of esophageal reflux and hiatal hernia. Longterm results with 1030 patients. J Thorac Cardiovasc Surg 1967;53:33–54.
- 5. Soper NJ. Laparoscopic management of hiatal hernia and gastroesophageal reflux. Curr Probl Surg 1999;36:765–838.
- Perdikis G, Hinder RA, Filipi CJ, et al. Laparoscopic paraesophageal hernia repair. Arch Surg 1997; 132:586–589.
- 7. Hashemi M, Sillin LF, Peters JH. Current concepts in the management of paraesophageal hiatal hernia. J Gastroenterol 1999;29:8–13.
- 8. Trus TL, Bax T, Richardson WS, et al. Complications of laparoscopic paraesophageal hernia repair. J Gastrointest Surg 1997;1:221–228.
- 9. Frantzides C. Laparoscopic repair of large hiatal hernia with polytetrafluoroethylene. Surg Endosc 1999;13:906–908.

Esophageal Stents

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Introduction Indications and Contraindications Types of Esophageal Stents Technique Complications Alternative Procedure Cost Summary References

INTRODUCTION

Esophageal cancer is the seventh most common cause of cancer-related deaths in men in the United States, with an estimated 12,300 new cases diagnosed each year. The tumor is often diagnosed at advanced stages of the disease, resulting in 60% of patients being incurable at the time of diagnosis (Fig. 1). Thus, palliative therapy is an important modality in the treatment of these patients (1).

The major goals in the palliative care of patients with primary and metastatic carcinoma involving the esophagus is the management of severe dysphagia, prevention of aspiration, providing adequate nutrition, and treating tracheoesophageal fistulas. A variety of different palliative therapies have been developed, each with their own limitations and complications. Although surgical palliation may be considered, it is associated with mortality rates of 5% to 60% (2–4). Radiation therapy palliates dysphagia in less than 40% of patients, and results are not apparent until approx 2 mo following initiation of treatment (5). Placement of a rigid esophageal endoprosthesis is traumatic and associated with high morbidity and mortality rates (21% and 15.8%, respectively) (6). Luminal patency with endoscopic laser therapy can be achieved in more than 90% of cases, however, this requires multiple treatment sessions (7). The development of self-expanding metallic stents (SEMS) has been a major breakthrough in the palliation of

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Fig. 1. Barium swallow showing obstructing esophageal carcinoma.

esophageal cancer. Because of the design and ease of insertion, they are less traumatic and may be placed in an outpatient setting. Immediate relief of dysphagia is achieved with fewer complications (8).

INDICATIONS AND CONTRAINDICATIONS

In addition to management of intraluminal obstruction and esophagorespiratory fistulas, SEMS can be utilized in the management of extrinsic esophageal obstruction owing to compressive mediastinal tumors (9,10). Contraindications include total luminal obstruction prohibiting passage of a guide wire, extremely limited life expectancy, actively bleeding lesions, and significant airway compression (11).

TYPES OF ESOPHAGEAL STENTS

Currently, there are three esophageal stents used in the United States. They are as follows (Table 1).

Gianturco Z-stents are made from 0.018-in stainless steel wire bent in a zig-zag fashion to form segments 2-cm long, which are connected using nonabsorbable suture to form lengths from 6 to 14 cm. The stent is 18 mm in its internal diameter, with the proximal and distal ends flared to 25 mm. The Z-stents are available in fully covered and

	Gianturco-Z stent ¹	Ultraflex ²	Wallstent II ³
Material	Stainless steel	Nickel titanium (Nitinol)	Elgiloy
Covering	Yes	Yes	Yes
Design	Zig-zag configuration	Mesh	Mesh
Self-expanding	+	+	+
Diameter before			
Implantation	28F	16F	18F
Shaft diameter			
Post implantation	18 mm	18,23 mm	20 mm
Flange diameter	21,25 mm	23,28 mm	28 mm
Degree of			
Shortening	0%-10%	30%-40%	30%
Radial force	++	+	+++
Fistula Closure	Yes	Yes	Yes

Table 1

¹Gianturco Z-Stent (Wilson-Cook Medical, Winston-Salem, NC)

²Ultraflex stent (Microvasive, Inc., Natick, MA)

³Wallstent II (Microvasive)



Fig. 2. Esophageal stents. (**A**) Covered Gianturco Z-Stent (Wilson-Cook Medical, Winston-Salem, NC). (**B**) Uncovered Gianturco Z-Stent (Wilson-Cook Medical, Winston-Salem, NC). (**C**) Ultraflex stent (Microvasive, Inc., Natick, MA). (**D**) Wallstent II (Microvasive) Covered Z stent.

partially covered versions (Fig. 2A,B), and some have a Dua antireflux valve for gastroesophageal junction tumors to prevent reflux of gastric contents.

The Ultraflex stent (Fig. 2C) is a self-expanding stent made of 0.15-in nitinol wire with a luminal diameter of 18 mm and a proximal flange diameter of 23 or 28 mm upon

deployment. Nitinol is a nickel titanium alloy that is unique for its shape memory properties. The stents vary in length from 7 to 15 cm. Ultraflex stents have less radial force that Wallstents (12). They are available coated and uncoated. The uncoated stent is more susceptible to tumor ingrowth and overgrowth (36%) (13).

The Wallstent (Fig. 2D) consists of eight stainless steel wires arranged in a spiral shape, forming a wire mesh. They are available in lengths of 10 cm and 15 cm. The stent is partially covered with a polyurethane coating. The conical configuration of the stent is designed to limit distal stent migration. It is available in two sizes: 1) small (proximal diameter of 24 mm, distal diameter 16 mm, total length 12 cm) for tumors without prestenotic dilatation; and 2) large (30-mm proximal diameter, 20-mm distal diameter, and a total length of 14 cm).

TECHNIQUE

Stents are placed under fluoroscopic control, and usually with endoscopic guidance. The choice of metal stents is based upon several variables, but predominantly on physician's experience and stent availability. Prior to the decision for SEMS placement, a barium swallow should be performed to provide an anatomic roadmap. However, in our experience, the barium study may overestimate the degree and length of narrowing due to under filling below a section of critical narrowing.

An upper endoscopy is mandatory, and this helps assess the esophageal wall integrity, point of critical narrowing, and length of the stricture or tumor. This helps determine the length of the stent that will be required for placement. Most centers will also do a bronchoscopic examination to assess the airway system prior to the stent placement.

Several techniques have been used to try and predict patients who may suffer from respiratory decompensation after the stent placement. In our institution, flow loop parameters, as well as oxygenation measurements, are performed prior to and after inflating a balloon dilator about the area were the stent will be placed for comparison. However, this has not been an accurate predictor because accurate flow loop measurements and oxygenation require an unsedated patient in the upright position.

For very tight stenotic lesions, balloon dilation of the esophagus to 27–30 Fr may be helpful. The proximal and distal ends of the lesion are marked internally by an endoscopic balloon of the same length and caliber of the planned stent is first inflated. If there is any evidence of oxygen by endoscopic submucosal injection of radio-opaque material such as ethiodol oil.

Depending on the make of the stent, it may be preloaded or for Z-stent, it may have to be back loaded into a 28-Fr delivery catheter. The delivery system is removed, and the stent inspected endoscopically (Fig. 3). Occasionally, infolding of the proximal funnel can occur with the Z stents, and this can be corrected using a 18-mm balloon dilator (Fig. 4).

COMPLICATIONS

The ease of insertion and effectiveness in relieving dysphagia has made placement of SEMS the current therapy of choice for palliation of unresectable esophageal carcinoma. On one hand, immediate palliation is achieved in 70–80% of patients. On the other hand, the incidence of postinsertion complications, such as stent migration, hemorrhage, and fistulization is high, with a reported incidence of 20–40%. Patients with prior radiation or chemotherapy seem prone to more frequent and serious complications.



Α

Fig. 3. Endoscopic view of the stent.



Fig. 4. Stent traversing esophageal lesion.

Stent migration is a problem associated with metal stents, particularly with covered stents (Gianturco-Z-Stents 11%, Wallstent 13%) compared to the uncovered stents (Ultraflex 1%, uncovered Wallstent 3%) (14). Risk factors for migration include tumors of the esophagogastric junction and tight malignant strictures. Technically, the risk of migration can be decreased by using larger and overlapping stents. Recurrent dysphagia sometimes occurs as a result of tumor in-growth through an uncovered stent (15). These patients may require debulking, dilation, or placement of a covered stent. Other reported stent complications include acute airway obstruction caused by airway compression upon deployment (16), tracheo-esophageal fistulas, chest pain, and perforation.

One retrospective study compared the Wallstent, Ultraflex, and Gianturco Z-Stent for palliation of malignant esophageal obstruction in a total of 87 patients with 96 implantations. All implantation procedures were successful, and complete sealing of esophago-airway fistulas were noted. The degree of dysphagia improved and was comparable in all three groups. The rate of reintervention and retreatment in the early period following stent placement was 22% in the Wallstent group, 37% in the Ultraflex group, and 10% with the Gianturco Z-stents. Subsequently, reintervention rates caused by complications during the follow-up period occurred in 43% in the Wallstents, 35% with the Ultraflex and 21% with the Z-stents (*17*).

Results from the first randomized, prospective study of 100 consecutive patients comparing three different stent types of covered expandable metal stents for palliation of dysphagia as a result of carcinomas of the esophagogastric junction demonstrated a similar degree of improvement in dysphagia with no statistical difference seen among the stent types. The stents used were Gianturco Z-stent, Ultraflex I, and Wallstent. Significant complications (perforation, migration, bleeding, severe chest pain, and procedure-related death) were seen more often with the Z-stent compared to the Ultraflex and Wallstent (36% vs 24% and 18%, respectively). However, these differences were not statistically significant (18).

ALTERNATIVE PROCEDURE

An endoscopic or radiologically placed percutaneous gastrotomy tube may be placed to help the nutrition, but should be used only as a last resort because these patients have progressive dysphagia, and eventually will have complications with swallowing their own saliva and other secretions.

COST

The cost of stent placement includes the stent, as well as that of upper GI endoscopy and fluoroscopy. The average cost of an uncovered stent is approx \$1400, whereas a covered stent costs about \$1600. The costs of endoscopy and fluoroscopy vary greatly depending upon the geographic location and the payer. In our institution, the physician fee is approx \$1000, whereas the reimbursement is estimated at \$950 and \$250 for an average private payer and Medicare, respectively.

SUMMARY

- 1. More than half of the cases of esophageal carcinoma are unresectable at the time of diagnosis.
- 2. Palliation of the primary symptom of dysphagia is worthwhile to improve swallowing, help prevent aspiration, and to improve nutrition and quality of life.

- 3. Esophageal stenting with self-expanding metallic stents is widely accepted, and the preferred modality for palliative care.
- 4. The designs of the metallic stents continue to evolve and, at present, the choice of stent used is based upon personal preference and previous experience. Larger, well-designed studies are needed to provide the endoscopist or radiologist with an evidence-based approach to appropriate stent selection.

REFERENCES

- 1. DeMeester TR, Barlow AP. Surgery and current management for cancer of the esophagus and cardia. Part II. Curr Prob Surg 1988;25:535–605.
- 2. Boyce HW Jr. Palliation of advanced esophageal cancer. Semin Oncol 1984;11:186-195.
- 3. Watson A. Surgery for carcinoma of the esophagus. Postgrad Med J 1988;64:860-864.
- 4. Postlethwait RW. Complications and deaths after operations for esophageal carcinoma. J Thorac Cardiovasc Surg 1983;85:827–831.
- Albertsson M, Ewers SB, Widmark H, et al. Evaluation of the palliative effect of radiatiotherapy for esophageal carcinoma. Acta Oncol 1989;28:267–270.
- 6. De Palma G, di Matteo E, Romano G, et al. Plastic prosthesis versus expandable metal stents for palliation of inoperable esophageal thoracic carcinoma: a controlled prospective study. Gastrointest Endosc 1996;43:478–482.
- Mellow MH, Pinkas H. Endoscopic laser therapy for malignancies affecting the esophagus and gastroesophageal junction. Analysis of technical and functional efficacy. Arch Intern Med 1995;145: 1443–1446.
- 8. Knyrim K, Wagner HJ, Bethge N, et al. A controlled trial of an expansile metal stent for alliation of esophageal obstruction due to inoperable cancer. N Engl J Med 1993;329:1302–1307.
- 9. De Gregorio B, Kinsman K, Katon R, et al. Treatment of esophageal obstruction from mediastinal compressive tumors with covered, self-expanding metallic A-stents. Gastrointest Endosc 1996;43: 483–489.
- 10. Bethge N, Sommer A, Vakit N. Palliation of malignant esophageal obstruction due to intrinsic and extrinsic lesions with expandable metal stents. Am J Gastroenterology 1998;93:1829–1832.
- Tygat GNJ, Jager H, Bartelsman J. Endoscopic prosthesis for advanced esophageal cancer. Endoscopy 1986;18:32–39.
- 12. Chan A, Shin F, Lam Y, et al. A comparison study on physical properties of self-expandable esophageal metal stents. Gastrointest Endosc 1999;49:462–465.
- 13. Acunas B, Rozanes I, Akpinar S, et al. Palliation of malignant esophageal strictures with self-expanding nitinol stents: drawbacks and complications. Radiology 1996;199:648–652.
- 14. Sieserma P, Hop C, van Blankenstein M, et al. A new design metal stent (Flamingo stent) for the palliation of malignant dysphagia: a prospective study. Gastrointest Endosc 2000;51:139–145.
- 15. Mayoral W, Fleischer D, Salcedo J, et al. Nonmalignant obstruction is a common problem with metal stents in the treatment of esophageal cancer. Gastrointest Endosc 2000;51:556–559.
- Libby E, Fawaz R, Leano A. Airway complications of expandable metal stents (Letter). Gastrointest Endosc 1999;49:136–137.
- May A, Hahn E, Ell C. Self expanding metal stents for palliation of malignant obstruction in the upper gastrointestinal tract. Comparative assessment of three stent types implemented in 96 implantations. J Clin Gastroenterol 1996;22:261–266.
- Sieserma P, Hop W, van Blankenstein M, et al. A comparison of 3 types of covered metal stents for the palliation of patients with dysphagia caused by esophagogastric carcinoma: a prospective, randomized study. Gastrointest Endosc 2001;54:145–153.

Endoscopic Therapy for Esophageal Varices

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INTRODUCTION

Varices along the gastrointestinal tract develop secondary to increased venous blood flow through portosystemic collaterals, most commonly as a consequence of portal hypertension. The formation of varices takes place at a variety of anatomic sites, with the varices at or near the gastroesophageal junction being the most common. The second most common localization of clinically significant varices is the stomach, where varices can be in continuity with esophageal varices or they can exist as a separate entity.

Cirrhosis is the most common underlying condition with prevalence of esophageal varices 50% to 60% (1). Approximately 30% of patients with esophageal varices have their condition complicated by variceal bleeding in the course of the disease (2). Each episode of variceal bleeding caries a mortality of approx 40% (3).

Important factors in pathophysiology of varices formation that are believed to influence risk of bleeding are the degree of portal hypertension with hepatic venous pressure gradient (HVPG), variceal size, degree of liver cirrhosis, and the degree of liver function preserved. It is recognized that patients with HVPG of less than 12 mmHg rarely have their disease complicated by variceal bleeding (4). Factors such as alcohol use, poor nutrition, thrombocytopenia, and coagulopathy may influence outcome during an

From: *Clinical Gastroenterology: An Internist's Illustrated Guide to Gastrointestinal Surgery* Edited by: George Y. Wu, Khalid Aziz, and Giles F. Whalen © Humana Press Inc., Totowa, NJ acute episode. Bleeding often precipitates encephalopathy and increases the risk of spontaneous bacterial peritonitis (SBP), which further complicates the management of these patients.

A common system used for endoscopic grading of esophageal varices classifies them by their size (5):

Grade I:	Small varices without luminal prolapse.
Grade II:	Moderate-sized varices with minimal luminal prolapse at gastroesophageal
	junction.
Grade III:	Large varices with significant luminal prolapse substantially obscuring the
	gastroesophageal junction.
Grade IV:	Very large varices completely obscuring the gastroesophageal junction.

Mainly, their anatomic location and relationship to esophageal varices classify gastric varices (6):

Type I:	Gastric varices that appear as an inferior extension of esophageal varices.
Type II:	Varices in the gastric fundus in continuity with esophageal varices.
Type III:	Isolated gastric varices in the fundus, body or antrum of the stomach.

ENDOSCOPIC TREATMENT OF ESOPHAGEAL VARICEAL HEMORRHAGE

Endoscopy is the most commonly utilized therapeutic intervention in the initial attempt to control active hemorrhage. Initial management depends on patient hemodynamic stability at the time of presentation. Hemodynamically unstable patients are resuscitated with supportive medical therapy, which includes intravascular volume resuscitation with iv fluids and blood products, and chemotherapeutic attempt to lower portal pressure with iv vasopressin or somatostatin. The patient's condition is monitored in the Intensive Care Unit. Endoscopy is performed as soon as the patient is hemodynamically stable, for both diagnostic and therapeutic purpose. Hemodynamically stable patients undergo endoscopy as an initial diagnostic test, and therapeutic intervention to control bleeding. Portal pressure lowering agents are used in conjunction to endoscopic intervention to prevent early rebleeding. Patients who fail endoscopic and medical treatment are referred for transjugular intrahepatic portosystemic shunt (TIPS) placement or surgical treatment. Liver transplantation may be considered in selected cases for patients with end-stage liver disease.

INDICATIONS FOR ENDOSCOPIC INTERVENTION

Endoscopy is indicated for initial diagnosis as well as control of acute variceal hemorrhage. Both endoscopic sclerotherapy and endoscopic variceal ligation are highly effective in controlling initial episodes of esophageal variceal bleeding (7). Endoscopic intervention is used for the initial control of bleeding, and as treatment for the prevention of recurrent esophageal variceal hemorrhage. Pharmacological therapy with nonselective beta-blockers is added to endoscopic treatment for prevention of recurrent hemorrhage (8). At the present time, endoscopic therapy is not recommended for the primary prophylaxis of a variceal hemorrhage (9).

CONTRAINDICATIONS

Endoscopic intervention should not be performed if the patient is hemodynamically unstable, when perforated viscus is suspected, and if the patient is combative, or is unwilling to cooperate. Endotracheal intubation should be considered for the prevention of aspiration, and for the treatment of patients with severe agitation or encephalopathy.

ENDOSCOPIC SCLEROTHERAPY (EST)

Indications and Technique

Endoscopic sclerotherapy is being performed on an emergent basis to stop acute bleeding, and selectively to prevent rebleeding after control of initial episode is achieved. The goal of sclerotherapy is initial thrombosis and further obliteration of varices by injection of sclerosing agent.

EST is performed with a short 25-gage needle that is directed into the veins (intravariceal injection) or into the esophageal wall next to the variceal vein (paravariceal injection). Both techniques are effective but the intravariceal injection is utilized most commonly. Several sclerosants are available for EST, including 1% sodium tetradecyl sulfate, 5% ethanolamine oleate, 5% sodium morrhuate, and 0.5%–1% polidocanol (not available in United States). 1 to 2 mL of sclerosant is injected under direct vision into each varix, starting just above the gastroesophageal junction (GEJ) (Fig. 1). The procedure is repeated at higher levels up to 5 cm from the GEJ. The injections should not be made at higher levels to avoid spinal cord injury. A maximal dose of 20 mL per session is recommended to avoid complications. Occasionally, two or more injections are needed to control bleeding from a very large varix. Preferentially, the injection is made just below the point of bleeding, though a precise location of source is not always possible, especially in cases with a very brisk bleeding.

After initial control of hemorrhage is achieved, EST is repeated initially 1 wk after an acute episode of bleeding, and then in 2-wk intervals thereafter, until varices are eradicated.

EST is also being used to control active bleeding from gastric (fundic) varices. Endoscopic management of bleeding gastric varices is more difficult, and usually higher volumes of sclerosing agent or multiple injections need to be used to control hemorrhage.

Complications

EST is associated with local and systemic complications. The local complications like chest pain, transient dysphagia, odynophagia, and small pleural effusion are common, but usually minor and self-limiting. Mucosal ulcerations resulting from tissue necrosis are common and are seen in up to 70% of patients 1 wk after therapy (10). The deep esophageal ulcerations are an independent risk for bleeding. The tissue necrosis is also responsible for postprocedure esophageal perforation, which carries significant mortality. Esophageal stricture formation is relatively common, though clinically significant dysphagia occurs in about 15% of cases. Proton pump inhibitors are being used to prevent local complications and to improve tissue healing (11). Mediastinitis and pericarditis are less common and in part depend on the technique and amount of sclerosing agent being used.

Uncommon, but serious, systemic complications include aspiration pneumonia, systemic bacteriemia with risk for bacterial endocarditis and organ abscess, spinal cord paralysis, spontaneous bacterial peritonitis, and portal vein thrombosis. Aspiration during procedure and hypoxia can be prevented by use of elective intubation among patients at high risk. Infectious complications are prevented by prophylactic use of antibiotics when indicated.



Fig. 1. Endoscopic sclerotherapy. (A) Endoscope with retracted sclerotherapy needle. (B) Endoscope with extended sclerotherapy needle. (C) Injection of sclerosing agent into varix.

ENDOSCOPIC VARICEAL BAND LIGATION (EVL)

Indications and Technique

Endoscopic variceal ligation (EVL), also referred to as variceal banding, is an endoscopic therapy for acute esophageal variceal bleeding, and for elective eradication of varices after the initial episode of hemorrhage. EVL technique for the esophageal varices is similar to endoscopic treatment of rectal hemorrhoids. The ligation is accomplished by placement of an elastic band on the varix, which strangulates a blood vessel, resulting in vessel thrombosis. The thrombosed varix undergoes necrosis and sloughs off, to be replaced by fibrous tissue in the process of mucosal healing.

A small cylinder, which is preloaded with bands, is loaded onto the tip of the endoscope and the connecting wire is passed down the biopsy channel of the endoscope to be attached to a band-releasing device mounted into other end of that channel. Since the introduction of devices preloaded with multiple bands, there is no need for endoscope removal after each ligation, therefore, there is no need for overtube use. The endoscope with the device is placed over the varix, which is then suctioned into the device's plastic cylinder at the end of an endoscope. With the use of the trigger device, a ligating band is deployed. After the ligation process is completed, suction is stopped, and a puff of air is used to release the ligated varix from the device (Fig. 2). It is important to start ligation at the level of gastroesophageal junction and proceed proximally, because banded varices may obstruct esophageal lumen, making access to varices below them impossible. When ligating an actively bleeding varix, the band is placed directly over the bleeding point or just below it, but never above it, for the same reason. Typically, five to ten bands are placed in one session.



Fig. 2. Esophageal variceal ligation. (**A**) Banding device including cylinder with rubber bands. (**B**). Banding device is placed over the varix. (**C**) Suction is applied to aspirate varix into the cylinder. (**D**) Upon the release of the trip-wire, the rubber band strips off the cylinder and closes around the aspirated varix. (**E**) Ligated varix with endoscope removed.

After initial control of hemorrhage is achieved, the patient undergoes elective ligation 1 wk later and in 2-wk intervals thereafter for complete eradication of varices.

Complications

EVL has less potential for complications then EST (12). The ligation of varices causes mucosal ulceration in as many as 90% of patients at 1 wk, but because tissue injury is superficial and no sclerosing agent is being used, there is less potential for local complications like perforation and mediastinitis. The reduced incidence of esophageal stricture has also been reported (13). A less-tissue necrosis with the use of EVL provides theoretical ground for reduction of systemic infectious complications, which are related to the degree of bacteriemia during and postprocedure. EVL carries a similar risk for respiratory complications as EST does; therefore, the same precautions are needed. Proton-pump inhibitors are also being used to promote mucosal healing.

ENDOSCOPIC SCLEROTHERAPY WITH TISSUE ADHESIVE BUCRYLATE (HYSTOACRYL) (FIG. 3)

Standard endoscopic methods used in the treatment of esophageal varices have not been found effective for gastric varices. In the United States, at present, most patients with type II and III gastric variceal hemorrhage are treated with transjugular intrahepatic portosystemic shunt (TIPS). A new sclerotherapy technique for bleeding gastric varices, utilizing cyanoacrylate glue (Histoacryl) injection, was introduced in Europe. In the United States, Histoacryl is not approved by the FDA for clinical use.



Retroflexion of scope with needle advanced into gastric varix



Indications and Technique

Cyanoacrylate compound in a liquid form has a consistency similar to water; therefore, it is suitable for intravariceal injection with the use of a sclerotherapy needle. Upon injection into a varix, cyanoacrylate undergoes a polymerization process, which is triggered by its contact with a physiological fluid such as blood. In this process, cyanoacrylate glue is transformed from liquid into a solid form compound, which obliterates variceal lumen, and provides a rapid homeostasis of active bleeding. At present, there is no standardization of injection technique in treatment of varices. A sclerotherapy needle is being used as an injector (Huang). Histoacryl is being used in its undiluted form, as well as in dilution with Lipiodol, which delays the polymerization process and allows the operator more time to complete the injection and remove the needle. Undiluted Histoacryl undergoes instantaneous transformation into the solid compound; such a rapid process may not allow complete varix obliteration and, in addition, may cause clogging of the endoscope channel. The accessory channel needs to be lubricated with silicone oil prior to the procedure in order to prevent adherence of cyanoacrylate. Personnel participating in the procedure need to wear protective goggles to avoid eye injury.

Endoscopic sclerotherapy with use of cyanoacrylate was shown to be highly effective in the treatment of active hemorrhage from gastric varices, as well as in elective variceal obliteration (14). Term obliteration is used, rather then eradication of varices because varices are filled with a solid substance and are visible up to several weeks after completion of treatment.

Complications

Endoscopic sclerotherapy using cyanoacrylate glue, a tissue adhesive, was reported to be a safe procedure, but not free from serious complications. A main concern with the use of cyanoacrylate is the risk of embolization. Reports of cerebral stroke (15), pulmonary embolism (16), portal vein embolism, and splenic infarction (17) raised questions about the safety of this procedure. Additional complications in the form of visceral fistulas were also reported (18). Though embolic complications are rare in most series, they carry the risk for a significant morbidity and mortality.

COST EFFECTIVENESS OF AVAILABLE THERAPIES

Data on the cost of treating an episode of variceal bleeding and cost comparison of particular therapeutic modalities is limited. The difficulty in comparing the cost of commonly used therapies is a result of institutional and national differences in cost calculations between different healthcare models.

Medical therapy was shown to be the most cost-effective form of primary prophylaxis for esophageal variceal hemorrhage (19). Estimated cost of treatment for the prevention of recurrent variceal hemorrhage is the lowest for medical treatment and increases gradually for the endoscopic intervention and TIPS, with surgical treatment being the most expensive therapy (20). A recent multicenter prospective trial comparing the cost of EVL and EST at 1-yr follow-up showed similar costs between EVL and EST. The total median direct cost was \$9700 for EVL and \$13,200 for EST with a p value of 0.46 (21).

SUMMARY

- 1. Endoscopic treatment of esophageal variceal hemorrhage has become the standard of treatment in recent years. Both EST and EVL are highly effective in controlling active variceal bleeding and variceal eradication.
- 2. EVL is reported to have a lower rate of rebleeding, mortality, and complications, however the initial choice of treatment for active bleeding depends greatly on the experience of the endoscopist.
- 3. The rate of variceal bleeding and limited visibility during the procedure may dictate EST as an initial choice because it is easier to perform under such circumstances.
- 4. Treatment of gastric varices with a newer sclerotherapy agent, Histoacryl, is promising, but there are concerns over its safety and the product is not approved for use in the United States.
- 5. Endoscopic therapy is not recommended for primary prevention of variceal hemorrhage at present.

REFERENCES

- 1. Navarro VJ, Garcia-Tsao G. Variceal hemorrhage. Crit Care Clin 1995;11:391-414.
- 2. Wiliams SG, Westaby D. Fortnightly review: management of variceal hemorrhage. Br Med J 1994;308: 1213–1217.
- 3. Graham DY, Smith JL. The course of patients after variceal hemorrhage. Gastroenterology 1981;80: 800–809.
- 4. Garcia-Tsao G, Grace ND, Groszmann R, et al. Portal pressures, presence of gastroesophageal varices and variceal bleeding. Hepatology 1985;5:419–424.
- 5. Paquet KJ. Prophylactic endoscopic sclerosing treatment of the esophageal varices-a prospective controlled randomized trial. Endoscopy 1982;14:4-5.

- 6. Hashizume M, Sugimachi K. Classification of gastric lesions associated with portal hypertension. J Gastroenterol Hepatol 1995;10:339–343.
- 7. Kitano S, Baatar D. Endoscopic treatment for esophageal varices: will there be a place for sclerotherapy during the forthcoming era of ligation? Gastrointest Endosc 2000;52:226–232.
- Lo GH, Lai KH, Cheng JS, et al. Endoscopic variceal ligation plus nadolol and sucralfate compared with ligation alone for the prevention of variceal rebleeding: a prospective, randomized trial. Hepatology 2000;32:461–465.
- 9. The Veterans Affairs Cooperative Variceal Sclerotherapy Group. Prophylactic sclerotherapy for esophageal varices in men with alcoholic liver disease: a randomized, single-blind, multicenter clinical trial. N Engl J Med 1991;324:1779–1784.
- Sarin SK. Endoscopic sclerotherapy for esophago-gastric varices: a critical reappraisal. Aust N Z J Med 1989;19:162–171.
- 11. Gimson A, Polson R, Westaby D, et al. Omeprazole in the management of intractable esophageal ulceration following injection sclerotherapy. Gastroenterology 1990;99:1829–1831.
- 12. Laine L, El-Newihi HM, Migikovsky B, et al. Endoscopic ligation compared with sclerotherapy for the treatment of bleeding varices. Ann Intern Med 1993;119:1–7.
- 13. Laine L, Cook D. Endoscopic ligation compared with sclerotherapy for treatment of esophageal variceal bleeding. A meta-analysis. Ann Intern Med 1995;123:280–287.
- 14. Kind R, Guglielmi A, Rodella L, et al. Bucrylate treatment of bleeding gastric varices: 12 years' experience. Endoscopy 2000;32:512–519.
- 15. See A, Florent C, Lamy P, et al. Cerebrovascular accidents after endoscopic obturation of esophageal varices with isobutyl-2-cyanoacrylate in 2 patients. Gastroenterol Clin Biol 1986;10:604–607.
- Roesch W, Rexeoth G. Pulmonary, cerebral and coronary emboli during bucrylate injection of bleeding fundic varices. Endoscopy 1998;30:S89–S90.
- 17. Cheng PN, Sheu BS, Chen CY, et al. Splenic infarction after histoacryl injection for bleeding gastric varices. Gastrointest Endosc 1998;48:426–427.
- 18. Battaglia G, Morbin T, Patarnelo E, et al. Visceral fistulae as a complication of sclerotherapy for esophageal and gastric varices using isobutyl-2-cyanoacrylate. Gastrointest Endosc 2000;52:267–270.
- 19. Teran JC, Imperiale TF, Mullen KD, et al. Primary prophylaxis of variceal bleeding in cirrhosis: a costeffectiveness analysis. Gastroenterology 1997;112:473–482.
- 20. Sharara AI, Rockey DC. Gastroesophageal variceal hemorrhage. N Engl J Med 2001;345(9):669-681.
- 21. Gralnek I A, Jensen DM, Kovacs TO, et al. The economic impact of esophageal variceal hemorrhage: Cost effectiveness implications of endoscopic therapy. Hepatology 1999;29:44–50.

II GASTRIC SURGERY

Surgical Treatment of Peptic Ulcer Disease

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CONTENTS

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INTRODUCTION

The surgical treatment of peptic ulcer disease (PUD) has undergone a radical shift in the past 10 years. This is primarily because of the recognition that most gastric and duodenal ulcers are caused by *Helicobacter pylori* (*H. pylori*). The recognition that PUD is an infectious problem, rather than a problem with excess acid production, and can be definitely cured, has rendered most peptic ulcer operations obsolete. Eradication of the *H. pylori* eliminates recurrence of the ulcer disease, whereas healing an ulcer with antacid therapy alone results in a 70–80% recurrence rate. Thus, most surgical literature prior to the recognition of *H. pylori* was based on controlling acid production is no longer relevant to modern day ulcer surgery.

This chapter will focus on the indications and operative techniques for gastroduodenal ulcer disease. The traditional understanding and surgical treatment of ulcer disease will be reviewed. The current approach to complications of ulcer disease will be detailed. The few areas where elective surgical treatment is applicable for PUD will be discussed. Finally, the complications and cost effectiveness of ulcer surgery will be reviewed.

OPERATIVE TECHNIQUES

The evolution of operations for ulcer disease has progressed as surgical techniques and understanding of gastric physiology advanced. Gastrojejunostomy and subtotal

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Fig. 1. Gastroenterostomy. An anastomosis is made on the dependent portion of the greater curvature of the antrum The direction of peristalsis of the stomach matches the small bowel (isoperistaltic anastomosis) to enhance gastric emptying. The vagus nerves are intact.

gastrectomy were initially used in the treatment of gastric cancer, but their value in treating ulcer disease was quickly appreciated. Gastrojejunostomy (Fig. 1) was thought to decrease overall gastric acidity and decrease acid exposure by accelerating gastric emptying. As the procedure was technically simple and was effective in the short term, it became the treatment of choice for ulcer disease in the early part of the 20th century. As the high incidence of recurrent ulceration with gastroenterostomy became appreciated, subtotal gastrectomy gradually became the operation of choice (Fig. 2).

The association of gastric hyperacidity with ulcer disease has been recognized since the 19th century. As the role of the vagus nerve in gastric secretion became better understood, truncal vagotomy was introduced into gastric surgery. Vagotomy alone was found to have an ulcer recurrence rate similar to subtotal gastrectomy, but the mortality rate was much higher after subtotal gastrectomy, so vagotomy was accepted as a safer alternative. In many patients, truncal vagotomy disrupted pyloric function resulting in gastric outlet obstruction, and the need for a drainage procedure. Thus, pyloroplasty was routinely added to vagotomy. Vagotomy and pyloroplasty became the operation of choice in the 1940s (Fig. 3).



Fig. 2. Subtotal gastrectomy with Billroth II anastomosis. The anastomosis is isoperistaltic and no vagotomy done. The afferent limb is kept short to prevent kinking.



Fig. 3. Truncal vagotomy and pyloroplasty. (A) A 5-cm gastroduodenotomy is made across the pylorus. (B) The incision is closed transversely (Heineke-Michulicz pyloroplasty) and truncal vagotomy accomplished.



Fig. 4. Truncal vagotomy and antrectomy with Billroth I anastomosis. (A) A distal 40-50% of the stomach is removed, taking more of the lesser curvature where the antrum extends more proximal. (B) A gastroduodenotomy is done on the greater curvature side of the gastric remnant and truncal vagotomy accomplished.

With the identification of gastrin and its role in acid secretion, the concept of antrectomy and vagotomy emerged. By removing both the gastrin and vagal stimulation of the partial cells, acid output could be greatly decreased. Vagotomy and antrectomy resulted in recurrent ulcer rates less than 1%. With improved operative techniques the mortality rate of a gastric resection was decreasing, and by 1970, vagotomy and antrectomy was considered the best operation for ulcer disease (Fig. 4). An important randomized study compared vagotomy and pyloroplasty, subtotal gastrectomy, and vagotomy and antrectomy found that recurrent ulcer was least with vagotomy and antrectomy but postgastrectomy side effects were greatest (1). This study helped to define how to best treat ulcer disease. With the young patients having the greatest threat of recurrence, vagotomy and antrectomy was used. With the elderly, and those with comorbidities, having the greatest threat of postgastrectomy syndrome, vagotomy and pyloroplasty was used.

Although vagotomy and antrectomy solved the problem of recurrent ulceration, postgastrectomy syndromes resulted in significant morbidity in 25% of the patients. Vagotomy decreased gastric contractions and also obliterated the reflex of the stomach to dilate with smell or eating food. Ablation of the pylorus resulted in uncontrolled emptying of the stomach, and loss of a barrier to bile entering the stomach. The postgastrectomy syndromes of dumping, alkaline reflux gastritis, and gastric stasis resulted, and proved to be a high price to pay for cure of ulcer disease.

The next evolution of ulcer surgery came when a way to decrease gastric acid secretion and yet preserve the pylorus was found. This advance was the development of proximal gastric vagotomy (Fig. 5) (2). This operation allowed denervation of the acid



Fig. 5. Proximal gastric vagotomy. The branches of the anterior and posterior vagus nerves to the portion of the stomach with parietal cells are divided. This preserves normal antropyloric function and obviates the need for a gastric drainage procedure. (A) Normal stomach. (B) After proximal gastric vagotomy. Shaded area = vagal denervation.

producing parietal cells, but the grinding function of the antrum and the emptying ability of the pylorus are preserved. Proximal gastric vagotomy (also called highly selective vagotomy or parietal cell vagotomy) was technically more demanding, and high recurrent ulcer rates were seen until the procedure was standardized. Once all the nuances of the procedure were appreciated, recurrent ulcer rates were 10% or less and postgastrectomy side effects were much less than those seen with vagotomy and pyloroplasty (3). Proximal gastric vagotomy was not as effective for gastric ulcers and was not used by most surgeons for this indication.

CURRENT SURGICAL TREATMENT OF ULCER COMPLICATIONS

Hemorrhage

The primary treatment of bleeding ulcers is endoscopic control followed by treatment for H. pylori if present. Even rebleeding is best treated by repeat attempts at endoscopic control (4). Surgery is indicated for significant bleeding (requiring over five units of blood) that cannot be controlled by endoscopy. Most uncontrolled bleeding ulcers are from the gastroduodenal artery in the posterior aspect of the duodenal bulb. Treatment is by duodenotomy, and ligation of the bleeding site (Fig. 6). The integrity of the pylorus should be preserved. Gastric ulcers should be treated with ulcer excision if amenable. Ulcers located in regions difficult to excise (cardia, prepyloric) should be biopsied and oversewn. Occasional large or penetrating ulcers may be best treated with distal gastrectomy for technical considerations or to rule out cancer.



Fig. 6. Controlling bleeding from gastroduodenal artery. (**A**) A longitudial duodenotomy is made distal to the pylorus. (**B**) The gastroduodenal artery in the posterior duodenal bulb ulcer is oversewn. (**C**) The duodenotomy is closed longitudinally.

Certain patients, such as those with arterial bleeding or a visible vessel on endoscopy, are at high risk for rebleeding. Although considered to be surgical indications in past, this is no longer the case. Improved endoscopic techniques and the difficulty of identifying the bleeding risk of individual patients have eliminated the rationale for operating in these patient groups. In *H. pylori* positive patients, treatment of the *H. pylori* is highly effective in preventing rebleeding. In *H. pylori* negative patients, the rebleeding rate is only 10–20%, which is too low to justify an elective surgery to prevent rebleeding. Should rebleeding occur in *H. pylori* negative patients, oversewing of the bleeder (Fig. 6) and proximal gastric vagotomy can be justified for duodenal ulcers (Fig. 5). In general, surgery has been relegated to controlling ulcer hemorrhage and not treating the ulcer disease.

Perforation

Patients with ulcer perforation should be assumed to be H. pylori positive unless there is evidence to the contrary. Duodenal ulcers and prepyloric gastric ulcers should be treated with omental patches only (Fig. 7). A laparoscopic approach offers a slight decrease in morbidity if such expertise is available. Some controversy exists regarding the treatment of gastric ulcers, especially in the antrum and body of the stomach. If feasible, wedge resection and closure of the defect is best as it rules out malignancy. Perforation that is not amenable to wedge resection requires a distal gastrectomy with inclusion of the ulcer. Reconstruction with a Billroth I (Fig. 4) is the recommended reconstruction as it will result in fewer postgastrectomy side effects.





Gastric Outlet Obstruction

Gastric outlet obstruction has nearly disappeared in the western world. Ulcers causing pyloric obstruction should be initially treated with endoscopic dilatation and treatment of *H. pylori* if present. Multiple endoscopic dilations may be needed. This will eventually be successful more than 50% of the time (5). In the remaining patients, surgery is indicated. Truncal vagotomy with gastroenterostomy or antrectomy are both acceptable procedures. Pyloroplasty should not be done because of the increased risk of suture line leak from the fibrotic pylorus, and because patients do not have as good long term outcome (6). As truncal vagotomy and gastroenterostomy are easily accomplished laparoscopically, this will likely become the procedure of choice.

COMPLICATIONS AND MANAGEMENT OF PEPTIC ULCER SURGERY *Early Complications*

The generic complications seen with gastric surgery are hemorrhage and infection. The most feared complication is suture line leakage, especially at the duodenal stump. Mechanical complications include anastomotic obstruction, jejunal volvulus, and afferent loop and efferent loop syndrome. Postoperative ileus can result in acute gastric dilatation. Gastric necrosis is unusual, but is occasionally seen (1 in 400 cases) with proximal gastric vagotomy owing to the extensive devascularization of the lesser curvature.

Significant postoperative hemorrhage should be managed with reoperation and control of bleeding. If the bleeding is intraluminal, endoscopy and coagulation or clipping may obviate reoperation. Care must be taken to minimize pressure on the suture line to prevent a suture line dehiscence. Delayed gastric emptying is a common annoying complication. This is generally a functional problem and resolves over time in most patients. Delayed gastric emptying can also be caused by stomal obstruction, usually from swelling or hematoma, and resolves within 2 wk. Early obstructive problems, not due to the anastomotic site, are best approached with early reoperation. Acute gastric dilatation is treated with gastric decompression until gastric ileus is resolved. Gastric necrosis requires reoperation and repair. Suture line leaks are generally treated with percutaneous drainage, antibiotics, and parenteral nutrition, in certain situations reoperation is necessary. Any significant abscesses as a result of the operation or to complications must be drained, preferably percutaneously.

Late Complications

The late complications of gastric surgery are referred to as postgastrectomy syndromes. The most clinically significant of these are dumping, alkaline reflux gastritis, and gastric stasis. Less clinically significant are small stomach syndrome, postvagotomy diarrhea, and the afferent and efferent loop obstruction. Nutritional side effects include anemia (primary iron deficiency and B_{12} deficiency), malabsorption, and vitamin deficiencies.

Dumping can generally be managed with dietary intervention. Occasional patients will require reoperation where Roux-en-Y gastrojejunostomy is the treatment of choice. Alkaline reflux gastritis responds poorly to medical management. Patients with significant symptoms require revisional gastric surgery. Because gastric stasis is often a component of the etiology of alkaline reflux gastritis, subtotal or near total gastrectomy in addition to the Roux-en-Y gastrojejunostomy should be done. Gastric stasis is best managed with near total gastrectomy. A more extensive discussion of postgastrectomy syndromes can be found in the chapter on reconstruction after distal gastrectomy.

Recurrent Ulcers Following Definitive Ulcer Surgery

Except for total gastrectomy, all ulcer operations carry some risk of ulcer recurrence. This varied from a 1% risk with vagotomy and antrectomy (Fig. 4) to greater than 50% risk for gastroenterostomy (Fig. 1) over 10 yr. Surgical treatment for recurrent ulcers in the past consisted of vagotomy or revagotomy, generally combined with further gastric resection. Results were unpredictable, reflecting the lack of understanding of *H. pylori*. Enigmas such as the higher incidence of recurrent ulceration with Billroth I vs Billroth II reconstruction could not be explained.

The understanding of the role of *H. pylori* in ulcer disease has resolved much of the confusion. The presence of bile in the stomach decreases the incidence of *H. pylori*. Thus, the greater presence of bile in the stomach after Billroth II likely resulted in less *H. pylori* and a lowered ulcer recurrence rate compared to a Billroth I or vagotomy and pyloroplasty. *H. pylori* is present in more than 90% of patients following proximal gastric vagotomy, which helps explain the high recurrence rate with this procedure. The Roux-en-Y gastrojejunostomy diverts bile from the stomach and is often used in revisional gastric surgery. The high ulcer recurrence rate after Roux-en-Y gastrojejunostomy may also be partly because of *H. pylori*, as the *H. pylori* infection rate increases following diversion of bile away from the stomach.

This new knowledge gives new approaches to treatment in patients with recurrent ulcer disease after gastric surgery. Patients with recurrent ulcers after proximal gastric vagotomy, vagotomy and pyloroplasty, or Billroth I reconstructions, are likely because of *H. pylori*. The eradication of the *H. pylori* will be curative. Ulcer recurrence after Billroth II reconstruction is not likely a result of *H. pylori* (7). The problem in this situation likely results from poor gastric emptying either from a mechanical or functional defect (8). In this case, medications will not be effective. Revision of the anastomosis is needed for mechanical obstruction. However, most gastric stasis following gastric surgery is on a functional basis. Thus, gastric stasis, not clearly because of mechanical causes, is best treated with near total gastrectomy.

SPECIAL CONSIDERATIONS IN ULCER SURGERY

Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

Symptomatic ulcers will develop in 2% of patients on NSAIDs for 1 yr, at least a fourfold increase over the risk in the general population. The relationship of NSAIDs to H. pylori is not well defined, but NSAIDs are clearly an independent risk factor. NSAID ulcers appear to be more likely to perforate and up to 50% of perforated ulcers are associated with NSAIDs. Cost-effective ulcer prophylaxis exists for NSAIDs (9). In addition, specific COX2 inhibitors appear to greatly decrease the risk of ulcers (10). Because of these factors, there is currently no role for elective ulcer surgery for patients on NSAIDs. Furthermore, ulcer complications should not have more aggressive surgery because patients are on NSAIDs.

Low-dose aspirin is commonly used to prevent vascular diseases. It slightly increases the risk of ulcer disease $(1.3 \times \text{the baseline})$. Surgical intervention to prevent ulcer complications of low dose aspirin is not indicated.

Smoking

Cigarette smoking is detrimental to mucosal protective mechanisms and increases the likelihood that gastric ulcers will develop. Although these ulcers are generally amenable to ulcer treatment, they can occasionally be refractory to healing. This can be a difficult clinical problem in a patient with symptoms. If multiple biopsies have been negative for cancer, it is highly unlikely to be a malignant ulcer. As the ulcers will heal with cessation of smoking, this is the treatment of choice. The surgeon should be careful about offering a definitive ulcer operation in this patient group. Symptoms are often times not eliminated, and postgastrectomy complications are high.

Zollinger Ellison Syndrome (ZE)

When ZE syndrome was first described, the treatment of choice was a total gastrectomy. As surgical treatment evolved, it became apparent that it was more effective to remove the gastrinoma than the stomach. In patients where the gastrinoma cannot be cured surgically, antacid medications (proton pump inhibitors) are adequate to prevent ulceration. In the rare patients where proton pump inhibitors are not acceptable, proximal gastric vagotomy is the surgical treatment of choice (Fig. 5). Proximal gastric vagotomy decreases the need for antacid therapy and may be cost effective for patients with unresectable ZE (11). Total gastrectomy for ZE is of historic interest only.

Giant Peptic Ulcer

Giant peptic ulcers have traditionally been associated with a high complication rate and have been treated surgically. This is yet another area where surgical principles have changed. With treatment of *H. pylori*, and modern acid suppression, most giant ulcers can be healed medically (12). Surgery is only indicated for complications of the ulcer disease.

Stress Gastritis

The incidence of stress related erosive gastritis has decreased dramatically in the past 20 yr because of antacid prophylaxis. For the occasional patient that has uncontrolled bleeding from stress gastritis, endoscopy is ineffective. The treatment of choice is high-dose proton pump inhibitors. Only rarely is surgery required, and then a near total gastrectomy is necessary.

COST EFFECTIVE SURGERY IN PUD

PUD can generally be cured with antibiotics to treat *H. pylori*. This advance has greatly decreased the need for elective surgery and overall cost of treating PUD. In those few patients that require an elective acid reduction procedure, proximal gastric vagotomy will be most cost effective by avoiding most postgastrectomy syndromes.

Ulcer hemorrhage can now be treated by endoscopy in most situations. Aggressive use of endoscopy decreases length of stay and possibly the need for surgery (13). If surgery is required, ligation of the bleeding is done with minimal disturbance of gastroduodenal anatomy and physiology. Recovery is rapid and length of stay decreased by avoiding gastrectomy.

Duodenal ulcer perforations are best treated with omental patches as they are faster, have a lower surgeon's fee, and result in a shorter length of stay than a definitive ulcer operation (Fig. 7). The omental patches are highly effective and ulcer disease can usually be treated postoperatively with antibiotics. Gastric perforation often requires a wedge resection or even gastrectomy. This increases cost and length of stay.

For patients with gastric outlet obstruction, vagotomy and gastroenterostomy can be done laparoscopically. Length of stay is determined more by the dilatation and poor function of the stomach rather than the operative procedure.

The most difficult decisions in ulcer disease concern revisional gastric surgery for recurrent ulcers and postgastrectomy syndromes. Most revisional gastric surgery is done at least in part because of poor motility of the gastric remnant. It is generally best to do a definitive (near total gastrectomy with Roux-en-Y gastrojejunostomy) operation at the first revisional gastric surgery. This has the advantage of preventing future revisional operations in a difficult operative field. The disadvantage is that small stomach syndrome is created in all patients. Most patients adapt eventually, a few develop Roux stasis syndrome and have difficulty maintaining their nutrition. However, these patients with Roux stasis syndrome would likely have the same problem or worse with lesser revisional surgery. The long-term solution to postgastrectomy syndromes is to avoid gastrectomy or pyloroplasty in the initial surgical treatment of ulcer disease.

SUMMARY

1. The discovery of *H. pylori* as the etiologic agent in most patients with gastroduodenal ulcers has had a huge impact on the surgical approach to PUD. Past treatment strategies were illogical and harmful.

- 2. Surgery needed for ulcer hemorrhage and perforation should be limited to treatment of the complication and later cure of the ulcer disease by *H. pylori* eradication.
- 3. Gastric outlet obstruction from ulcer disease has decreased greatly in incidence, and can often be treated with dilatation. If this fails, vagotomy and gastroenterostomy is the treatment of choice.
- 4. There is little role for the elective treatment of ulcers, even in patients taking NSAIDs, smokers, or with giant peptic ulcers.
- 5. Elective ulcer surgery may be indicated in rare patients with persistent *H. pylori* despite antibiotic treatment, *H. pylori* negative ulcers, and ZE syndrome, and are usually best treated with proximal gastric vagotomy to avoid postgastrectomy syndromes.
- 6. Stress gastritis and postgastrectomy ulcers requiring surgery will usually require near total gastrectomy.

REFERENCES

- 1. Goligher JC, Feather DB, Hall R, et al. Several standard elective operations for duodenal ulcers: Ten to sixteen year clinical results. Ann Surg 1979;189:18–24.
- Johnston D, Wilkinson AR. Highly selective vagotomy without a drainage procedure in the treatment of duodenal ulcer. Brit J Surg 1970;57:289–296.
- 3. Chan VM, Reznick RK, O'Rourke KK, et al. Meta-analysis of highly selective vagotomy versus truncal vagotomy and pyloroplasty in the surgical treatment of uncomplicated duodenal ulcer. Can J Surg 1994;37:457–464.
- 4. Lau JY, Sung JJ, Lam YH, et al. Endoscopic retreatment compared with surgery in patients with recurrent bleeding after initial endoscopic control of bleeding ulcers. N Engl J Med 1999;340:799–801.
- 5. Lau JY, Chung SC, Sung JJ, et al. Through-the-scope balloon dilation for pyloric stenosis: Long-term results. Gastrointest Endosc 1996;43:98–101.
- 6. Csendes A, Maluenda F, Braghetto I, et al. Prospective randomized study comparing three surgical techniques for the treatment of gastric outlet obstruction secondary to duodenal ulcer. Am J Surg 1993; 166:45–49.
- Lee YT, Sung JJ, Choi CL, et al. Ulcer recurrence after gastric surgery: Is Helicobacter pylori the culprit? Am J Gastroenterol 1998;93:928–931.
- 8. Browder W, Thompson J, Youngberg G, et al. Delayed ulcer recurrence after gastric resection: A new postgastrectomy syndrome? Am Surg 1997;63:1091–1095.
- 9. Ko CW, Deyo RA. Cost-effectiveness of strategies for primary prevention of nonsteroidal anti-inflammatory drug-induced peptic ulcer disease. J Gen Intern Med 2000;15:400–410.
- Silverstein FE, Faich G, Goldstein JL, et al. Gastrointestinal toxicity with celecoxib vs nonsteroidal anti-inflammatory drugs for osteoarthritis and rheumatoid arthritis: The CLASS study: A randomized controlled trail. JAMA 2000;284:1247–1255.
- 11. McArthur KE, Richardson CT, Barnett CC, et al. Laparotomy and proximal vagotomy in Zollinger-Ellison syndrome: Result of a 16-year prospective study. Am J Gastroenterol 1996;91:1105–1111.
- 12. Simeone DM, Hassan A, Scheiman JM. Giant peptic ulcers: A surgical or medical disease? Surgery 1999;126:474–478.
- 13. Cooper GS, Chak A, Way LE, et al. Early endoscopy in upper gastrointestinal hemorrhage: Associations with recurrent bleeding, surgery, and length of hospital stay. Gastrointest Endosc 1999;49:145–152.

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INTRODUCTION

Gastric cancer remains the second most common cancer in the world, with an estimated 1 million new cases annually (1). During this same time period, deaths from gastric cancer will approach 840,000.

The risk factors for gastric cancer have been well documented to include a diet high in salt and smoked foods, gender (male), as well as atrophic gastritis. Race (African-American) and low socioeconomic status are commonly associated with a higher prevalence of gastric cancer, but are probably not independent risk factors. *Helicobacter pylori (H. pylori)* gastric infection is currently the most heavily studied potential carcinogen for gastric cancer. This association of *H. pylori* with gastric cancer is primarily based on epidemiological, and prospective follow-up data that demonstrates an increased risk of gastric cancer in patients who have *H. pylori* infections. The risk is estimated to be about 0.5% per year. Individuals who have had prior gastrectomy also have been reported to have a greater incidence of so-called gastric stump cancer. New data suggests that *H. pylori* related gastric ulcers, but not duodenal ulcers, are associated with gastric cancers. The appearance of gastric stump cancer occurs 15 yr or more after the primary resection, with an overall risk that is 0 to 5 times higher than in individuals without previous gastric resections.

A family history of gastric cancer diagnosed at an early age (< 40 yr old) and/or a known history of Hereditary Non-polyposis Colon Cancer (HNPCC) have also been demonstrated to increase the risk of gastric cancer in future generations. These cri-

From: *Clinical Gastroenterology: An Internist's Illustrated Guide to Gastrointestinal Surgery* Edited by: George Y. Wu, Khalid Aziz, and Giles F. Whalen © Humana Press Inc., Totowa, NJ teria define a population of patients who have a greater risk of developing carcinoma of the stomach.

PATIENT SELECTION

The primary therapy for gastric cancer is surgical resection. The type of surgical resection depends primarily on the location of the primary lesion, the size of the primary lesion, and the overall stage of disease at presentation. Advances in computed tomography (CT), endoscopy staging, as well as greater use of laparoscopy have allowed both the surgeon and the gastroenterologist to tailor treatment based on the stage of disease. Laparoscopy remains the single most sensitive means of detecting peritoneal and small hepatic metastases.

A greater awareness of upper-gastrointestinal (GI) tract cancers, increased use of endoscopy, as well as the increasing ability to identify subtle signs of gastric mucosal cancers, have resulted in a greater number of patients being diagnosed at an earlier stage of disease. Patients with small (< 3 cm) mucosal (T1a) lesions (Table 1 and 2) favorable endoscopic appearance (flat and no ulceration), and well-differentiated histology can be effectively treated with endoscopic mucosal resection (EMR) (2). This technique has been applied with increasing frequency in Japan for the treatment of favorable mucosal lesions.

The two most common techniques for gastric EMR are the grasp-and-pull and the cupand-suction method (3). The grasp-and-pull technique utilizes a double-channel endoscope, and after careful marking of the planned margins of resection, the submucosa is injected with saline or sodium hyaluronate to raise the submucosal from the muscularis. The raised lesion is then grasped and pulled into the channel, and a snare is placed at the base of the lesion. The mucosa is then resected and the margins are then carefully evaluated to ensure complete resection.

The cup-and-suction method requires only a single-channel endoscope. After marking the margins, the lesion is raised with a submucosal injection. A clear cap is attached to the end of the scope and the lesion is then aspirated into the cup with the placement of a snare at the base of the lesion. The aspiration is then released, allowing for evaluation of the lesion to ensure the snare encompasses the entire margin, and the lesion is then resected. Again, careful evaluation after resection is indicated to ensure the lesion is superficial and that all margins were excised.

This trend in treating patients with early gastric cancer with minimally invasive techniques originated from studies demonstrating that mucosal cancer with the earlier favorable characteristics have a very low incidence of lymph node metastasis. Patients with small mucosal lesions demonstrating these favorable histologic characteristics have been found to have a lymph node metastasis rate of 0% to 5% (4–8).

The treatment of more invasive early gastric cancer (T1b) has also been redefined to take advantage of the benefits of laparoscopic approaches to resect the primary with an adequate dissection of the perigastric N1 nodes. In the case of large (> 4.5 cm) poorly differentiated tumors invading into the submucosa, the risk of N2 nodal involvement is sufficiently high to include these nodes in the resection (7-10). We perform a D2 lymphadenectomy in the latter scenario case because cure is still possible for T1 tumors with N2 nodes (7,8).

More invasive cancers, defined by endoscopic ultrasound as invading the muscularis mucosa or deeper, which continues to be the most common presentation in Western

Primary tumor (T)	Regional lymph nodes (N)	Distant metastasis (M)
TX: Primary tumor cannot be assessed	NX: Regional lymph node(s) cannot be assessed	MX: Distant metastasis cannot be assessed
T0: No evidence of primary	N0: No regional lymph node metastasis	M0: No distant metastasis tumor
Tis: Carcinoma <i>in situ</i> : Intraepithelial tumor without invasion of lamina propria	N1: Metastasis in 1 to 6 lymph nodes	M1: Distant metastasis
T1: Invasion lamina propria/ submucosa	N2: Metastasis in 7 to 15 lymph nodes	
T2: Invasion muscularis propria/subserosa	N3: Metastasis in more than 15 regional lymph nodes	
T3: Penetrates serosa w/o invasion of adjacent structures		
T4: Invades adjacent structures	4	

Table 1 TMN Classification for Gastric Cancer

Table 2 Staging of Gastric Cancer						
Stage 0:	Tis	N0	M0			
Stage IA:	T1	N0	M0			
Stage IB:	T1	N1	M0			
-	T2	N0	M0			
Stage II:	T1	N2	M0			
-	T2	N1	M0			
	T3	N0	M0			
Stage IIIA:	T2	N2	M0			
e	T3	N1	M0			
	T4	N0	M0			
Stage IIIB:	T3	N2	M0			
Stage IV:	T4	N1,2	M0			
e	Any T	N3				
	Any T	Any N	M1			

centers, should be treated with a formal resection and adequate lymph node dissection. Recent changes in the American Joint Committee on Cancer (AJCC) staging guideline have mandated that a minimum of 15 lymph nodes must be removed and examined (Table 1). A recent report from Memorial Sloan Kettering Cancer Center (MSKCC) Department of Surgery have emphasized the importance of adhering to these requirements for accurate nodal staging (9,10). This has placed greater emphasis on achieving an adequate lymphadenectomy to standardize staging worldwide. The presence of lymph node metastases is a powerful prognostic factor. The more complete the lymph node dissection, the better the staging. With accurate staging, sound decisions regarding the need for postoperative adjuvant therapy can be made. The scope of a D1 lymphadenectomy or an extended D2 lymphadenectomy has been well described (Fig. 1).



Fig. 1. The defined lymph nodes for resection of gastric lesions within the distal, middle, and proximal stomach. (A) Proximal stomach: paraesophageal, left gastric, hepatic artery, celiac, suprapyloric, and infrapyloric, splenic arery, splenic hilum. (B) Middle stomach: left gastric, hepatic artery, celiac, left cardia, splenic arery, splenic hilum. (C) Distal stomach: left gastric, hepatic artery, celiac, right cardia.

The mean number of nodes evaluated by the pathologist at MSKCC was 14 following a D1 (limited) lymphadenectomy, 18 for D1+ (resecting some N2 nodes), and 26 for the standard D2 (extended). The D2 lymphadenectomy satisfies all staging requirements and can be done safely in experienced hands by avoiding pancreaticosplenectomy (11,12). The D2 lymphadenectomy has not resulted in better overall survival in the randomized data (13). Follow-up data from the Dutch trial presented at the 4th International Gastric Cancer Congress in April 2000, suggested that for node-positive patients, there is a survival advantage following a D2 dissection for the subset of T2N1 and T3N2 gastric cancer patients. A similar observation was made in 1998 by the German Gastric Cancer Study Group (14,15). A reasonable minimum approach to the lymphadenectomy would be to ensure that all perigastric nodes adjacent to the primary (D1) are removed and extending that to include the left gastric artery nodes for all advanced cancers. Taking the left gastric artery at its origin removes the site where N2 lymph node involvement is most frequently located (16).

This chapter will review the technical aspects of a proper surgical resection for gastric cancer.

TOTAL GASTRECTOMY

The first successful total gastrectomy (TG) for cancer was performed in 1897, 16 yr after Billroth's historic pylorectomy, by Swiss surgeon Carl Schlatter. This procedure involves removing the entire stomach from the distal esophagus to the proximal duodenum. TG is performed in many major medical centers for the treatment of gastric cancer. The primary indications for TG is to obtain a minimum of a 5-cm proximal margin for large tumors or the body, fundus and /or cardia of the stomach, or with tumors growing in a diffuse pattern (linitis plastica). The proximal margin length is best determined and should be recorded at the initial endoscopy. This is particularly important for lesions arising from the lesser curve.

After diagnostic endoscopy, a CT of the abdomen must be done to rule out the presence of metastatic disease, and to evaluate the possible extent of resection. The presence of metastatic disease is a relative contraindication to performing a TG because most patients can be palliated with chemotherapy.

After removing the entire stomach, reconstruction with the proximal jejunum creating a Roux-en-Y limb (Fig. 2) is the most commonly performed technique. Common variations seen with reconstruction involve the construction of a jejunal pouch. Improvements in oral intake and better weight gain have been attributed to using a pouch. There have been at least six randomized controlled trials evaluating various surgical reconstruction techniques following total gastrectomy (17-22), and no clear conclusion can be drawn regarding the optimal reconstruction technique. These trials suffer from either small sample size, varied use of nonstandard quality of life indexes, and/or they lack appropriate controls. Efforts continue to design simple, effective, and physiologic means of reconstructing the GI tract.

Postoperative radiographic evaluation (Fig. 3) is commonly performed to rule out an anastomotic leak (Fig. 4) between the fifth and seventh postoperative day. Because clinical signs will usually precede a clinically significant anastomotic leak in most instances, a gastrografin swallow can be used selectively to confirm clinical suspicion.



Fig. 2. Common reconstruction technique with a Roux-en-Y limb. After total gastrectomy jejunal pouch is constructed and connected to esophagus.

COMPLICATIONS

The more common major perioperative complications seen after total gastrectomy are pneumonia and leakage of the anastomosis. Anastomotic leakage is seen more frequently following an esophageal anastomosis than gastrojejunostomy because the esophagus lacks the strength of a serosa. In a review of 724 patients seen at MSKCC who underwent resection that required an esophageal anastomosis, the overall leak rate was 7%. Other major complications include cardiac dysrhythmias, pulmonary embolism, or bleeding.

The long-term consequences of total gastrectomy are related to an inability to absorb sufficient nutrition to gain weight. A large majority of patients undergoing total gastrectomy will lose between 5% and 10% of their overall body weight in the immediate postoperative period (6–12 wk). The weight loss is multifactorial, but in part is related to limited intake because of early satiety and/or increased losses from diarrhea. This can be effectively treated with diet modifications of frequent small, high-calorie meals, and avoiding fatty meals. Symptoms of fat intolerance can develop, which often respond to pancreolipase supplementation.

Dumping syndrome, which is the rapid emptying of undigested food into the intestine and small bowel immediately after a meal, can lead to nausea, emesis, bloating, and diarrhea. Delayed symptoms, including weakness and perspiration, can occur approx 2– 4 h after a meal. The most effective therapy for dumping syndrome is a diet of small, frequent meals that are low in simple carbohydrates, and the avoidance of drinking liquids with meals. Antidiarrhea agents may also be useful.

Another complication following total gastrectomy is stricture formation at the esophagojejunostomy anastomosis. This may arise after a small leak has healed second-



Fig. 3. Normal gastrografin swallow after total gastrectomy.

arily. A tightly stapled or hand-sewn anastomosis or ischemia at the anastomosis can lead to a stricture. Symptoms can present as dysphagia 1–6 wk following total gastrectomy. Almost all of these strictures can be treated with endoscopic dilations. The number of dilations is variable among patients, but two to three dilations are not uncommon.

A less common consequence of total gastrectomy is bile reflux. This can be effectively treated with Sucrafate. One of the primary reasons for this complication is construction of a short Roux-en-Y limb that is less than 45 cm in length. Creating a Roux-en-Y limb of adequate length essentially eliminates this problem.

PARTIAL GASTRECTOMY

A partial gastrectomy for gastric cancer is performed either in the form of a proximal subtotal gastrectomy or a distal subtotal gastrectomy. The proximal gastrectomy is a procedure that has increased in frequency as the incidence of small early stage proximal gastric cancer increases. The decision to perform either of these procedures is dependent on the location of the primary lesion. Early (T1b) gastric cancers and small advanced lesions located in the proximal one-third of the stomach (Fig. 5) can be treated with a proximal resection, provided that a generous gastric pouch remains. Lesions located in the distal two-thirds of the stomach (Fig. 6) are often treated with a distal subtotal resection.


Fig. 4. Anastomotic leak demonstrated on gastrografin swallow following total gastrectomy.

The preoperative radiographic evaluation for these patients is the same as for patients who require a total gastrectomy, and consists of a CT of the abdomen to rule out metastatic disease and to evaluate the possible nodal involvement. Once metastatic disease has been ruled out and the primary lesion can be resected with a partial gastrectomy, this is performed with a gastrojejunostomy reconstruction (Fig. 7).

COMPLICATIONS

The types of perioperative complications with a proximal gastrectomy or distal gastrectomy have several similarities and differences. The primary difference between the two types of partial gastrectomy (distal vs proximal) is the anastomotic leak rate. Patients who have undergone a proximal gastrectomy have a much higher leak rate (11.5%) compared with patients who have undergone a distal gastrectomy (< 1%) and has been reported to be even higher than for a total gastrectomy. Proximal gastrectomy is reputedly associated with profound bile reflux. This is more related to the size of the gastric remnant than simply the operation. With a patulous gastric remnant, and an anastomosis that sits in the abdomen, much of the debilitating symptoms of bile reflux can be avoided. Other common complications are related to pulmonary dysfunction following a major abdominal surgery.



Fig. 5. A proximal one-third gastric cancer.



Fig. 6. A primary gastric cancer of the antrum.

The long-term consequences of partial gastrectomy are related to impaired motility. Impaired motility can present with poor emptying of the Roux-en-Y limb, which can lead to early satiety or emesis. This dysmotility can be treated with prokinetic agents such as Reglan or erythromycin with moderate success; the vast majority of patients will improve



Fig. 7. Reconstruction of a distal gastrectomy with a gastrojejunostomy.

in a short period (3–4 wk) of time with conservative management. Another symptom of dysmotility is reflux esophagitis, because of delayed emptying, which can be effectively treated with Sucralfate as a topical therapy and/or prokinetic agents.

SUMMARY

- 1. The surgical therapy for gastric cancer has become more stage dependent
- 2. The need for a large resection for early gastric cancer is not indicated, and either EMR or laparoscopic resection can be utilized as the first step to surgical therapy in properly selected patients.
- 3. The location of the primary tumor remains the main determinant of the type of surgical resection required in patients with more advanced stage disease.
- 4. Neither total nor subtotal gastric resection offers any survival advantage to patients with gastric cancer, and the type of resection is determined on achieving an adequate surgical resection margin.
- 5. The complications associated with the surgical therapy of gastric cancer are common; however, most are transient and will resolve 6 to 8 wk after resection.

REFERENCES

- 1. World Health Organization. The world health report. Geneva: WHO, 1997.
- 2. Hiki Y. (1996) Endoscopic mucosal resection (EMR) for early gastric cancer. Nippon Geka Gakkai Zasshi 97:273–278.
- 3. Rembacken BJ, Gotoda T, Fujii T, et al. Endoscopic mucosal resection. Endoscopy 2001;33:709–718.
- 4. Hochwald SN, Brennan MF, Karpeh MS, et al. Is lymphadenectomy necessary for early gastric cancer? Ann Surg Oncol 1999;6:664–670.
- 5. Ohgami M, Otani Y, Kumai K, et al. Curative laparoscopic surgery for early gastric cancer: five years experience. World J Surg 1999;23:187–192.
- 6. Perri F, Iuliano R, Valente G, et al. Minute and small early gastric cancers in a Western population: a clinicopathologic study. Gastrointest Endosc 1995;41:475–480.
- 7. Maehara Y, Orita H, Okuyama T, et al. Predictors of lymph node metastasis in early gastric cancer. Br J Surg 1992;79:245–247.

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- 8. Moreaux J, Bougaran J. Early gastric cancer. A 25-year surgical experience. Ann Surg 1993;217:347-355.
- 9. Karpeh MS, Leon L, Klimstra D, et al. Lymph node staging in gastric cancer: is location more important than Number? An analysis of 1,038 patients. Ann Surg 2000;232:362–371.
- 10. American Joint Committee on Cancer. AJCC Cancer Staging Manual, 1997.
- 11. Smith JW, Shiu MH, Kelsey L, et al. Morbidity of radical lymphadenectomy in the curative resection of gastric carcinoma. Arch Surg 1991;126:1469–1473.
- Maruyama K, Sasako M, Kinoshita T, et al. Pancreas-preserving total gastrectomy for proximal gastric cancer. World J Surg 1995;19:532–536.
- Bonenkamp JJ, Hermans J, Sasako M, et al. Extended lymph-node dissection for gastric cancer. Dutch Gastric Cancer Group. N Engl J Med 1999;340:908–914.
- Cuschieri A, Weeden S, Fielding J, et al. (1999) Patient survival after D1 and D2 resections for gastric cancer: long- term results of the MRC randomized surgical trial. Surgical Co- operative Group. Br J Cancer 1999;79:1522–1530.
- 15. Siewert JR, Bottcher K, Stein HJ, et al. Relevant prognostic factors in gastric cancer: ten-year results of the German Gastric Cancer Study [see comments]. Ann Surg 19998;228:449–461.
- Maruyama K, Sasako M, Kinoshita T, et al. Reasonable lymph node dissection in radical gastrectomy for gastric cancer: introduction of computer information system and lymphography technique by India-ink]. Nippon Geka Gakkai Zasshi 1989;90:1318–1321.
- Nakane Y, Okumura S, Akehira K, et al. Jejunal pouch reconstruction after total gastrectomy for cancer. A randomized controlled trial. Ann Surg 1995;222:27–35.
- 18. Troidl H, Kusche J, Vestweber KH, et al. Pouch versus esophagojejunostomy after total gastrectomy: a randomized clinical trial. World J Surg 1987;11:699–712.
- Schmitz R, Moser KH, Treckmann J. Quality of life after prograde jejunum interposition with and without pouch. A prospective study of stomach cancer patients on the reservoir as a reconstruction principle after total gastrectomy]. 1994:Chirurg 65:326–332.
- 20. Fuchs KH, Thiede A, Engemann R, et al. Reconstruction of the food passage after total gastrectomy: randomized trial. World J Surg 1995;19:698–705.
- Schwarz A, Buchler M, Usinger K, et al. (Importance of the duodenal passage and pouch volume after total gastrectomy and reconstruction with the Ulm pouch: prospective randomized clinical study. World J Surg 1996;20:60–66.
- 22. Bozzetti F, Bonfanti G, Castellani R, et al. Comparing reconstruction with Roux-en-Y to a pouch following total gastrectomy. J Am Coll Surg 1996;183:243–248.

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Reconstruction After Distal Gastrectomy

Nitin Rangnekar, MD and Brent W. Miedema, MD

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INTRODUCTION

Distal gastrectomy has been a standard part of the abdominal surgeon's armamentarium since 1881 when Billroth performed the first distal gastrectomy for malignancy. The primary indications for distal gastrectomy in the past have been peptic ulcer disease and gastric tumors. Establishing the continuity of the gastrointestinal tract following distal gastrectomy has a rich and fascinating history. No other reconstruction of the gastrointestinal tract has produced such a variety of approaches (1).

Distal gastrectomy, in the treatment of ulcer disease, has waxed and waned over the past century. Gastroenterostomy was the earliest surgical approach to ulcer disease but had more than a 50% ulcer recurrence rate. Thus, subtotal gastrectomy became the treatment of choice. With the understanding of the vagus nerves in acid production, vagotomy with drainage became popular and the use of distal gastrectomy decreased markedly. With recognition of gastrin and the role of the antrum in ulcer etiology, a less radical distal gastrectomy (antrectomy) was combined with vagotomy, which could virtually eliminate ulcer disease. With increased understanding of postgastrectomy syndromes, vagotomy and antrectomy were gradually replaced by improving medical therapy or proximal gastric vagotomy. With the current understanding of ulcer disease as a *Helicobacter pylori* (*H. pylori*) infection that can be cured with antibiotics, gastrectomy has nearly disappeared in the treatment of ulcer disease.

Currently, primary distal gastrectomy is done almost exclusively for malignancy or the suspicion of malignancy. There is also a large population of patients that have had a prior gastrectomy, and require revisional gastric surgery. This chapter will review the currently used gastric reconstructions following distal gastrectomy or in revisional gastric surgery. The physiologic derangements and complications of these reconstructions will be detailed. Medical and surgical approaches to the side effects of gastric reconstructions will be outlined.

GASTRIC RECONSTRUCTION

Following distal gastric resection, the continuity of the small bowel with the stomach must be reestablished. The goal is to accomplish this in a way that will minimize the risk of postoperative complications. A thorough knowledge of the physiology of vagal innervation and gastric emptying are the main factors that will determine optimal gastric reconstruction. The condition of the patient, the extent of gastric resection, and surgeon preference may also play a role.

Reconstruction following resection of the stomach is accomplished in three general manners: 1) Billroth I (gastroduodenostomy); 2) Billroth II (loop gastrojejunostomy); 3) Roux-en-Y (end gastrojejunostomy). Modifications of the Roux-en-Y to create a reservoir include the Hunt-Lawrence pouch and the Tanner Roux-19 pouch. Each of the different reconstructions, and the extent of gastric resection, produces specific early and late postoperative complications.

The extent of distal gastric resection can vary (Fig. 1) depending on the pathology and goals of the procedure. An antrectomy removes 40–50% of the distal stomach and includes a greater portion of the lesser curvature where gastrin producing cells extend more proximally. A partial distal gastrectomy removes 50–80% of the stomach. A subtotal gastrectomy removes 80–99% of the stomach, but preserves the fundus. A neartotal gastrectomy removes 99% of the stomach including the fundus, leaving a rim of gastric tissue attached to the esophagus to be used for the anastomosis.



Fig. 1. Nomenclature for gastric resections and percentage of stomach removed. The antrum extends proximally on the lesser curvature and must be removed in all gastrectomies.



Fig. 2. Antrectomy with Billroth I reconstruction.

BILLROTH I RECONSTRUCTION

The Billroth I reconstruction is a gastroduodenostomy following distal gastrectomy (Fig. 2). This procedure involves Kocherization (after Theodor Kocher), which releases the duodenum laterally from the peritoneal reflection so as to reduce tension on the anastomosis. The distal stomach is transected proximally, usually by a stapling device. The duodenum is transected just distal to the pylorus, and the two ends are anastomosed, either by handsewn technique or using a mechanical stapling device. There are a number of modifications of the Billroth I operation (Fig. 3), depending upon the exact construction of the anastomosis.

INDICATIONS

Billroth I is the preferred reconstruction following distal gastrectomy. The purported advantage of a Billroth I is that it is more physiologic. By having the gastric content pass



Fig. 3. Variations of the Billroth I reconstructions. (A) Billroth I. (B) Horsley. (C) von Haberer-Finney. (D) von Haberer. (E) Shoemaker.

through the duodenum, the normal pancreaticobiliary stimulation and negative feedback signals to the stomach are maintained. The immediate mixing of gastric content and duodenal secretions enhance digestion and absorption. There is less contact of the bile with the stomach, decreasing the incidence of alkaline reflux gastritis. There are no loops to become kinked and the incidence of gastric stasis appears to be less.

CONTRAINDICATIONS

The most important contraindication to performing a Billroth I anastomosis is duodenal bulb scarring, from previous ulcers. An anastomosis into a fibrotic duodenum has a high risk of leak or stenosis. In addition, scarring makes mobilization of the duodenum more difficult and can result in tension on the gastroduodenal anastomosis. Billroth I is contraindicated if it will result in tension at the anastomosis. As any anastomosis under tension has a high risk of leak.

COMPLICATIONS

The most important complication of the Billroth I procedure is anastomotic leakage. This creates intra-abdominal spillage with peritonitis, often results in enterocutoneous fistula, and has a high risk of mortality. Management can include percutaneous or open drainage, often requiring prolonged bowel rest and total parenteral therapy. Conversion to a Billroth II may be required. Intra-abdominal abscesses or wound infections occur after gastric surgery, especially in patients with gastric stasis where increased enteral fluid or bezoars are present. Acute dilatation of the stomach can occur spontaneously or because of stomal obstruction. This usually responds to gastric tube decompression. Stenosis at the anastomosis can also occur, but can often be treated with endoscopic dilatation.



Fig. 4. Subtotal gastrectomy with Billroth II reconstruction. The anastomosis to the jejunum is made either with the afferent loop at the greater curvature, (isoperistaltic) or at the lesser curvature, (antiperistaltic) depending on the patient's build.

BILLROTH II RECONSTRUCTION

A Billroth II (Fig. 4) is a loop gastrojejunostomy and was first performed by Billroth for a large pyloric cancer. After distal gastrectomy, the proximal stomach is anastomosed to a jejunal loop, performing a gastrojejunostomy. It is important to remove the entire antrum, because any retained antrum secretes gastrin and is a potent cause of ulcer recurrence. The jejunal loop can be brought up either anterior to the transverse colon (antecolic), or through the transverse mesocolon (retrocolic) for the gastrojejunostomy. The afferent limb is usually sutured to the greater curvature and should lie comfortably without tension or kinking. In performing gastrectomy for benign disease, there is no clear evidence that either an antecolic or retrocolic gastrojejunostomy provides better emptying. For malignant disease, many surgeons feel that the retrocolic position may predispose to obstruction from enlargement of lymph nodes or serosal implants in the mesocolon. The afferent limb should be as short as possible without putting tension on the anastamosis. This will help avoid the afferent loop syndrome. There are many modifications of the Billroth II gastrojejunostomy (Fig. 5).



Fig. 5. Billroth II with some of its modifications and the year it was described.

INDICATIONS

The Billroth II reconstruction is preferred if the duodenum has scarring or tension would result from a Billroth I reconstruction. The advantage of the Billroth II is its simplicity. Only one anastomosis is required (plus the duodenal stump closure) and a widely patent anastomosis is technically easy to perform. The primary disadvantage of a Billroth II is that the gastric remnant is exposed to pancreaticobiliary secretions. This can result in alkaline reflux gastritis. Also, because the feedback mechanisms of the duodenum are bypassed, dumping is generally more of a problem than after a Billroth II reconstruction. However, most patients tolerate a Billroth II well and it is the construction of choice if a Billroth I is not safe.

CONTRAINDICATIONS

A Billroth II should not be done if a subtotal (greater than 80%) gastrectomy has been accomplished. A high incidence of alkaline reflux esophagitis will result and is very difficult to treat medically.

COMPLICATIONS AND MANAGEMENT

Anastomotic leaks are unusual after Billroth II. If they occur, it is usually owing to an inadequate gastric blood supply and requires revision by further distal gastrectomy and another Billroth II or Roux-en-Y reconstruction. Recurrent ulcers are common and can lead to stomal stenosis. Dilatation or revision will then be necessary. Billroth II also has

a greater incidence of dumping and alkaline reflux gastritis than Billroth I and these complications are discussed later.

The most feared complication after Billroth II is duodenum stump blow out. This can be prevented by placing a decompression tube in the duodenum if a difficult duodenum stump closure is encountered. Postoperative duodenal stump blow out is treated with percutaneous drainage and prolonged parenteral nutrition therapy. Closure will usually occur in 3 to 4 wk. If diffused peritonitis develops, open laparotomy to control the duodenal leak maybe needed. With a difficult duodenal stump dissection, risk of bile duct or pancreas injury increases. Thus, pancreatitis or jaundice is occasionally seen. Acute volvulus of the jejunal limbs involved in the Billroth II anastomosis has also been reported.

ROUX-EN-Y RECONSTRUCTION

The Roux-en-Y anastomosis is named after Caesar Roux, a Swiss surgeon. He popularized this operation in the 1880s to prevent bilious vomiting following gastrojejunostomy. The Roux procedure completely diverts pancreaticobiliary secretions away from the stomach (Fig. 6).

The Roux limb is created by transecting the jejunum 15–20 cm from the ligament of Treitz (at the duodenojejunal junction). The distal end of the transected jejunum is then anastomosed to the remnant of the stomach. The proximal end of the jejunum, which is a continuation of the duodenum, is then anastomosed to the jejunum approx 40 cm from the gastrojejunostomy. The jejunum between the gastrojejunostomy and the jejunoje-junostomy is the Roux limb. With trial and error, the optimal length of the Roux limb has been found to be approx 40 cm. A shorter Roux limb does not reliably prevent reflux of pancreaticobiliary secretions to the stomach. A longer Roux limb is associated with gastric stasis and malabsorption. The Roux limb can be brought either antecolic (anterior to the transverse colon) or retrocolic (through the mesentery of the transverse colon) to create the gastrojejunostomy. Each has its advantages, the antecolic is preferred in malignancies to prevent tumor obstruction of the Roux limb. The retrocolic anastomosis is preferred in benign conditions as it is a more direct route to the stomach reducing anastamotic tension, and it may give better gastric drainage.

INDICATIONS

The most common indication for the Roux-en-Y reconstruction is a subtotal, near total, or total gastrectomy. The Roux-en-Y is also used in revisional gastric surgery to correct alkaline reflux gastritis or dumping. The Roux limb diverts pancreaticobiliary secretions away from the gastric remnant, thus eliminating the risk of alkaline reflux gastritis. It prevents dumping because of orally propagating contractions that originate in the Roux limb and slow gastric emptying.

CONTRAINDICATIONS

A Roux-en-Y should not be done in the setting of delayed or marginal gastric emptying. The Roux will further delay gastric emptying and revisional surgery will be required (2). If a Roux is indicated in the setting of poor gastric emptying, it should be accompanied with a near total gastrectomy.



Fig. 6. Roux-en-Y gastrojejunostomy (note truncal vagotomy and antrectomy). The Roux limb should be 40 cm in length. The afferent limb from the ligament of Treitz to the enteroenterostomy is 15–20 cm.

COMPLICATIONS

The Roux procedure can cause poor emptying of the stomach, but can also cause delayed emptying of the Roux limb. Poor emptying of the Roux limb can result in bacterial overgrowth syndromes. This can be treated with antibiotics, but a high index of suspicion is needed to make the diagnosis. A minor disadvantage of Roux gastriojejunostomy is that an extra anastomosis is needed; thus, the risk and length of the operative procedure is increased.

The Roux limb is frequently used in revisional gastric surgery. Because of scarring, revisional gastric surgery has a greater risk of injury to the blood supply of the stomach than the initial gastric surgery. Therefore, gastric necrosis is seen more in revisional gastric surgery. Gastric necrosis requires emergent reoperation, excision of the necrosis, and reanastomosis of the Roux limb to the viable stomach or esophagus.

GASTRIC RESEVOIR RECONSTRUCTION

A near total gastrectomy mandates a Roux-en-Y type reconstruction. Otherwise, the extremely bothersome syndrome of alkaline reflux esophagitis results. The question then becomes whether a standard Roux-Y or some sort of Roux-Y-reservoir is best for



Hunt-Lawrence Fouch

Fig. 7. Gastric reservoir reconstruction for small stomach syndrome. The Hunt-Lawrence pouch and Tanner Roux-19 pouch are the most common gastric reservoir operations. Arrows show the general direction of peristaltic waves.

the patient. The two most common reservoir procedures are both based on the Roux-Y. These are Hunt Lawrence Pouch and the Tanner 19 Roux-en-Y (Fig. 7).

A straight Roux-en-Y gastrojejunostomy is the most technically simple procedure following near-total gastrectomy, and thus likely to have less immediate postoperative complications and cost. Patients with a gastric reservoir after primary gastric resection appear to have fewer alimentary symptoms over the long term following near-total or total gastrectomy for cancer. The data is less certain for patients that require a neartotal gastrectomy for postgastrectomy syndromes, and further studies are needed to make a recommendation.

MORBIDITY FOLLOWING DISTAL GASTRECTOMY

Postgastrectomy syndromes are late complications following gastric surgery caused by physiologic derangements as a result of the surgery. The primary alterations that result in postgastrectomy syndromes are truncal vagotomy and ablation or bypass of the pylorus. Vagotomy decreases the compliance and contractility of the stomach (3). Gastric contractions, which start as a peristaltic wave in the proximal stomach, are needed to transport proximal gastric content toward the distal stomach and duodenum. Antrectomy decreases the mechanical digestive function. The antrum grinds the food into small particles. Bypass of the pylorus results in uncontrolled gastric emptying. An intact pylorus has a separating action that prevents the passage of large particles or volumes into the intestine (4) while controlling reflux of duodenal content into the stomach. The small bowel also has a role in gastric emptying by offering resistance to inflow from the stomach, and by activating neural and hormonal mechanisms that provide feedback to the stomach to slow its emptying. Bypass of the duodenum eliminates many of these reflexes. Alterations in acid and enzyme secretion, hormonal regulation, and intrinsic factor production are also present after resection.

It is important to know the exact anatomic reconstruction performed, along with physiologic disturbances, which can exacerbate symptoms. Diagnostic modalities that help define this are upper endoscopy and gastrointestinal barium studies. For most postgastrectomy syndromes, a trial of conservative management for at least a year is usually justified. If symptoms cannot be controlled medically, revisional surgery is the best option.

The major early complications of gastric surgery include hemorrhage, and leakage from the anastomotic suture lines. Mechanical complications include stomal obstruction, volvulus of one of the jejunal loops behind the anastomotic loop, afferent loop syndrome and malplacement of the anastomosis. Serious hemorrhage is uncommon and is usually iatrogenic secondary to unligated blood vessels. These occasionally require reoperation. Stomal obstruction, usually secondary to stomal edema, generally responds to conservative management. Electrolyte correction and parenteral nutrition are helpful adjuncts. Mortality after elective gastric resection without vagotomy for ulcer disease is 1% (5). The mortality increases to 2% when gastrectomy is accompanied by vagotomy.

POSTGASTRECTOMY SYNDROMES

The most important common postgastrectomy syndromes are dumping, alkaline reflux gastritis, and gastric stasis. Less common postgastrectomy syndromes include small stomach syndrome, postvagotomy diarrhea, afferent loop syndrome, efferent loop syndrome, and recurrent ulcer. Most patients also develop iron deficiency anemia likely caused by exclusion of the duodenum from the enteral stream. The duodenum is the primary site of iron absorption. Poor mixing of the bile and food can result in malabsorption. B12 and folate deficiencies are common nutritional complications. Owing to the hypochlorhydria following vagotomy, postgastrectomy patients may be at greater risk for developing cancer in the gastric remnant. The incidence of postgastrectomy syndromes has decreased overall as a result of the marked decrease in the number of gastric surgeries performed, especially for peptic ulcer disease. This is mainly because of the recognition of *H. pylori* and its causal relationship with peptic ulcer disease. Postgastrectomy syndromes have been reported in 5% to 50% of patients, in most studies the incidence is near 25% (6). For unknown reasons, females have a higher incidence of postgastrectomy syndromes.



Fig. 8. Dumping syndrome. Rapid gastric emptying after truncal vagotomy, partial gastrectomy, and gastrojejunostomy.

DUMPING SYNDROME

Dumping is one of the most common postgastrectomy syndromes and may be divided into an early form and a late form, based on the time interval between food ingestion and onset of symptoms. Both are caused by ingestion of hyperosmolar carbohydrate-rich food. Early dumping begins within 10–30 min of a meal and is characterized by both gastrointestinal manifestations and vasomotor symptoms. The gastrointestinal manifestations include fullness, crampy abdominal pain, nausea, vomiting, and explosive diarrhea. Vasomotor manifestations include diaphoresis, weakness, dizziness, flushing, and palpitations. Patients with dumping tend to decrease their food intake because of the intense discomfort and can become malnourished.

The primary mechanisms leading to the dumping syndrome are loss of reservoir function of the stomach and the rapid emptying of hyperosmolar carbohydrates into the small intestine (Fig. 8). Decreased gastric reservoir is caused by a loss of proximal gastric receptive relaxation and accommodation with vagotomy and loss of gastric capacity with gastric resection. Ablation or bypass of the pylorus results in loss of the control of gastric emptying. In Billroth II reconstruction, loss of duodenal feedback with inhibition of gastric emptying also contributes to rapid gastric emptying.

Sudden appearance of large amounts of hyperosmolar carbohydrates in the small intestine leads to fluid shifts from the intravascular space into the bowel lumen (7). In addition, several enteric hormones, including serotonin, gastric inhibitory polypeptide (GIP), vasoactive inhibitory peptide (VIP), and neurotensin, are released during dumping and are probably responsible for some of the vasomotor manifestations of early dumping (8-10). Rapid emptying of the stomach correlates closely with dumping syn-

drome (11). The diagnosis of dumping is primarily made on clinical grounds, however gastric emptying studies may aid in confirmation of the diagnosis.

The initial treatment of dumping is dietary. Decreased meal size, increasing frequency of meals, avoiding concentrated carbohydrates, and taking liquids between meals, are some of the mainstays of dietary therapy. The somatostatin analog, octreotide, also has been effective in treating dumping, likely because of its inhibitory effect on the release of enteric hormones (12). Only1% of these patients will ultimately fail medical management and require surgery.

A variety of surgical procedures have been used to treat dumping. The Roux-en-Y procedure significantly delays gastric emptying and is very effective in reducing dumping symptoms. Other surgical procedures for dumping include conversion of Billroth II to Billroth I, isoperistaltic and antiperistaltic jejunal interposition, and pyloroplasty reversal. These procedures have not been as consistently successful as the Roux-en-Y. The disadvantage to using the Roux-en-Y procedure for dumping is that many of these patients develop post-operative gastroparesis that eventually requires a near total gastric resection.

Late dumping occurs 2–4 h following a meal, and is a form of reactive hypoglycemia from excessive insulin release (13). Unlike early dumping symptoms, late dumping is relieved by ingestion of liquids that contain sugar. Dietary therapy is usually successful and revisional surgery almost never necessary.

ALKALINE REFLUX GASTRITIS SYNDROME

Prolonged contact of pancreaticobiliary secretions with the gastric mucosa can produce damage. The quantity of bile entering the stomach does not correlate with symptoms, but slower gastric emptying does. Thus, the syndrome is likely from both bile reflux to the stomach and delayed clearing of the bile. The syndrome is characterized by bilious vomiting and epigastric abdominal pain. Marginal ulceration, anastomotic stricture, afferent-loop syndrome, and chronic gastroparesis are included in the differential diagnosis. The diagnosis of alkaline gastritis to a certain extent remains one of exclusion. Endoscopic appearance of the stomach is characteristic and shows a beefy red appearance of the gastric mucosa. Barium studies can help to rule out other causes of the pain and vomiting. Quantitative radionuclide biliary scanning usually shows enterogastric biliary reflux.

No specific medical therapy is available for alkaline reflux gastritis. Prokinetic agents, proton pump inhibitors, H_2 antagonists, and cholestyramine all have inconsistent results. Roux-en-Y biliary diversion is the treatment of choice. Vagotomy, if not performed earlier, is done to prevent marginal ulceration. Some surgeons recommend adding subtotal gastrectomy to speed gastric emptying. Jejunal interposition between the gastric remnant and the duodenum has also been successfully used to treat this condition.

GASTRIC STASIS SYNDROME

Gastric stasis (also called gastric atony or gastroparesis) results from two postgastrectomy derangements. Vagotomy decreases the frequency and amplitude of gastric contractions. Gastric resection disturbs the motility of the stomach, also impairing gastric emptying. These patients have postprandial epigastric pain, nausea, and vomiting of partially digested food eaten hours or even days before. They may be malnourished and



Fig. 9. Gastric stasis after truncal vagotomy, partial gastrectomy and Roux gastrojejunostomy.

often restrict themselves to a liquid diet, which is tolerated better than solids. Bezoars are common and are seen in up to 12% of postgastrectomy patients (14). Gastric atony (Fig. 9) is a major complication of gastric surgery. This may be further exacerbated by diabetes, hypothyroidism, and neurologic disorders. It is important to determine whether the impedence to gastric outflow is mechanical or functional. Endoscopy, contrast radiography, and scintigraphy usually allow differentiation.

Medical therapy with prokinetic agents such as metoclopramide and erythromycin has limited success. Severely symptomatic patients generally require revisional surgery. Because vagal innervation cannot be restored, operative therapy is aimed at decreasing the reservoir capacity of the stomach. A near-total gastric resection removes the atonic stomach, and a Roux-en-Y reconstruction is needed to prevent bile reflux esophagitis.

A subtype of the gastric stasis syndrome is the Roux Stasis Syndrome. Patients with the Roux-en-Y gastroenterostomy are at especially high risk for delayed gastric emptying. This is caused by disordered motility in the Roux limb, which increases the resistance to gastric emptying. Truncal vagotomy may also diminish the strength of Roux contractions. Roux stasis syndrome is especially resistant to medical treatment and often requires near total gastrectomy with Roux-en-Y gastrojejunostomy. Even after neartotal gastrectomy, persistence of significant Roux stasis syndrome is present in up to one-third of patients. The only surgical option at this point is a gastric reservoir procedure, which has unpredictable results in this patient group.

SMALL STOMACH SYNDROME

This syndrome is a result of loss of reservoir function when 80% or more of the stomach is removed. It differs from gastric stasis syndrome in that gastric emptying is normal. Symptoms include early satiety, epigastric pain after eating, and vomiting. Some patients develop severe weight loss, malnutrition, and anemia secondary to folate, vitamin B_{12} or iron deficiency.

Dietary treatment is often successful and consists of increasing the frequency and decreasing the size of the meals, adding supplemental vitamins, iron, and pancreatic enzymes. Surgical treatment aims at increasing gastric capacity by creating a gastric reservoir for the patients with a Hunt-Lawrence pouch (15) or the Tanner Roux-19 pouch (16). Both of these reconstructions carry the risk of stasis and ulceration, and are performed only in patients with severe symptoms that have failed conservative management.

POSTVAGOTOMY DIARRHEA SYNDROME

Vagal innervation is an important factor in the control of pancreaticobiliary secretion and intestinal absorption. Truncal vagotomy may result in excessive small bowel secretions or bile acids with resulting diarrhea. Diarrhea occurs in up to 25% of patients following a complete (truncal) vagotomy with gastric drainage or resection (17). Less than 2% of patients have incapacitating symptoms. The syndrome is characterized by frequent watery stools, usually unrelated to meals, and occurring at night. Medical therapy includes dietary alterations with low fluid content, frequent feedings, increasing dietary fiber, and adding substances such as pectin to slow intestinal transit. Medications include cholestyramine, which may help bind bile salts, and somatostatin, which is effective in some patients. Surgical therapy is rarely recommended, but when needed, consists of interposition of a 10 cm antiperistaltic jejunal limb 100 cm distal to the ligament of Treitz (18).

AFFERENT LOOP SYNDROME

The afferent-loop syndrome results from obstruction of the afferent limb of a loop (Billroth II) gastrojejunostomy. This is generally caused by an excessively long afferent limb. The recognition that short afferent limbs are preferable has greatly decreased the incidence of this complication. The symptoms of this syndrome are fairly classic and include intermittent epigastric or right upper-quadrant pain, relieved by projectile bilious vomiting. If the vomitus contains both bile and food, and pain is unrelieved after vomiting, one should consider alkaline reflux gastritis as more likely than afferent loop syndrome. Chronic afferent loop syndrome results from anastomotic stricture, adhesion formation, stomal ulceration, carcinoma, and jejunogastric intussusception. The syndrome does not exist following either a Billroth I resection or vagotomy and pyloroplasty. It occurs in both an acute and a chronic form. The acute form appears within the first 1–2 wk after the operation. The acute postoperative form may be life threatening

and may predispose to duodenal stump dehiscence and afferent limb necrosis. Rapid surgical intervention is indicated in the acute form.

The chronic form of afferent loop syndrome is associated with a dilated afferent limb and can occur 30 or more yr after a Billroth II procedure (19). The dilated limb can be identified by barium or radionuclide studies. The chronic form may be associated with bacterial overgrowth in the afferent limb. The main diagnostic difficulty is differentiating afferent loop syndrome from alkaline reflux gastritis. This is because bilious vomiting can occur with either syndrome. In afferent loop syndrome, sudden release of bile into the gastric remnant after food has already emptied from the stomach results in bilious vomiting and relief of pain. Endoscopy helps in separating afferent loop syndrome from alkaline reflux gastritis. Once a diagnosis is made, surgical correction is indicated. The Billroth II anastomosis may be revised to a Billroth I or Billroth II with a shorter afferent limb. Roux-en-Y gastrojejunostomy has also produced good results.

EFFERENT LOOP SYNDROME

The efferent loop syndrome is a purely mechanical problem characterized by gastric outlet obstruction at or near the gastrojejunostomy. The etiology may be kinking or adhesions of the efferent limb, or internal herniation behind the gastroenterostomy. Symptoms include diffuse abdominal pain, nausea, and bilious vomiting. The syndrome may occur months to years after the operation. The diagnosis may be confirmed by contrast studies, which demonstrates obstruction of the efferent limb. Surgical therapy is usually required. The most common finding is an adhesive band obstructing the efferent limb near the gastric anastomosis (20). Correctional procedures may include revision of the anastomosis or conversion to a Billroth I or Roux-en-Y gastrojejunostomy.

COST

Cost for distal gastrectomy and reconstruction are highly variable. In our institution, the surgeon's fee for gastrectomy with Roux-en Y reconstruction is approx \$3500, whereas the Medicare reimbursements for these procedures range between \$900 and \$1200.

SUMMARY

- 1. Primary distal gastrectomy continues to be indicated, primarily, in patients with a gastric tumor or a suspicion of tumor. However, there is a huge population of patients that have had gastric surgery for peptic ulcer disease and present with post gastrectomy syndromes.
- 2. The primary reconstructions following distal gastrectomy are gastroduodenostomy (Billroth I), loop gastrojejunostomy (Billroth II), and end gastrojejunostomy (Rouxen-Y). After near total gastrectomy a reservoir can be created using a Roux gastrojejunostomy and may provide long-term benefits especially for patients requiring gastrectomy for tumors.
- 3. Unfortunately, all reconstructions following partial gastrectomy are associated with a high risk of long-term complications. The most common postgastrectomy syndromes are dumping, alkaline reflux gastritis, and gastric stasis.
- 4. The Billroth I reconstruction has the lowest incidence of postgastrectomy syndromes and is the reconstruction of choice if it can be done safely.
- 5. A Billroth II should be done if a Billroth I cannot be performed safely. The Roux-en-Y reconstruction is usually not done primarily after distal gastrectomy, but should be

reserved for certain types of revisional gastric surgery and if subtotal gastrectomy is necessary.

6. Most patients with postgastrectomy syndromes can be can be stabilized on dietary or medical therapy. When postgastrectomy symptoms are severe, revisional gastric surgery is generally beneficial.

REFERENCES

- Herrington JL. Historical aspects of gastric surgery. In: Scott HW, Sawyers JL, eds., Surgery of the Stomach. Duodenum and Small Intestine. 2nd ed. Blackwell Scientific, Boston, MA, 1992; pp. 1–28.
- 2. Hocking MP, Vogel SB, et al. Delayed gastric emptying of liquids and solids following Roux Y biliary diversion. Ann Surg 1981;194:494–501.
- 3. Hom S, Sarr MG, Kelly KA, et al. Postoperative gastric atony after vagotomy for obstructing peptic ulcer. Am J Surg 1989;157:282.
- 4. Meyer JH, Thompson JB, Cohen MB, et al. Sieving of solid food by the canine stomach and sieving after gastric surgery. Gastroenterology 1979;76:804–813.
- 5. Welch CE, Rodkey GV. Partial gastrectomy for duodenal ulcer. Am J Surg 1963;105:338-346.
- 6. Thompson JC, Weiner T. Evaluation of surgical treatment of duodenal ulcer: Short and long-term effects. Clin Gastroenterol 1984;13:569–600.
- 7. Machella TE. The mechanism of the post-gastrectomy "dumping" syndrome. Ann Surg 1949;130:145–159.
- 8. Drapanas T, McDonald JC, Stewart JD. Serotonin release following instillation of hypertonic glucose into the proximal intestine. Ann Surg 1962;156:528–536.
- 9. Sagor GR, Bryant MG, Ghatei MA, et al. Release of vasoactive intestinal peptide in the dumping syndrome. Br Med J 1981;282:507–510.
- 10. Sirinek KR, O'Dorisio TM, Howe B, et al. Neurotensin, vasoactive intestinal peptide, and Roux-en-Y gastrojejunostomy: Their role in the dumping syndrome. Arch Surg 1985;120:605–609.
- 11. Donovan IA, Gunn IF, Brown A, et al. A comparison of gastric emptying before and after vagotomy with antrectomy and vagotomy with pyloroplasty. Surgery 1974;76:729–732.
- 12. Primrose JN, Johnston D. Somatostatin analogue SMS 201–995 (octreotide) as a possible solution to the dumping syndrome after gastrectomy or vagotomy. Br J Surg 1989;76:140–144.
- 13. Shultz KT, Neelon FA, Nilsen LB, et al. Mechanism of post gastrectomy hypoglycemia. Arch Int Med 1971;128:240.
- 14. Goldstein HM, Cohen LE, Hagen RO, et al. Gastric bezoars: A frequent complication in the postoperative ulcer patient. Radiology 1973;107:341–344.
- 15. Hunt CJ, Cope JS. Modified technique for total gastrectomy with formation of a food pouch from the jejunum. Am Surg 1952;18:85–90.
- Tanner NC. Personal observations and experiences in the diagnosis and management of ulcer disease and disabilities that follow peptic ulcer operations. Surg Clin North Am 1976;56:1349–1363.
- 17. Sawyers JL. Management of postgastrectomy syndromes. Am J Surg 1990;159:8.
- Craft II, Venables CW. Antiperistaltic segment of jejunum for persistent diarrhea following vagotomy Ann Surg 1968;167:282–286.
- 19. Matsusue S, Kashihara S, Takeda H, et al. Three cases of afferent loop obstruction: The role of ultrasonography in the diagnosis. Jpn J Surg 1988;18:709–713.
- Herrington JL, Swayers JL. Complications following gastric operations. In Schwartz SI, Ellis H, eds., Maingot's Abdominal operations, 9th ed. Appleton and Lange, Norwalk, CT, 1989, pp. 701–730.

11 Surgery for Obesity

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INTRODUCTION

Obesity affects almost 50% of the population in the United States. Obesity is more accurately expressed using the body mass index (BMI), which is calculated as the body weight in kilograms divided by the height in meters squared. An individual with BMI over 40, more than twice his or her ideal body weight, is considered morbidly obese. Recent estimates indicate that 4% of the general population, or more than 10 million Americans, are morbidly obese. The National Institutes of Health Consensus established that obesity is correlated to many health problems (1). Hypertension, cardiomyopathy, noninsulin-dependent diabetes, sleep apnea, infertility, ovarian tumors, prostate tumors, depression, and other psychological alterations are among the most commonly associated conditions. Life expectancy is significantly shorter for the morbidly obese than for those with normal weight.

Medical treatment for obesity includes diet, exercise, behavioral modification, and pharmacotherapy. The medical treatment may initially control, but not ultimately cure obesity. It has been shown that more than 99% of the morbidly obese fail to maintain a significant weight loss for a period longer than 2 yr. This failure was the catalyst for the creation of surgical procedures to treat this problem. Many different techniques have been developed in the almost four decades since the first bariatric procedures were

From: *Clinical Gastroenterology: An Internist's Illustrated Guide to Gastrointestinal Surgery* Edited by: George Y. Wu, Khalid Aziz, and Giles F. Whalen © Humana Press Inc., Totowa, NJ described. The shortcomings, severe complications, and high mortality of the early procedures initially gave bariatric surgery a bad reputation. In recent years, surgical treatment of obesity has become increasingly popular as a result of significant media coverage, great results, and well-known personalities having successfully undergone this type of surgery. From 1999 to 2000, the number of bariatric procedures performed in the United States doubled from 20,000 to 40,000.

The American College of Surgeons and the American Society for Bariatric Surgery have developed guidelines for those surgeons interested in performing this type of surgery. Hospitals must be staffed with personnel educated and committed to fulfilling the special needs of these patients. Specialized equipment in the operating rooms and surgical wards is often required. It has been demonstrated that using a multidisciplinary approach to evaluate candidates for surgery enhances the overall success of the procedure. This multidisciplinary group should consist of the following: bariatric surgeon, psychiatrist or psychologist, pulmonologist with expertise in sleep apnea, cardiologist, nutritionist, a program coordinator and, in selective cases, a gastroenterologist and endocrinologist.

INDICATIONS

We have adopted several guidelines for evaluating patients for gastric bypass. The patient must initially attend a concise but thorough presentation about the process of patient evaluation, treatment, and follow-up after the surgery. We make this mandatory because we believe the outcome of the surgery is based on the patient's understanding of the procedure, its role in the process of weight loss, as well as its limitations. The patient must meet the following criteria before being considered for surgery: minimum age 18 yr, BMI of at least 35 in the face of medical co-morbidities or BMI greater than 40 if the patient is free of medical co-morbidities. The patient is asked to complete a written exam covering the major points of the surgery, the implication of life-long commitment to dietary regimen, potential benefits, and complications. This is done to confirm that the patient has realistic expectations of the intended outcome. Last, the patient must demonstrate the existence of strong psychological support by relatives or friends. Having met these criteria, the patient is considered for medical clearance by the multidisciplinary team. This surgery would be contraindicated in any patient who does not fulfill these criteria.

The Roux-en-Y gastric bypass (RYGB) and vertical banded gastroplasty (VBG) are the most broadly practiced bariatric surgeries in the world. These procedures have evolved through the years as a result of surgeon experience, improvement of stapling devices and techniques, and patient outcome. The goal of bariatric surgery is to ensure adequate weight loss with minimal complications while relieving medical co-morbidities. This results in an overall improvement of the patient's quality of life. Over the last 10 yr, RYGB has become the technique of choice because of greater resultant weight loss and better long-term results. Because VBG maintains normal passage of food through the duodenum it avoids the gastric complications that may be difficult to manage after exclusion operations. Moreover, studies have demonstrated that calcium, phosphate, and iron absorption are preserved by this technique (2). VBG is not indicated in the subgroup of patients categorized as superobese (BMI>60) because the expected weight loss is much less compared to that with RYGB.

CONTRAINDICATIONS

These procedures are contraindicated in patients below the age of 18, a BMI less than 35 with medical co-morbidities, or less than 40 without medical co-morbidities. They should not be performed on patients who lack a life-long commitment to dietary regimen, who do not have realistic expectations of the intended outcome, or who lack strong psychological support by relatives or friends.

PROCEDURE

The key to VBG is the development of a pouch and an outlet of appropriate size (Fig. 1). The esophagus is bluntly dissected at the level of the esophagogastric junction with a 36-38 French bougie in place and encircled with a Penrose drain. This maneuver will provide the required control for the mobilization and creation of the pouch. An Endto-End Anastomosis (EEA) stapler is used to create centrally located defects in the anterior and posterior walls of the stomach. A TA-90B stapler is passed through the defect and directed toward the angle of His such that the resultant staple line is approx 4-6 cm in length. The stapler is then fired through the full thickness of the stomach creating a 20-mL pouch. At least four parallel rows of staples are placed to prevent future breakdown of the pouch. A nonabsorbable mesh band measuring 7×1.5 cm is placed circumferentially at the base of the pouch to control the outlet into the stomach. A piece of omentum is sutured over the mesh to prevent adhesions to the liver and kinking of the outlet. Surgical staples are also used to create the stomach reservoir in a Roux-en-Y gastric bypass (Fig. 2). One of the essential differences between VGB and RYGB is the complete transection of the stomach in the RYGB and the creation of a 30-cm³ capacity gastric pouch. The small bowel is transected distal to the ligament of Treitz and a 75-cm limb used to create a hand-sewn or stapled (21 mm EEA) retrocolic, retrogastric pouchjejunostomy. The jejuno-jejunostomy is likewise either hand-sewn or stapled. After testing the pouch anastomosis with air via intraoperative endoscopy, the limb of small bowel is secured to the mesentery to avoid internal hernias. A Jackson- Pratt drain is placed near the anastomosis of the pouch. The radiological representation of both procedures is distinctively different (Fig. 3).

COMPLICATIONS

The most feared complication of VBG is dehiscence of the staple line. The risk of breakdown is about 20% per patient per year. In the event of breakdown of the staple line, the stomach may return to its original size resulting in no further weight loss.

Another common complication of the VBG is narrowing of the pouch outlet, which occurs in approx 3% of patients. Patients present with nausea, vomiting, and dysphagia. Initially, treatment consists of conservative measures such as instructing the patient to eat smaller meals, chew food very well, and supplement the diet with high protein drinks. Because neither procedure is readily reversible, all attempts are made to manage strictures nonoperatively. Unfortunately, endoscopic dilation is only successful in 10% of cases for the VBG. When nonoperative measures fail to relieve symptoms the surgeon may attempt restapling with gastric division or rebanding at different levels. Surprisingly, band erosion into the hollow viscus is very rare.

Leaks are a very concerning and potentially fatal complication. Postoperative tachycardia, low urinary output, fever, and respiratory distress should alert the surgeon to



Fig. 1. A diagram of vertical banded gastroplasty (VBG). Surgical staples and a polypropylene mesh collar are used to construct a small gastric pouch of limited capacity.



Fig. 2. A diagram of Roux-en-Y gastric bypass (RYGB). The esophagus is transected and anastomosed with a stapler to create gastric pouch.

promptly search for any evidence of a leak. Expeditious reexploration with repair of leak and adequate drainage avoid further deterioration and eventual demise secondary to sepsis.

Other complications include dehydration, vitamin deficiency (B₁₂), pouch stomal edema, pouch dilatation, wound infection (15%–30%), abdominal wall hernias (21%), intestinal obstruction (6%), cholecystitis (12%), splenic tear (2.5%), pulmonary embolism (3%), marginal ulceration (9%), and intra-abdominal abscesses (2.3%) (3).



Vertical banded gastroplasty

Roux-en-Y gastric bypass

Fig. 3. Radiographic appearance of the procedures: (A) VBG. The body of the stomach fills with barium due to reflux of constrast after traversing the surgical pouch. The sensation of postprandial fullness is produced by the filling of the pouch. (B) Early phase barium study of a RYGB.

There are not many nutritional problems directly attributed to these procedures. Protein and caloric malnutrition are extremely rare despite the small amount of protein and calories that these patients consume during the early period after the surgery. Intolerance to certain food types is characteristic with red meat being one of the most difficult foods to tolerate. Dumping syndrome is specific to gastric bypass and occurs in 30%–40% of patients. This makes RYGB a better procedure for those patients who have trouble controlling their carbohydrate intake.

Vitamin deficiencies do occur after either VBG or RYGB making multivitamin supplementation mandatory in these patients. Vitamin B_{12} deficiency is a problem specific to those patients with RYGB. The etiology of this deficiency is not clear but has been attributed to a decrease in acidity of the pouch, decreased production of intrinsic factor, and decreased absorption of B_{12} -intrinsic factor compound. Vitamin B_{12} deficiency occurs in some VBG patients, but is thought to result from poor digestion or inadequate consumption of red meat. Patients undergoing either procedure should receive supplemental vitamin $B_{12}(4)$. Iron deficiency may occur and is more commonly associated with RYGB particularly in menstruating females. The problem is a consequence of minimal intake and poor absorption. Serum vitamin and iron levels should be monitored yearly to rule out deficiencies.

POSTOPERATIVE CARE

In our experience, patients can usually be extubated in the operating room. Antibiotics are discontinued on the first postoperative day and low molecular weight heparin is used for the duration of the hospitalization. On the second postoperative day, patients are allowed to drink small amounts of water not to exceed more than 30 cm³ every 15 min. If the patient tolerates this well, the diet is advanced to clear liquids the next day. The patient is discharged on the fourth postoperative day.

RESULTS

Weight loss is one of the best indicators of a successful surgery as it ultimately alleviates comorbidities, and, therefore improves the quality of life of the patient. The mean weight loss at 5 yr for VBG is 58% of the excess body weight. Several series have shown that patients slowly regain a portion of the weight that is initially lost. More than 30% of VBG patients require conversion to another bariatric procedure secondary to erosion of the band, weight gain or inability to tolerate solid food. This is in distinct contrast to results obtained with RYGB. One study of patients 16 yr after RYGB showed that more than 90% of patients had maintained significant weight loss. Approximately 70% of excess body weight will be lost in the first 16-24 mo after RYGB. This may be followed by a period of weight gain of up to 15-20% of excess body weight. Prior to 1990, VGB was the most commonly performed procedure for weight loss. Although mortality and morbidity are comparable between VGB and RYGB, the more substantial amount of weight loss and improved long-term results have recently made RYGB more popular among bariatric surgeons. Within the last decade, RYGB was performed in more than 75% of patients undergoing surgical treatment of obesity in the United States.

Along with the dramatic reduction in body weight is the improvement in the overall health of the patient. More than 90% of the co-morbid problems are more readily controlled or completely alleviated over time postoperatively. One of the most substantial improvements occurs in patients with diabetes mellitus. Almost 90% of patients with adult-onset diabetes or glucose intolerance become euglycemic. This is true for insulin-dependent, as well as noninsulin-dependent diabetics (5). The normalization of glucose metabolism occurred with surprising speed, even before there was significant weight loss. This cannot be attributed solely to the limitation of caloric intake because RYGB patients experience a more significant improvement than do VBG patients. Modification of glucose metabolism may be secondary to changes in enterohormones of the bypassed segment of the GI tract, delayed transit from the stomach, and undigested food in the midjejunum. It has been suggested that RYGB could be the most effective treatment for adult-onset diabetes mellitus (6).

A significant number of patients with morbid obesity suffer from the obstructive sleep apnea syndrome or the obesity-hypoventilation syndrome. There are reports that 60-80%

of these patients do not suffer from these ailments after weight loss. In the same manner, there is a significant improvement in all the pulmonary function tests after the weight loss occurs.

Hypertension is one of the most common medical disorders associated with obesity. Resolution or improvement of diastolic hypertension occurs in approx 70% of individuals, but occurs more commonly in those patients with a lower postoperative BMI. The severity of cardiac dysfunction decreases, as does the degree of dyspnea associated with congestive heart failure. Arthralgia in major joints such as knee, hips, and vertebrae improves rapidly and most significantly with weight loss. There is a clear correlation of these improvements with the chronicity of the condition and the amount of weight loss. Infertility has been corrected with weight loss in a significant number of females. Last, it has been shown that bariatric surgery is the long-term procedure of choice for severely obese patients with pseudotumor cerebri. It has been shown to have a much higher rate of success than cerebrospinal fluid-peritoneal shunting reported in the literature. It is thought that the resolution of intracranial hypertension is related to a decrease in intraabdominal pressure associated with central obesity. Smith et al. reported a decrease mortality in patients undergoing RYGB. Two groups of morbidly obese patients were followed for 8 yr; one group underwent bariatric surgery, whereas the control group did not. Patients in the latter group was five to six times more likely to die from an obesityrelated co-morbidity than those who lost weight following bariatric surgery.

ALTERNATIVE PROCEDURES

Laparoscopy has revolutionized general surgery during the last decade. Every year, more procedures are performed using laparoscopic technique or minimally invasive access approach. Despite the technical differences in access, the operative principles for VBG and RYGB are the same. Studies comparing bariatric procedures using the standard and the laparoscopic techniques have shown the following.

Both VBG and RYGB can be performed safely by laparoscopy. RYGB is technically a more difficult procedure to perform laparoscopically than VBG. Only a few centers in the United States are currently performing laparoscopic RYGB. Average weight loss is the same with both approaches. Postoperative pain and time to return to full preoperative activity are decreased with laparoscopic procedures. Wound infection and incisional hernias are significantly reduced using the laparoscopic techniques. Superobesity and Pickwickian body habitus are relative contraindications to the laparoscopic approach. There is a steep learning curve, especially for the laparoscopic RYGB. Advanced laparoscopic skills are a must. It is estimated that more than 50% of RYGB procedures will be done laparoscopically in the near future (7).

Adjustable banding has recently been introduced in Europe and South America. There have been more than 50,000 procedures performed outside the United States. The use of laparoscopy and the use of inflatable silicone bands have made this procedure popular. The bands are placed laparoscopically and the patient is discharged in 1-2 d. The adjustment is made in the outpatient setting. Weight loss is comparable to the VBG. Perioperative morbidity and mortality are very low. Displacement of the band is not infrequent and pouch dilatation is a potential complication. Many of these complications can be managed by emptying the band, but some may require surgery. Experience with the adjustable band is increasing and thus far results are still short term. The adjustable

band was approved in the United States in the year 2001 and should be part of the repertoire of the bariatric surgeon. It will represent an attractive alternative for those patients that exhibit good compliance and are not "sweet eaters."

Bilio-pancreatic diversion is a procedure used by a small number of bariatric surgeons more commonly in Europe and Canada. Only 1–2% of the surgeons in America perform this procedure. It is a combination of a gastric restriction with malabsorption. The patient can eat almost the normal amount of food, but without absorbing most of the fat and carbohydrate content. The metabolic abnormalities are less than those of jejuno-ileal bypass. Bilio-pancreatic diversion is technically a more demanding procedure than others with a higher incidence of complications. The weight loss is similar to that of RYGB. One of the most undesirable side effects is the uncontrollable flatulence that the patient may develop.

COST

Management of obesity-related medical problems costs Americans more than \$30 billion annually. This does not take into account the amount of money spent on dieting and dietary supplements. The cost of bariatric surgery varies in different parts of the United States and depends on the extent of the preoperative evaluation. At our institution, the average cost for the uncomplicated patient is approx \$12,000. If the procedure is done laparoscopically, it may add \$2,000–\$3,000 to the cost of the operation. We feel this is a viable alternative to the cost of expensive medications and multiple hospital admissions for obesity-related complications.

SUMMMARY

- 1. VBG and RGB have shown to be successful methods for weight loss for the morbidly obese.
- 2. The success rate is based on the relief of medical co-morbidities and patient satisfaction.
- 3. The surgeon should carefully select the procedure based on each patient's needs.
- 4. Education is the key to realistic expectations and patient satisfaction.
- 5. Multidisciplinary approach is crucial to proper preoperative evaluation, patient education, perioperative management, and long-term follow-up.

REFERENCES

- 1. NIH Conference. Gastrointestinal surgery for severe obesity. Consensus development conferences panel. Ann Intern Med 1991;115:956–961.
- 2. Mason EE, Doherty C, Cullen JJ, et al. Vertical Gastrosplasty: Evolution of vertical banded gastroplasty. World J Surg 1998;122:919–924.
- 3. Poires WJ, MacDonald KG. The Surgical treatment of morbid obesity. In: *Current Opinion in General Surgery*. Daly JM, ed., Current Science, Philadelphia, PA, 1993, pp. 195–205.
- 4. Poires WJ, Swanson MS. Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes mellitus. Ann Surg 1195;222:339–352.
- 5. MacDonald DG, Long SD, Swanson MS, et al. The gastric bypass operation reduces the progression and mortality of non-insulin dependent diabetes mellitus. J Gastrointest Surg 1997;1:213–220.
- 6. Rhode BM, Arsenau P, Cooper BA, et al. Vitamin B₁₂ deficiency after gastric surgery for obesity. Am J Clin Nutr 1996;63:103–109.
- 7. Schirmer BD. Laparoscopic bariatric surgery. Surg Clin N Am 2000;80:1253-1267.

12 Percutaneous Enterostomy Tubes

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CONTENTS

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INTRODUCTION

Enteral alimentation is the primary and preferred route for nutritional support. Enteral nutrition maintains intestinal integrity, reduces bacterial translocation, and maintains normal gut flora while preserving the enteral immune system. Furthermore, it has been shown to significantly reduce morbidity and mortality in high-risk surgical patients, and it is more convenient, cost effective, and fraught with fewer complications. Parenteral nutrition should be recommended for patients where enteral feeding is contraindicated or for patients who are unable to meet their nutritional demands (1).

An intact and functional gastrointestinal tract is a major requirement for enteral feeding (2). Several methods for feeding catheter placement have been described and include the following:

- 1. Percutaneous gastrostomy (endoscopic/radiologic)
- 2. Percutaneous jejunostomy (endoscopic/radiologic)
- 3. Percutaneous gastrojejunostomy
- 4. Surgical gastrostomy
- 5. Surgical jejunostomy

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG)

A gastrostomy is a fistulous communication between the stomach and the abdominal surface. It is generally indicated in patients who require supplemental nutrition for more

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Table 1
Indications for Gastrostomy

Impaired swallowing mechanism Facial trauma Neoplasms of the oral cavity or esophagus Inadequate caloric intake Gastric decompression Gastric diversions

Table 2
Contraindications to Gastrostomy

Absolute Contraindications:
Mechanical obstruction
Limited life expectancy
Relative Contraindicatons:
Massive ascites
Continuous peritoneal dialysis
Coagulopathy
Hepatomegaly
Gastric varices
Neoplastic or infiltrative diseases of the stomach
Prior subtotal gastrectomy

than 30 d (3). Dysfunction of the swallowing mechanism as a result of a primary neurologic impairment or disorder (i.e., CVA, dementia, amyotrophic lateral sclerosis, multiple sclerosis) is the most common indication (4) (Table 1). Facial trauma and neoplasms of the oral cavity or esophagus are other indications for PEG placement. It has been advocated in patients with a normal swallowing mechanism who are unable to maintain adequate caloric requirements.

Gastric decompression for malignant intestinal obstruction or advanced carcinomatosis is the second most common indication for PEG placement. It has been utilized for high-risk patients with gastric outlet obstruction owing to multiple abdominal adhesions where surgery is otherwise contraindicated. Furthermore, PEG placement has been used for gastric diversions in patients with radiation enteritis or enterocutaneous fistulas.

Mechanical obstruction and a limited life expectancy are absolute contraindications to PEG placement (2) (Table 2). Relative contraindications to placement include massive ascites, continuous peritoneal dialysis, coagulopathy, portal hypertension, hepatomegaly, gastric varices, neoplastic or infiltrative diseases involving the gastric wall, and a prior subtotal gastrectomy. PEGs can safely be placed in patients with recent abdominal surgery, or through burn wounds or donor sites without an increased risk of complications. The presence of a preexisting ventriculoperitoneal shunt should not preclude PEG placement. Special situations may be encountered, which make placement of a feeding tube technically difficult and riskier, thereby requiring modifications in the standard techniques of tube placement.

Extra precautions should be taken in patients who have had extensive abdominal surgery to avoid inadvertently passing the feeding tube through interposed bowel (5).

PEG tube placement should never proceed unless an adequate access site has been confirmed with finger palpation and transillumination. However, it is often difficult to transilluminate patients who are obese or have a thick abdominal wall. In such patients, an adequate percutaneous site can readily be palpated. A larger incision is often required followed by retraction of the fat tissue until access to the anterior rectus fascia has been obtained, after which placement of a standard PEG tube can proceed using the conventional technique (6). Wound closure can be attained with sutures or surgical clips.

Pregnancy is not a contraindication to PEG tube placement. Safe percutaneous approaches have been demonstrated in case reports as late as 26 wk of pregnancy (7). However, the risk of conscious sedation and the necessity of nutritional support should be strongly considered in these patients. Sedation can be safely administered with the assistance of an anesthesiologist.

Ascites has been considered a relative contraindication to PEG placement because of the associated risks of peritonitis and abdominal fluid leakage. However, large volume paracentesis prior to and a week following PEG tube placement in conjunction with broad spectrum antibiotics have been associated with a good outcome (8). It is important to stress that there have been no prospective clinical trials to confirm the safety of this technique.

Endoscopic Placement

PULL METHOD FOR PEG

A fiberoptic gastroscope is introduced into the patient's mouth, and under direct vision, advanced into the stomach where a complete esophagogastroduodenoscopy is performed to ensure that there is no obstruction or other contraindication to the introduction of a PEG. Under endoscopic visualization, a needle and cannula are introduced through mouth across the anterior abdominal wall through which a guidewire is threaded and pulled with the endoscope in an antegrade fashion through the mouth. The distal end of the gastrostomy tube is attached to the wire loop and with gentle traction is pulled into the stomach through the anterior abdominal wall (Fig. 1). A wide variety of PEG tubes are commercially available with either soft malleable internal bumpers or internal bumpers with inflatable balloons (Fig. 2). The PEG tube is secured using an external bolster. A low profile PEG port is available for mobile patients and eliminates the need for a permanently protruding tube. A separate tube is used for feeding that is attached to a resealable cap (Fig. 3). Repeat endoscopy is occasionally performed to confirm tube placement (2).

PUSH METHOD (SACHS-VINE TECHNIQUE) FOR PEG

The gastrostomy tube is pushed over a guidewire in a retrograde fashion through the mouth and abdominal wall. The feeding tube apparatus is fixed to the abdominal wall as described for pull method. There has been no significant difference regarding success or complications of placement in either method (2).

INTRODUCER METHOD (RUSSEL TECHNIQUE)

An upper endoscopy is carried out in the usual fashion as aforementioned followed by insertion of a needle and cannula through the anterior abdominal wall. A tapered introducer and peel-away sheath is introduced over a guidewire. The tract is subse-



Fig. 1. A typical endoscopically placed PEG tube. A wide variety of internal bumpers are commercially available including soft malleable internal bumpers and internal bumpers consisting of inflatable balloons.

quently dilated and a gastrostomy tube with an inflatable balloon is inserted over the sheath and into the stomach. Once proper positioning is achieved, the sheath is peeled away and the balloon is inflated and pulled against the gastric wall and secured with an external bumper.

ADVANTAGES AND DISADVANTAGES

- 1. Surgery is not required for any of these methods.
- 2. The introducer method is preferable when there is a large obstructing lesion in the pharynx or esophagus that would prevent the passage of the internal bolster into the stomach.
- 3. Pneumoperitoneum associated with tract dilatation in the introducer technique and loss of gastric insufflation are the main disadvantages of this technique. There is no difference in the complication or success rate when compared to the push method (2).
- 4. Flat external bumpers with removable external feeding tubes allow freedom of movement and minimal interference with clothes.

PERCUTANEOUS RADIOLOGICAL GASTROSTOMY

Gastrostomy placement may be achieved under fluoroscopic or computed tomography (CT) guidance. Gastric insufflation is accomplished by insertion of a nasogastric or orogastric tube. The surrounding structures are identified fluorscopically to avoid inadvertent injury. A catheter is then inserted into the distended stomach and a guidewire is passed through the cannula over which the feeding catheter is pushed or pulled.



Fig. 2. Various gastrostomy tubes. (A) A tube with a thin-walled shell internal bolster that collaspes when tension is applied to the tube for removal. (B) A tube with a self-inflating internal bolster that collapses when the tube is transected for removal. (C) A tube with two feeding ports, and a water-filled internal bolster which requires drainage of the water for removal. (D) A tube with one feeding port, and a water-filled internal bolster. (E) A tube with a low-profile external bolster and a water-filled internal bolster.

SURGICAL GASTROSTOMIES

Several modifications in surgical gastrostomy have been developed since its introduction. Available methods include both open and closed techniques.

Open Technique

1. Stamm gastrostomy (5). The Stamm gastrostomy is considered the standard today. The procedure may be performed under general or local anesthesia as part of a major abdominal procedure or for feeding access alone. The aim of the procedure is to create a serosalined fistula between the stomach and the anterior abdominal wall (Fig. 4).



Internal bolster

Fig. 3. A low-profile PEG port. For patients who are mobile, a low-profile PEG port eliminates the need for a permanently protruding feeding tube. A reseatable cap allows attachment of a separate tube for feeding.



Fig. 4. An internally anchored PEG tube. Surgically placed PEG tubes can be anchored to the external surface of the stomach, which may be preferable in situations where there is a problem using the surface of the stomach closest to the abdominal wall.

2. Witzel gastrostomy. The Witzel gastrostomy is a modification of the Stamm gastrostomy. A serosal tunnel is created around the gastrostomy tube to prevent leakage of gastric contents. The anterior gastric wall fixed to the posterior abdominal wall along the length of the serosal tunnel (Fig. 5) (5).

Closed Technique

With the closed technique, placement of the feeding catheter is accomplished laparoscopically. This procedure is performed under general anesthesia. The anterior



Fig. 5. An internally anchored surgically placed jejunostomy tube

gastric wall is lifted and secured to the posterior abdominal wall. The gastrostomy device is inserted and secured to the abdominal wall with U-sutures tied over an external bolster or button (5).

COMPLICATIONS

Procedure related mortality and morbidity are low and comparable to the open techniques (3) (Table 3).

PEG VS RADIOLOGICAL OR SURGICAL GASTROSTOMY

Although studies have shown no difference in morbidity and mortality when comparing surgical gastrostomy to PEG (9), PEG tube placement has been found to be more cost effective and offers the advantage of reducing operative and recovery time. Operative gastrostomies should be reserved for patients in whom endoscopy cannot be performed or when an anatomic aberration precludes a safe percutaneous approach, or for patients who are going to the operating room for another surgical procedure.

The major advantage of percutaneous radiologic gastrostomy over endoscopic gastrostomy is the ability to gain gastric access in the presence of pharyngeal or esophageal obstruction. It likewise prevents oral contamination and can be performed under local anesthesia, thereby providing a cost advantage over PEG. Conversely, clinically
Complications	
Major Complications:	
Aspiration	
Gastrocolocutaneous fistula	
Perforation	
Hemorrhage	
Peritonitis	
Necrotizing fasciitis	
Premature gastrostomy removal	
Tumor seeding of PEG stoma	
Tube migration through gastric wall	
Minor Complications:	
Peristomal wound infection	
Tube leakage/Fragmentation	
Tube migration with obstruction of the pyloric channel	
Tube migration into small bowel	

Table 3

significant findings detected on pregastrostomy endoscopy may lead to changes in medical management.

The majority of the data on PEGs has been obtained from case reports and retrospective studies. A review of data derived from six large series evaluating PEG placement through various methods showed that successful PEG placement was accomplished in 98% of cases. Procedure-related mortality was 0.7%, with aspiration and peritonitis accounting for 90% of cases. Mortality associated with the development of a major complication was 25%. Major complications were found in 1–4% of patients, whereas minor complications were reported in 4–13% of cases. In another more recent review investigating data obtained from 1758 cases, the reported major and minor complication rates were 2.8% and 6%, respectively, (10).

The data on open gastrostomies have demonstrated a wide variation in the reported morbidity and mortality. In a review of the literature, the reported major complication rate for open gastrostomies ranged from less than 2% to as high as 75% with minor complication rates of 0-13% (11).

Major complications following PEG placement include aspiration, perforation, peritonitis, premature gastrostomy tube removal, tube migration, gastrocolocutaneous fistula, hemorrhage, necrotizing fasciitis, and tumor seeding of the PEG stoma. Minor complications are common and include peristomal wound infection, inflammation and leakage around the gastrostomy tube, granulation tissue formation, tube occlusion and fragmentation, and tube migration.

Aspiration Pneumonia

Aspiration pneumonia resulting from PEG placement occurs in 1% of patients and carries with it a very high mortality rate exceeding 50% (11). Risk factors include compromised patient positioning and poor airway management. Perioperative risks are reduced by aggressive evacuation of gastric contents and avoiding excessive sedation and insufflation. In the postoperative period, it is often associated with oropharyngeal aspiration. However, it may result from aspiration of gastric contents. Recommenda-

tions as to when to initiate enteral feeding vary widely. Some authors advocate feeding 12-24 h following gastrostomy tube placement. However, several studies have demonstrated that early feeding within 3-6 h can be safely pursued (12).

Peristomal Leak

Peristomal leakage typically occurs within a few d following PEG placement and is a result of loosening of the external bolster or poor tissue healing and wound breakdown. The latter is usually seen in diabetics and in patients who are severely malnourished. It may likewise result from poor tissue perfusion and subsequent wound breakdown associated with a tight external bolster.

The focus of therapy is aimed at correcting any underlying co-morbidity such as malnutrition or hyperglycemia, loosening of the external bolster, and local measures to prevent wound breakdown (such as powdered absorbing agents or skin protectants such as zinc oxide). Placement of a larger gastrostomy tube through the same PEG tract wound tends to further dilate and distort the tract and retard wound healing, thus compounding the problem. The PEG tube may be removed for 24–48 h to permit slight wound closure prior to reinsertion of a replacement tube through the preexisting tract. This technique is most effective for PEG tube tracts that leak 1 mo following initial placement and are ineffective for patients with early tract leakage, as the majority of these patients develop poor wound healing from their underlying disease process.

In most patients, the PEG tube may have to be removed to permit wound closure to allow placement of a new PEG tube at a different site on the abdominal wall. Placement of a new PEG tube and initiation of feedings with 50% closure of the previous PEG tube tract will not have a significant impact on leakage or inhibition of wound healing through the old PEG site (13).

Pneumoperitnoneum

Pneumoperitnoneum is a common finding following PEG placement and in the absence of peritoneal signs should not be an indication to withhold or discontinue enteral feeding. It is felt to be a consequence of gastric insufflation associated with the endoscopic procedure and needle puncture of the gastric wall. Subcutaneous air resulting from air introduced between the cutaneous and subcutaneous tissues has likewise been described and in the absence of other findings should not preclude enteral feeding (14).

Gastroparesis

Some patients may develop a transient gastroparesis following PEG tube placement resulting in nausea and vomiting. In rare instances, patients with significant pneumoperitoneum may develop an ileus requiring bowel rest and nasogastric decompression. Clinical manifestations include the presence of postprocedure abdominal distention, vomiting, and the absence of bowel sounds. In this subset of patients, it is imperative to exclude the presence of a gastric or duodenal perforation. Enteral feeding should be held until resolution of the ileus occurs (13).

Tube Obstruction

One of the most frequently encountered problems is tube dysfunction secondary to clogging from medications or enteral formula. All medications should be dissolved in water or administered in liquid form if at all feasible. The importance of flushing water

through the PEG tube following delivery of medications and enteral feedings should be reinforced to both the patient and the caregivers. Bulking agents such as psyllium and certain resins such as cholestyramine should never be given through the PEG (13). In occasions when tube occlusion does occur, flushing the tube with a 60-cm^3 syringe is recommended. Warm water is the best irrigant, and is superior to other liquids such as juices or colas (14). In the event this fails, a PEG tube brush can be used to clear the obstruction (13).

Deterioration of the PEG tube as characterized by the presence of pitting, ballooning and a characteristic smell is another common cause of tube dysfunction. This may result in leakage or tube breakdown, making tube feedings difficult or impossible. Yeast implantation on the wall of the tube has been demonstrated to result in this problem.

Peritonitis

Inadvertent and premature removal of the PEG prior to tract maturation results in peritonitis in 0-1% of cases. It may likewise result from perforation of a viscus, preexisting gastric ulcer and leakage around the gastrostomy site. Emergent operative management is indicated in the presence of fever, leukocytosis, abdominal pain, and tenderness. In the absence of peritoneal signs, immediate PEG replacement may be accomplished endoscopically. If the location of the tube remains in question, a fluoroscopic study with a water-soluble contrast agent infused through the PEG should be performed to confirm tube position and to demonstrate the presence or absence of a leak (15).

Hemorrhage

Hemorrhage is a rare complication of PEG placement and occurs in 0-2.5% of cases. It may result from trauma to the esophageal or gastric mucosa, peptic ulcer disease, or trauma to a gastric vessel. Therapy is aimed at applying traction with the internal bumper to tamponade the bleeding vessel, and correcting any underlying coagulopathy. Traction should not exceed 48 h to avoid PEG tube tract wound breakdown. Surgical intervention is rarely necessary (13).

Infection

Peristomal wound infections are one of the most common complications of PEG placement and occur in as many as 8-30% of patients. Antibiotic prophylaxis has been demonstrated to significantly reduce the risk of peristomal wound infections. A single prophylactic dose of Cefazolin administered 30 min prior to PEG placement has been shown to reduce peristomal wound infections significantly from 28.6% to 7.4% (16,17).

Necrotizing fasciitis is a potentially fatal complication if not diagnosed early and treated with expedient and aggressive surgical debridement. It is evident 3–14 d following PEG placement and is characterized by high grade fevers, skin edema followed by cellulitis, and crepitance. It is associated with small abdominal incisions, excessive traction, and lack of prophylactic antibiotics (18). Patients with an impaired immune system, diabetes, malnutrition, and wound infections are at higher risk.

LATE COMPLICATIONS

Tube Migration

Migration of the gastrostomy tube through the gastric wall followed by re-epithelization over the internal bumper or the "buried bumper syndrome" is another complication of PEG

placement. This complication has been reported with earlier tube designs utilizing rigid internal bumpers. It results from excessive pressure between the internal and outer bumper resulting in pressure necrosis and ulceration with tube migration towards the anterior abdominal wall. It usually manifests 3-4 mo following PEG placement and is associated with abdominal pain, resistance to feeding, peritubal leakage, and resistance to tube manipulation. It may occasionally present as peritubal wound infections, necrotizing soft tissue infections, and abscess formation. Therapy consists of prompt tube removal (19). If the internal bumper is collapsible, the PEG tube can be removed with gentle external traction. Rigid internal bumpers on the other hand, may have to be removed by PEG-wound tract cut down or endoscopically using the push-pull T technique. With the push-pull T technique, the external portion of the PEG is cut and pulled with a snare and with the assistance of a second operator is simultaneously pushed into the gastric lumen and endoscopically removed. Once the PEG has been removed, a new PEG tube can be inserted through the preexisting tract under endoscopic surveillance. Careful catheter care with specific attention to excessive traction limits this complication. The external bumper should be maintained against the anterior abdominal wall and gauze pads should be placed over the external bumper and not beneath, so as not to create additional pressure on the PEG tube. In addition, the PEG tube should be pushed forward and rotated during daily nursing care to ensure that the internal bumper had not eroded into the gastric mucosa. It is advisable to return the PEG to its original position after rotation.

Fistula Formation

Gastrocolocutaneous fistulas are rare, but potentially serious complications following PEG tube placement, which result from inadvertent injury to the colon at the time of PEG insertion (20). They may manifest several months following initial placement as a result of delayed colonic injury from tube migration and erosion into the colon. Acutely, patients may present with peritonitis, peristomal wound infections, necrotizing fascitiis or bowel obstruction. Severe diarrhea resembling tube feeding as a result of placement of the replacement catheter into the colonic lumen may likewise be seen. Diagnosis is made with contrast studies and treatment involves removing the catheter and replacement once the fistulous tract closes. Surgery may be necessary to correct the internal gastrocolonic fistula.

Prevention of this complication entails a combination of adequate transillumination and finger palpation of the abdominal wall in choosing an appropriate site rather than either technique alone. When adequate positioning remains uncertain, an 18–22 gage needle attached to a syringe may be passed through the chosen PEG site prior to PEG tube insertion. The presence of a sudden gush of air or stool into the syringe as the plunger is withdrawn may suggest the presence of interposed bowel. This technique, however, has not been subjected to a prospective evaluation.

Granulation Tissue

Polypoid granulation tissue can develop from sc tissue at the ostomy site. Such tissue can bleed and drain making the area difficult to keep dry. Silver nitrate cauterization after xylocaine jelly is applied for local anesthesia is usually quite satisfactory.

Tube Removal

Removal of PEG tubes intentionally or inadvertently is usually followed by prompt wound closure. Tract maturation generally occurs within 1 wk following insertion, but may be delayed in patients who are severely malnourished or who are on steroids. PEG tubes that are removed within the first 4 wk following PEG tube placement should not be reinserted blindly at the bedside because the PEG tube tract may have not matured adequately and may result in PEG tube placement within the peritoneal cavity. A contrast study should always be performed to confirm proper tube position prior to initiation of enteral feeding if tube position remains uncertain. If the replacement tube lies within the peritoneal cavity it should be removed immediately and placement may be accomplished endoscopically through the preexisting PEG tube site. Prompt replacement of the feeding catheter through a mature tract is recommended because the gastrocutaneous tract closes within 24 h. Replacement catheters may consist of Foley catheters or commercially available replacement catheters.

PEGs should be removed in patients who no longer require enteral nutrition or in patients with peristomal wound infections, gastrocolocutaneous fistulas, tube malfunction, and peristomal leakage because of progressive enlargement of the fistulous tract (21).

Several methods of tube removal are available depending of the configuration of the internal bumper (Fig. 2). PEG tubes with stiff and rigid bumpers often require endoscopic removal. Some authors have advocated cutting the PEG tube at skin level thereby allowing the retained piece to pass through the stool. However, complications arising from the retained piece have been reported (22). PEGs with soft and malleable internal bumpers may be pulled through the stoma, thus obviating the need for endoscopic removal. Tubes with inflatable internal bolsters like Foley catheters need to be deflated by suction at the port. Self-inflating bolsters require cutting of the tube to allow deflation, prior to traction removal.

The term PEJ is used to imply placement of a feeding catheter through a gastrostomy tube into the jejunum. Regardless of the method of jejunostomy tube placement, indications include tracheal aspiration, partial or complete gastric resection, gastric pull up, gastroparesis, postoperative feeding during major operative procedures, occluded or nonfunctioning gastrojejunostomy, and gastric outlet obstruction owing to a gastric or pancreatic mass (23).

As aforementioned, aspiration pneumonia is a serious medical complication associated with a high mortality rate. Often, it is difficult to distinguish between aspiration as a result of aspirated oropharyngeal secretions and refluxed gastric contents. Although it has been suggested that jejunal feedings reduce the risk of aspiration, a review of the literature analyzing aspiration associated with gastric and jejunal feedings has been inconclusive (24).

Patients who have had a previous gastric resection often lack a gastric reservoir (25). The high location of the stomach within the rib cage makes PEG tube placement technically difficult because of the limited capacity to transilluminate the abdominal wall. The same holds true for patients who have had a gastric pull-up following esophageal resection. In addition, these patients have a higher risk of aspiration.

Abnormalities in gastric motility occur in a variety of disorders including diabetes and certain neurologic disorders such as Parkinson's and multiple sclerosis. Enteral feeding through a jejunostomy tube allows delivery of nutrients beyond the malfunctioning stomach (Table 4).

Jejunostomy tubes may be inserted endoscopically or surgically. Placement of a PEJ requires initial placement of a 20- to 28-F gastrostomy tube through which an 8- to 12-F jejunostomy tube is inserted and threaded endoscopically into the distal duodenum or jejunum (Fig. 6). PEJ placement is limited by the technical difficulty associated with

Table 4		
Indications for]	-Tube	

Tracheal aspiration Gastroparesis Partial or complete gastric resection Gastric pull-up Postoperative feeding during major operative procedures Occluded or malfunctioning gastrojejunostomy Gastric outlet obstruction owing to gastric or pancreatic mass



Fig. 6. An endoscopically placed G-J tube. For patients in whom feeding infusions directly into the stomach are contraindicated, e.g., gastroesophageal reflux, a J-tube can be placed through a gastrostomy tube to permit infusion directly into the duodenum/jejunum.

inserting the tube distally into the distal duodenum or jejunum and frequent tube migration proximally into the stomach (23).

Several techniques for surgical jejunostomy have been described and the current procedures include the following: Witzel jejunostomy, Roux-en-Y jejunostomy, needle catheter jejunostomy, button jejunostomy, and percutaneous peritoneoscopic jejunostomy.

The Witzel jejunostomy entails the creation of a 2- to 4-cm serosal tunnel between the proximal jejunum and abdominal wall. The length of the seromuscular tunnel is subsequently affixed to the abdominal wall and the external portion of the catheter secured to the skin with a suture (Fig. 7) (5). The disadvantage of this technique is the potential for small bowel obstruction associated with larger balloon catheters in view of the narrower small bowel lumen and migration of the catheter distally.

In a Roux-en-Y jejunostomy, the jejunum is cut approx 20 cm distal to the ligament of Treitz and the proximal end is anastomosed to the distal jejunum, creating a short limb. The free end is allowed to mature externally through a permanent stoma or attached to the abdominal wall following insertion of a mushroom catheter, Foley catheter, or skin level device (Fig. 8). This procedure offers the best long-term results for jejunal feeding (5).

Laparoscopic jejunostomies require the induction of general anesthesia. A loop of jejunum is brought to the posterior abdominal wall under laparoscopic surveillance and is secured to the abdominal wall with a bolster or clamp. A needle is inserted through the



Fig. 7. An externally anchored surgically placed J-tube.



Fig. 8. A Roux-en-Y jejunostomy with a low-profile port.

abdominal wall and into the jejunum followed by insertion of a feeding catheter over a guidewire into the jejunum with the introducer (Fig. 9).

Laparoscopic jejunostomies are safe and efficacious and may be placed perioperatively at the time of laparoscopic gastrostomy for gastric decompression (26). The



Fig. 9. An externally anchored surgically placed J-tube.

incidence of conversion to an open jejunostomy is higher in patients with prior abdominal surgery (27).

COSTS

1. G-Tubes:

Surgical G-tube costs approx \$3500 including anesthesia. Endoscopic G-tube costs approx \$2300. Radiological G-tube costs approx \$600.

2. J-Tubes:

Surgical J-tube costs approx \$3500 including anesthesia. Endoscopic GJ-tube costs approx \$2600. Radiological J-tube costs approx \$600.

SUMMARY

- 1. Whenever possible, enteral rather than parenteral feeding should be used in patients requiring nutritional support as it is essential for the integrity of intestinal tract, gut immune response, and is associated with fewer complication.
- 2. In patients with deglutitive dysfunction, enteral nutrition can be provided by percutaneous gastrostomy tubes, which can be placed endoscopically, radiologically, or by open surgery.
- 3. Gastrostomy tubes are usually placed in the stomach. However, in patients at a higher risk of aspiration or previous gastric surgery, these can be placed in the jejunum.
- 4. Placement of gastrostomy tubes is technically easy and well tolerated with very few short or long-term complications.

REFERENCES

- 1. McClave SA, Lowen CC, Snider H. Immunonutrition and enteral hyperalimentation of critically ill patients. Dig Dis Sci 1992;37:1153–1161.
- Safadi B, Marks J, Ponsky J. Percutaneous endoscopic gastrostomy. Gastrointest Endosc Clin N Am 1998;8:551–568.
- 3. AGA technical review. Enteral nutrition part 2: 2000 Uptodate.www.uptodate.com: 1.

- 4. Ponsky JL, Gauderer MWL. Percutaneous endoscopic gastrostomy: a nonoperative technique for feeding gastrostomy. Gastrointest Endosc 1981;27:9–11.
- 5. Foutch PG, Talbert GA, Waring JP, et al. Percutaneous endoscopic gastrostomy in patients with prior abdominal surgery: Virtues of the safe tract. Am J Gastroenterol 1988;83:147–150.
- 6. Bender JS. (1992) Percutaneous endoscopic gastrostomy placement in morbidly obese (letter). Gastrointest Endosc; 38(1):97–98.
- Sheehan NJ, Crosby MA, Grimm IS, et al. The use of percutaneous endoscopic gastrostomy in pregnancy. Gastrointest Endosc 1997;46:564–565.
- 8. Kynci JA, Chodash HB, Tsang TK. Peg in patient with ascites and varices (letter). Gastrointest Endosc 1995;42:100–101.
- 9. Steigmann GV, Goff JS, Silas D, et al. Endoscopic versus operative gastrostomy: Final results of a prospective randomized trial. Gastrointest Endosc 1990;36:1–5.
- 10. Foutch PG. Complications of percutaneous endoscopic gastrostomy and jejunostomy: Recognition, prevention and treatment. Gastrointest Clin N Am 1992;2:231.
- 11. Shellito PC, Malt RA. Tube gastrostomy: Technique and complications. Ann Surg 1985;201:180–185.
- 12. Choudry U, Barde CJ, Markert R, et al. Percutaneous endoscopic gastrostomy. A randomized prospective comparison or early and delayed feeding. Gastrointest Endosc 1996;44:164–167.
- Delegge MH. Prevention and management of complications from percutaneous endoscopic gastrostomy. Rose B, ed., UpToDate Inc., Wellesley, MA, 2000, Version 10-2.
- 14. Stathopoulus G, Rudberg MA, Harig JM. Subcutaneous emphysema following PEG. Gastrointest Endosc. 1991;37:374–376.
- Bender JS, Levison MA. Complications after percutaneous endoscopic gastrostomy removal. Surg Laparosc Endosc 1991;1:101–103.
- Panos MZ, Railly H, Moran A, et al. Percutaneous endoscopic gastrostomy in a general hospital. Prospective evaluation of indications, outcome and randomized comparison of two tube designs. Gut 1994;35:1551–1556.
- 17. Jain NK, Larson DE, Schroeder KW, et al. Antibiotic prophylaxis for percutaneous endoscopic gastrostomy. A prospective randomized double blind clinical trial. Ann Int Med 1987;107:824–828.
- Greif JM, Ragland JJ, Ochsner MG, et al. Fatal necrotizing fasciitis following percutaneous endoscopic gastrostomy. Gastrointest Endosc 1986;32:292–294.
- Klein S, Heare BR, Soloway RD. "Buried bumper syndrome", a complication of percutaneous endoscopic gastrostomy. Am J Gastroenterol 1990;85:448–451.
- 20. Saltzberg DM, Anand K, Juvan P, et al. Colocutaneous fistula: An unusual complication of percutaneous endoscopic gastrostomy. JPEN 1987;11:86–87.
- 21. Ponsky JL. Percutaneous endoscopic gastrostomy: Techniques of removal and replacement. Gastrointest Endosc Clin N Am 1992;2:215.
- 22. Wilson WCM, Zenone EA, Spector H. Small intestinal perforation following replacement of a percutaneous endoscopic gastrostomy tube. Gastrointest Endosc 1992;36:62–63.
- Shike M, Latkany L. Direct percutaneous endoscopic jejunostomy. Gastrointest Endosc Clin N Am 1998;8:569–580.
- 24. Lazarus BA, Murphy JB, Culpepper L. Aspiration associated with long-term gastric versus jejunal feeding: A critical analysis of the literature. Arch Phys Med Rehabil 1990;71:46–53.
- 25. Tsuburaya A, Noguchi Y, YoshikawaT, et al. Long term effect of radical gastrectomy on nutrition and immunity. Surg Today 1993;23:320–324.
- 26. Sangster W, Swanstrom L. Laparoscopic guided feeding jejunostomy. Surg Endosc 1993;7:308-310.
- 27. Hotokezaka M, Adams RB, Miller AD, et al. Laparoscopic percutaneous jejunostomy for long term enteral nutrition. Surg Endosc 1996;10:1008–1011.

III SMALL BOWEL SURGERY

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CONTENTS

INTRODUCTION INDICATIONS CONTRAINDICATIONS DESCRIPTION OF SMALL BOWEL RESECTION PHYSIOLOGICAL CHANGES SHORT BOWEL SYNDROME COMPLICATIONS AND MANAGEMENT ALTERNATIVES AND COSTS SUMMARY REFERENCES

INTRODUCTION

The small intestine is an absorptive organ that plays a critical role in digestion. In an adult, the small intestine is 3–8 m long (average of 620 cm or approx 22 ft) (1) in vivo with a microscopic mucosal architecture that consists of innumerable villi, which create a tremendous absorptive surface area. Whereas 8–10 L of fluid enter the small bowel daily, only 500 mL to 1.5 L make it to the cecum. In addition to the efficient absorption of water, the absorption of simple sugars, small peptides, amino acids, chylomicrons, and lipid micelles occur in the small intestine. Finally, the absorption of vitamins and minerals critical to many physiologic processes also occurs here. Surgical diseases of this organ are quite uncommon. In fact, the most common operation involving the small intestine is lysis of adhesions for small bowel obstruction. Usually, there is no small bowel resection during that operation. Fortunately, the small intestine has plenty of reserve and resections of short segments are well tolerated.

INDICATIONS

Small bowel resections are most commonly performed for benign disease. The most common of these are intestinal ischemia and Crohn's disease. Intestinal ischemia may be a local phenomenon involving vascular compromise of a solitary loop of small intes-

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tine. Examples of this situation include strangulation of an inguinal hernia or volvulus of a loop of small bowel around an adhesion. In these situations, the loop of involved intestine can be resected and a primary anastomosis is performed. These patients generally do extremely well postoperatively. In contrast, patchy or widespread intestinal ischemia may be caused by embolism, mesenteric arterial or venous thrombosis, a nonocclusive (low flow) state, or midgut volvulus secondary to a congenital malrotation. In these cases, long lengths of ischemic intestine may require resection. With widespread mesenteric ischemia there is a significant mortality rate that can be as high as 60-70% (2,3). The patients that do survive may be left with a length of small intestine that is inadequate for absorptive requirements, resulting in the short bowel syndrome. This situation will be discussed in a subsequent section.

Crohn's disease is a transmural inflammatory bowel disease of unknown etiology that primarily affects the small intestine. The majority of patients are managed medically and surgery for Crohn's disease is intended only to palliate symptoms and treat complications (4). Complications of Crohn's disease that require surgery include stricture formation, bowel obstruction, hemorrhage, perforation, abscesses, and fistulization. Patients with Crohn's disease often require multiple operations (5). Therefore, operative strategies are designed to limit bowel resections in order to preserve intestinal length. At surgery, only grossly diseased bowel is resected. Frozen section examinations are not needed because histology does not impact the incidence of recurrent disease. Stricturoplasty is a technique that enlarges the lumen without a resection. Stricturoplasty is routinely used in cases of stricture formation to avoid excessive small bowel resections.

Small bowel tumors are quite uncommon. Primary small bowel tumors are divided almost evenly between benign and malignant lesion. Benign lesions include leiomyomas, adenomas, and lipomas. Primary malignancies of the small bowel include adenocarcinoma, the most common at 50%, lymphoma, leiomyosarcoma, and carcinoid tumor. Some malignant tumors such as melanoma or lymphoma may metastasize to the small intestine. Patients with a small bowel tumor may present with bowel obstruction or bleeding. The tumor can serve as a lead point for an intussusception, which usually results in intermittent intestinal obstruction. In cases of obstruction, the offending lesion is easy to find intraoperatively by simple palpation of the bowel. When small tumors hemorrhage, localization may be difficult and may require intraoperative enteroscopy. Although some benign small bowel tumors are amenable to endoscopic removal, the majority will require a segmental small bowel resection.

Meckel's diverticulum is a congenital, true diverticulum, which occurs in the distal two feet of ileum. The majority of Meckel's diverticula remain asymptomatic and thus undetected during the patient's life. Meckel's diverticula may cause symptoms including gastrointestinal bleeding, perforation, or small bowel obstruction. Thus, complications of Meckel's diverticula are another set of rare indications for small bowel resection. Controversy does exist as to whether an asymptomatic Meckel's diverticulum should be resected if found incidentally during an abdominal operation (6,7).

CONTRAINDICATIONS

There are no common contraindications that are specific to small bowel resection. As with any major surgical operation, a patient's medical condition could contraindicate a surgical procedure under general anesthesia.

There are several uncommon situations that can specifically contraindicate small bowel resection. One is the situation where a patient's peritoneal cavity has been obliterated by peritonitis, radiation, or multiple abdominal operations. In this situation, the intestines are fused to each other and to the abdominal wall with dense adhesions. These cases of a "frozen abdomen" carry an increased risk of creating enterotomies during surgery. Despite repairing an enterotomy, the patient is at risk for leakage and/or fistula formation from the suture line. It can be difficult for the surgeon to judge when to forge ahead in such situations and when to back out.

A second situation where small bowel resection may be contraindicated is when a patient is at risk for the short bowel syndrome. With this condition, the patient has inadequate intestinal length to fulfill absorptive functions needed to sustain adequate nutrition. These situations can arise in patients who have had multiple small bowel resections for Crohn's disease or if a patient requires resection of a great length of small intestine as a result of mesenteric ischemia. In cases of intestinal ischemia for which intestinal length may be of issue, the smallest resection of only grossly irreversibly ischemic bowel should be done at the initial operation. A planned "second look" operation is performed at 24– 26 h postoperatively so that areas of ischemia have time to become clearly demarcated and the maximum amount of small intestine is preserved.

A third situation is when a patient's small bowel is obstructed secondary to an unresectable intraabdominal malignancy such as a colorectal or ovarian cancer. In such a case, the only option may be to palliate with an intestinal bypass of the involved segment without resection. Creation of an ostomy proximal to a distal obstruction would also serve as a means of palliation.

DESCRIPTION OF SMALL BOWEL RESECTION

Thanks to the profuse collaterals within the mesenteric arterial arcades, surgeons may resect segments of small bowel anywhere along its length with little concern of compromising the blood supply. This is in contrast to colon resection, where the blood supply must be carefully considered. The resection margins are selected and the small bowel is divided proximally and distally with a linear stapler. The mesentery is divided between hemostats and the contents of each hemostat are ligated with suture material. Because the small intestinal lumen is usually no greater than 1 inch in diameter, on occasion, surgeons will find that they have compromised the lumen after a hand-sewn end-to-end anastomosis. Therefore, many surgeons have adopted stapling techniques over suturing for small intestinal anastomoses. The technique of side-to-side linear stapling results in a "functional end-to-end" anastomosis. This technique is outlined in Fig. 1A–F.

At times an ileostomy must be constructed during intestinal surgery. This is most commonly done during colonic surgery as opposed to surgery on the small bowel. Ileostomies are used temporarily after a colon resection if an ileum to colonic anastomosis is deemed unsafe because of poor condition of the bowel (ischemia, edema, inflammation) or because of factors such as fecal contamination of the peritoneal cavity. An ileostomy may also be constructed as a permanent stoma after a total proctocolectomy for ulcerative colitis or familial polyposis. An ileostomy may also be constructed to divert the fecal stream away from a tenuous distal colo-colonic anastomosis. Such a temporary ileostomy is also frequently used after creation of a J-pouch with ileoanal anastomosis subsequent to a total colectomy for ulcerative colitis or familial polyposis.



Fig. 1. (A) View of small intestines after a segmental resection. (B) View of small intestines lined up for formation of an anastomosis using a linear stapler, (note silk sutures to maintain configuration). (C) Diagram of a linear stapler (assembled). (D) View of small intestines with the jaws of the linear stapler within the two limbs of bowel. (E) The stapler is fired. The instrument places parallel staple lines and cuts the common wall in between staple rows thus creating a large common lumen. (F) The open end is stapled or sutured closed resulting in side-to-side anastomosis.



Fig. 2. (A) Ileum is brought up through the abdominal wall. Sutures are placed as described in the text. (B) Sutures are tied thus everting the ileum and creating a manageable ileostomy.

The ileostomy in this circumstance would be taken down during a future second operation, once the colonic (or ileoanal) anastomosis has healed. The method of creation of an ileostomy is illustrated in Fig. 2. Suture placement is designed to evert the ileum thus creating the "rosebud" or nipple appearance. These sutures involve three tissue bites, skin (or dermis), outer wall of ileum (3 to 4 cm from the open end), and finally a full thickness bite through the intestinal wall at the open end. Tying these three-bite sutures creates the eversion. This everted configuration allows for a tight-fitting stomal appliance and good skin protection from the effluent.

Patients with ileostomies are prone to dehydration and electrolyte abnormalities. These complications occur most commonly during the first few months after surgery. As time goes on, physiologic adaptation and behavioral (dietary) adaptations occur and complications became less frequent.

PHYSIOLOGICAL CHANGES

The small intestine is an essential organ in digestion. As aforementioned, microscopic examination of the mucosal surface reveals the remarkable topography of endless villi, which create a tremendous surface area for absorption of water and nutrients. Certain segments of the small intestine preferentially absorb specific nutrients, vitamins, or minerals. For example, iron is absorbed primarily in the duodenum. Calcium and folate are both most avidly absorbed by the proximal small bowel. Conversely, bile salts, the fat-soluble vitamins A, D, E, and K, and intrinsic factor bound vitamin B_{12} are preferentially absorbed by the terminal ileum. These facts carry clinical import in patients having various segments of small bowel resected. In patients who have extensive ileal resections, monthly B_{12} injections may be required for to prevent anemia or other effects of deficiency (1,8). In addition, oral supplementation of vitamin D and calcium may be required to prevent osteomalacia (1,8). In general, proximal small intestinal resections are better tolerated than distal resections because the distal ileum has superior adaptive capabilities.

SHORT BOWEL SYNDROME

Short bowel syndrome (SBS) has been defined as having an inadequate small bowel length with associated malabsorption. The syndrome is characterized by watery diarrhea, dehydration, fluid and electrolyte abnormalities, and malnutrition. SBS usually occurs if greater than 70% of the small bowel has been resected or if less than 180 cm of the small bowel remains (9). These numbers vary depending on whether it is jejunum or ileum remaining, with the latter being preferable. Preservation of the ileocecal valve is also physiologically beneficial and allows patients to tolerate a greater length of small bowel resection.

Patients with SBS not only have an inadequate absorptive surface but also have an increased intestinal transit time. These patients have an elevated serum gastrin level and the excess gastric acid that is produced exacerbates the diarrhea (1,8). The mechanism for the hypergastrinemia is not known and this state is usually transient (1,8). In addition, the loss of brush border hydrolases causes inadequate carbohydrate breakdown, contributing to osmotic diarrhea. If the terminal ileum has been resected, bile acids are not well absorbed, which results in sodium and water secretion in the colon, again adding to diarrhea. Loss of the bile acid pool will cause steatorrhea and malabsorption of fatsoluble vitamins (A, D, E, and K). This disruption of the enterohepatic circulation of bile can lead to both cholesterol gallstones and oxalate kidney stones.

Thirty-five to forty percent of patients with SBS have been found to develop gallstones (10). Risk factors for the development of cholelithiasis in patients with SBS include small intestinal length less than 120 cm, absent ileocecal junction, long-term total parenteral nutrition (TPN), and Crohn's disease (10). Cholestasis secondary to the use of TPN contributes to the formation of gallstones, although the administration of cholecystokinin may help to prevent biliary stasis. In stool, oxalate usually binds to intraluminal calcium to form an insoluble complex and is excreted. With fat malabsorption, calcium binds to free fatty acids resulting in large amounts of unbound oxalate. The free oxalate is absorbed in the colon and is eventually concentrated in the kidney leading to stone formation. Patients with hyperoxaluria should be placed on a low-oxalate lowfat diet to decrease urinary oxalate (1). In addition, cholestyramine can be added to bind free intraluminal oxalate and supplemental enteral calcium given to increase calciumoxalate binding (1).

Patients with short bowel syndrome are difficult to manage and often endure long periods of hospitalization. These patients may require long-term or even life-long total parenteral nutrition to meet their nutritional requirements. Some patients experience enough physiologic adaptation to recover and sustain themselves with enteral feedings. Others require periodic intravenous fluid and dietary supplementation. Home health care has progressed enough that many patients may receive intravenous supplementation at home, thus avoiding repeated hospitalization.

Medical therapy with H2 blockers or proton pump inhibitors can control gastric hypersecretion (1,8). Octreotide and antidiarrheal medications such as loperamide and

narcotics may be helpful by decreasing stool volume and intestinal transit time (1,8). Ultimately, getting nutrients into the gut lumen is important to both physiologic adaptation and to ultimate recovery. The addition of glutamine to the diet with parenteral growth hormone may be beneficial to the mucosa and reduce or eliminate parenteral nutrition needs in some patients (11).

Surgery for SBS is reserved for patients who have continued malabsorption and malnutrition despite maximum medical therapy or problems related to TPN. These problems include sepsis, venous thrombosis, liver injury, and high costs. Surgical options are designed to slow intestinal transit, optimize intestinal function, and increase surface area. The segmental reversal of a short segment (10 cm) of small bowel is an option that can slow intestinal transit time and may decrease or eliminate TPN requirements (12). As aforementioned, preservation of the ileocecal valve is beneficial and all ostomies should be closed if possible. These maneuvers can increase absorption and intestinal transit time (9). In the worst cases of SBS, small intestinal transplantation may be the only answer. The majority of these operations are performed on children with a 5-yr patient survival of approx 50% (1). Thus, this operation should be reserved for only those patients with life-threatening TPN complications without another surgical option.

COMPLICATIONS AND MANAGEMENT

Although short bowel syndrome only occurs in cases of massive small intestinal resection, there are several complications that may occur even with resections of short segments of small bowel. In cases where small bowel resection was limited (less than 1 m resected), patients may have diarrhea, dehydration, and electrolyte abnormalities in the early postoperative period. These cases are easy to manage with adequate hydration and replacement of electrolytes. Physiologic adaptation within the remaining small bowel will occur within weeks to a few months postsurgery. Additional and more problematic complications include anastamotic leak, enterocutaneous fistula formation, stricture, and postoperative small bowel obstruction.

There are three essential requirements for a successful intestinal anastomosis. The requirements are an adequate lumen, an adequate blood supply, and a lack of tension. Tension on the suture or staple line or an inadequate blood supply may result in a leak of the anastomosis. Leakage from a small bowel anastomosis may be clinically insidious at first compared to a colon leak. This is because of the smaller bacterial load in the small bowel. Eventually, most anastamotic leaks will present with signs of sepsis including tachycardia, oliguria, fever, abdominal pain, and leukocytosis. Such cases typically require urgent reoperation with revision or reconstruction of the anastomosis. At times, the creation of an ostomy is indicated because there is a high rate of anastamotic failure in the setting of sepsis and contamination within the abdomen. A small, contained leak may manifest itself as an abscess or as an enterocutaneous fistula through the surgical incision. If the leak seals, an abscess may be drained percutaneously with CT scan or ultrasound guidance. Most leaks, however, do require reoperation.

Enterocutaneous fistulas are more common in cases where small bowel has been resected for Crohn's disease or in cases where the small bowel has been previously irradiated. These fistulas are classified as high-, moderate-, or low-output depending on the volume over 24 h. High-output fistulas are those that put out more than 500 mL over 24 h (13). High-output fistulas are less likely to close with supportive measures. Low-

output fistulas put out less than 200 mL per 24 h (13) and are prone to spontaneous closure over time. Factors known to hinder healing and closure of fistulas include distal bowel obstruction, foreign body presence, malignancy, undrained adjacent abscess, short fistula tract to the skin, and wide-mouthed fistulas. Patients who are on steroids or have irradiated bowel have diminished healing capabilities and are also less likely to have spontaneous closure of their fistula.

The treatment for patients who develop enterocutaneous fistulas is based on multiple factors. A septic patient may require early operation for resection of the involved bowel segment and drainage of any abscesses. An abscess in a stable patient may be amenable to percutaneous drainage. Otherwise, the patient is classified by fistula volume as aforementioned. It is obligatory to rule out a distal bowel obstruction with the use of contrast radiographic studies. Patients with low or moderate volume fistulas with no distal obstruction and no sepsis may be treated with supportive care. They should be made NPO and be placed on TPN to support their nutritional needs. Some patients can progress to taking elemental oral dietary supplements during their treatment. Fistula drainage should be controlled if possible with the use of an ostomy bag to prevent skin irritation and breakdown. Octreotide, the somatostatin analogue, slows gastric emptying and small bowel transit, and decrease fistula output and expedite spontaneous closure in some patients (13). When a fistula does not close by 4-6 wk, operative resection is usually necessary to remedy the problem (13).

Early postoperative small bowel obstruction may be an aggravating complication after small bowel resection. This is an obstruction occurring within 30 d of an operation that diagnostically, can be difficult to differentiate from a postoperative ileus. These early obstructions may be managed conservatively with intravenous hydration and nasogastric decompression. The majority of these cases do resolve with this conservative management although some patients may require reoperation.

ALTERNATIVES AND COSTS

A review of the list of indications for small bowel resection (ischemia, tumor, Crohn's disease, Meckel's diverticulum) reveals a situation where nonsurgical options are few. At first glance, one would point out Crohn's disease as a disorder with multiple treatment options. In reality, however, Crohn's disease is a medical, not a surgical, problem. Patients require surgery because of complications associated with Crohn's disease (obstruction, abscess, bleeding, and so on) not on Crohn's disease *per se*. So, in fact, there are not any critical cost issues concerning surgery for disorders of small intestine.

SUMMARY

- 1. The small intestine is an important part of the gastrointestinal system and is essential for survival.
- 2. Surgical diseases of the small intestine are unusual. Most small bowel resections are for benign disease.
- 3. The ample physiologic reserve created by the great length and absorptive surface area of the small bowel allow for resections of short intestinal segments without physiologic consequences.
- 4. Patients requiring resection of significant lengths of small intestine (e.g., greater than 50%) will be affected by changes in physiology. Loss of great lengths of small bowel is usually

due to mesenteric ischemia and less commonly from multiple resections for Crohn's disease over the life of the patient. Such patients may experience "short bowel syndrome," characterized by severe fluid and electrolyte abnormalities in addition to malnutrition.

REFERENCES

- 1. Scolapio JS, Fleming CR. Nutrition and specific gastrointestinal disease states, short bowel syndrome. Gastroenterol Clin 1998;27:467–479.
- 2. McKinsey JF, Gewertz BL. Acute mesenteric ischemia. Surg Clin N Am 1997;77:307-318.
- 3. Boley SJ, Brandt LJ, Sammartano RJ. History of mesenteric ischemia. The evolution of a diagnosis and management. Surg Clin N Am 1997;77:275–288.
- 4. Nissan A, Zamir O, Spira R, et al. A more liberal approach to the surgical treatment of Crohn's disease. Am J Surg 1997;174:339–341.
- 5. Heimann TM, Greenstein AJ, Lewis B, et al. Comparison of primary and reoperative surgery in patients with Crohn's disease. Ann Surg 1998;227:492–495.
- 6. Cullen JJ, Kelly KA, Moir CR, et al. Surgical management of Meckel's diverticulum. An epidemiologic, population-base study. Ann Surg 1994;220:564–568.
- 7. Gottlieb MM, Beart RW Jr. surgical management of Meckel's diverticulum. Ann Surg 1995;222:770.
- 8. Stringer M, Puntis JW. Short bowel syndrome. Arch Dis Child 1995;73:170-173.
- 9. Thompson JS, Langnas AN. Surgical approaches to improving intestinal function in the short-bowel syndrome. Arch Surg 1999;134:706–709; discussion 709–711.
- 10. Thompson JS. The role of prophylactic cholecystectomy in the short-bowel syndrome. Arch Surg 1996;131:556–560.
- 11. Byrne T, Persinger R, Young L, et al. A new treatment for patients with short-bowel syndrome, growth gormone, glutamine, and a modified diet. Ann Surg 1995;222:244–254.
- 12. Panis Y, Messing B, Rivet P, et al. Segmental reversal of the small bowel as an alternative to intestinal transplantation in patients with short bowel syndrome. Ann Surg 1997;225:401–407.
- 13. Tassiopoulos AK, Baum G, Halverson JD. Small bowel fistulas. Surg Clin N Am 1996;76:1175–1181.

14 Urinary Diversion Surgery

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CONTENTS

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INTRODUCTION

The use of intestine to substitute for the diseased or absent bladder represents an important surgical innovation in gastrointestinal surgery to emerge during the past century. Intestinal urinary diversions have evolved from simple conduits to true continent storage vessels that may, in some circumstances, be anastomosed to the urethra to function as a neobladder. All segments of the bowel have been utilized toward this end. It is the purpose of this chapter to outline the basic types of urinary diversions most commonly in use in North America and Europe today and to review the complications associated with them.

INDICATIONS FOR URINARY DIVERSION

In the large majority of cases, a urinary diversion is performed because the bladder has been removed. Muscle invasive transitional cell cancer of the bladder remains the most common indication for an intestinal urinary diversion, although it is also occasionally performed for some benign disease processes. In other circumstances, such as the neurogenic bladder or incompetent urinary sphincter which are not amenable to conservative treatment, the bladder may be left *in situ*, whereas the urine is diverted into an intestinal segment.

The choice of urinary diversion should be tailored toward the patient's needs and abilities. An incontinent diversion such as the ileal conduit is relatively easy to care for and the reoperation rate is low (1-4). On the other hand, some patients may prefer a

continent diversion or neobladder, using either a program of intermittent catheterization or Valsalva's maneuver to empty the reservoir (5,6). Senescence is not, by itself, a contraindication for a continent diversion (7). Some younger patients who lack the motivation or the manual dexterity required to care for a continent diversion, however, may be better served with a conduit. From an economic standpoint, there is little difference in the cost of construction for an incontinent vs continent reservoir; current Medicare reimbursements for an ileal conduit and continent diversion are \$2100 and \$1500, respectively.

CONTRAINDICATIONS TO URINARY DIVERSION

The patient with a history of inflammatory bowel disease poses a challenge to the surgeon in the selection of an appropriate bowel segment for diversion. Clearly, the use of the large bowel is to be avoided in the patient with ulcerative colitis. Likewise, the terminal ileum should not be used in patients with a history of Crohn's disease. This is particularly problematic because Crohn's disease may involve the entire gastrointestinal tract. In this situation, the use of jejunum or stomach is preferable to the terminal ileum in the construction of the diversion. Ileal conduits are relatively contraindicated in children because of the risk of associated long-term chronic pyelonephritis and renal deterioration. Contraindications for orthotopic neobladder include tumor in the prostatic urethra, or in females, tumor at or near the bladder neck.

TYPES OF URINARY DIVERSION

History

The earliest attempts at urinary diversion occurred in the mid-19th century. This idea was based on the observation that birds possessed a cloaca, through which both urine and feces were expelled. Improving surgical techniques culminated in the successful creation of the ureterosigmoidostomy in 1911, which was to become the most commonly employed form of urinary diversion for the next 40 yr (8). The operation had the advantages of being relatively easy to perform, and it allowed the patient to be continent via the anal sphincter. It became increasingly evident, however, that ureterosigmoidostomies had treacherous long-term complications. First, the anastomoses of the ureters to the intact fecal stream lead to unacceptably high rates of pyelonephritis and renal deterioration. Second, the exposure of urine to the entire length of the large bowel often created severe life-threatening metabolic acidosis. Finally, a high rate of adenocarcinomas was observed to occur near the uretero-intestinal anastomosis (8-11).

The problems associated with urterosigmoidostomy necessitated improvements in urinary diversions. Some surgeons came to prefer cutaneous ureterostomies, whereby the ureters were simply brought up to the skin as stomas. Intuitively, such an operation seemed sensible because it avoided many of the complications of ureterosigmoidostomy. In practice, however, cutaneous ureterostomies suffered from high rates of stomal stenosis. They are occasionally useful, however, as palliative procedures (9).

Ileal Conduit

The solution to the problems inherent to both ureterosigmoidostomies and cutaneous ureterostomies came in the early 1950s, when Bricker introduced the ileal conduit (12). The ileal conduit is incontinent and does not actually store urine, but its large "rosebud" stoma allows for proper ostomy appliance placement, and the end-to-side refluxing

ureteral anastomosis is technically easy to perform (Fig. 1). Moreover, urinary absorption with a conduit device was minimized, averting the severe acidosis observed with ureterosigmoidostomies. Like all externally draining ostomies, the choice of stomal location is important for the purposes of appliance application and leak prevention.

Bricker's operation remains the gold standard for urinary diversions today. Conduits created from large bowel have also been used for urinary diversion, but they possess no significant advantage over those fashioned from ileum. Candidates for an ileal conduit include adult patients requiring cystectomy. The ileal conduit may also be a better operation for patients in whom postoperative pelvic radiation is anticipated, because it is typically placed outside of the radiation field. Recent studies have demonstrated that patients with ileal conduits adapt well to the operation, and may not necessarily report an inferior quality of life compared to patients who receive a continent diversion (13,14).

Continent Urinary Diversions

Gilchrist described a technique for the creation of continent urinary diversions shortly after the introduction of the ileal conduit (15). In this type of diversion, the ascending colon was utilized as a continent reservoir, whereas the ileocecal valve served as the continence mechanism. The operation was not widely embraced, but its basic principles served as the model for subsequent continent reservoirs. Renewed interest in continent diversions emerged in the 1970s and 1980s, however, in response to patient demands and the realization that the ileal conduit was not an acceptable diversion in the pediatric population.

The most commonly performed continent catherizable urinary diversion utilized in the United States today is the so-called Indiana pouch (16,17). Like Gilchrist's operation, this urinary diversion utilizes the ascending colon, ileocecal valve, and terminal ileum to create a continent reservoir. It differs from this procedure, however, in that the colonic segment is detubularized, and the ileocecal valve plicated to improve continence (Fig. 2). The short segment of terminal ileum is brought to the skin surface as a flush, catherizable stoma. In some cases, an efferent stomal limb may be constructed of appendix (the "Mitrofanoff principle") (18). This is particularly useful in children, whereby the appendiceal limb can be brought out inconspicuously through the umbilicus.

The rationale for bowel detubularization and reconfiguration is that a detubularized segment of bowel maximizes the volume for a given surface area. Furthermore, detubularized reservoirs maintain relatively constant pressures with increasing radius due to distension, following Laplace's law:

Reservoir pressure = Wall tension/ radius

In spite of adherence to these principles, however, reconfigured bowel still remains peristaltic to some degree, occasionally resulting in high-pressure contractions and incontinence. These reservoirs may initially possess a small capacity, but their volumes will increase over time as they are allowed to store urine.

Orthotopic Neobladders

Detubularized bowel reservoirs can also be utilized as orthotopic neobladders. A commonly utilized neobladder was designed by Studer et al. (19). This operation creates a reservoir from a 60-cm segment of terminal ileum, which is then anastomosed to the urethra. Neobladders from combined ileocecal segments and sigmoid colon may also be created (20-23).

Α



Fig. 1. Construction of the ileal conduit. (Reprinted with permission: Shapiro E, Ileal conduit urinary diversion. In Marshall F, ed. Operative Urology. WB Saunders, Philadelphia, PA 1991.) (A) Segment of terminal ileum isolated with adequate mesenteric blood supply. (B) Ileal segment resected and ileal continuity reestablished. (C) Ureters anastomosis to ileum in end to side fashion. (D) Distal ileal end pulled through the abdominal wall. (E) Rosebud stoma created.



Fig. 2. Construction of an Indiana Pouch with catherizable stoma. (A) Segment of ascending colon and terminal ileum resected with preservation of blood supply. (B) Ureters anastomosed to cecal segment and terminal ileum acting as efferent limb for catheterization. (C) Cecal segment split and pulled over to form a pouch to minimize pouch pressure. (D) Efferent limb brought out for catheterization.

Neobladders may be performed in both sexes and can allow for relatively normal micturition. These patients are able to sense fullness of the reservoir, which they then empty by Valsalva's maneuver. Daytime continence rates approach 100% in some series, but enuresis may occur in some patients (19,22,24,25).

COMPLICATIONS OF URINARY DIVERSIONS AND THEIR MANAGEMENTS

Metabolic and Medical

Using the intestine as a substitute for the urinary bladder can lead to significant complications. The bowel epithelium is an absorptive surface, whereas the transitional epithelium is relatively impermeable to most substances. Any urinary diversion that utilizes bowel will absorb, to some extent, urinary solutes. With the exception of stomach, the more proximal the bowel segment, the greater the reabsorption characteristics. The degree of such absorption is proportional to the duration of exposure, so that continent reservoirs increase the risk of metabolic derangements. The risk of metabolic complications from these operations also increases with decreasing renal function. Certain drugs, such as methotrexate and phenytoin, may be excreted by the kidneys and

reabsorbed on the bowel surface to achieve potentially toxic serum levels. The patient with hepatic insufficiency is also more prone toward ammonia toxicity. Calculation of creatinine clearance is made difficult in patients with urinary diversions made of bowel, because the bowel epithelium passively reabsorbs creatinine. To minimize this effect, a diuresis must be established (10).

Acidemia is a common problem in patients with urinary diversions made of small or large bowel. The colon and ileum secrete sodium and bicarbonate ions, while reabsorbing ammonium and chloride. This potentially results in a hyperchloremic metabolic acidosis, which may be treated with appropriate alkalinizing agents, such as bicarbonate or citrate salts. Colonic diversions may also lose potassium, resulting in hypokalemia. The chronic exposure of jejunum to urine leads to large water and sodium chloride losses with increased potassium and proton reabsorbtion; the resulting hypochloremic, hyponatremic, hyperkalemic metabolic acidosis may be life threatening. Treatment for this problem includes the administration of sodium chloride for the condition. Thus, jejunum is to be avoided in the construction of urinary diversions whenever possible (10).

The terminal ileum is the primary site for vitamin B_{12} and bile salt absorption. If long segments of terminal ileum are used to construct a urinary diversion, the patient may become vitamin B_{12} -depleted. This process may take 5 yr, however, to deplete the body's stores of this nutrient (26). Similarly, bile salts are reabsorbed in the ileum, so that resection of this portion of the bowel may cause a bile-salt osmotic diarrhea. This condition may be ameliorated with oral loperamid or cholestyramine (10,27). Finally, the chronic metabolic acidosis associated with urinary diversions has been demonstrated to result in bone demineralization (28–30).

Occasionally, a segment of stomach can be used in urinary diversions, especially in the absence of other utilizable bowel segments or in patients with renal insufficiency. Instead of developing metabolic acidosis, however, the loss of chloride ions and protons from gastric secretion results in a hypochloremic metabolic alkalosis, requiring oral H2-agonists or omeprazole treatment (10).

Urinary Tract Infection

The incidence of pyelonephritis among patients with urinary diversions is 3-13%, although nearly 100% are colonized with bacteria (1,2,7). The diagnosis of obstruction must be ruled out with appropriate radiographic imaging studies when pyelonephritis is suspected, because adequate drainage of the obstructed system is required to clear the infection.

Surgical Complications of Urinary Diversions

Surgical complications from urinary diversions may occur both in the immediate perioperative period, as well as several months or even years later. Most patients who undergo bowel surgery will have a postoperative ileus. This is usually self-limiting, but prolonged, increased nasogastric-tube outputs or the inability to tolerate oral intake must alert the physician to the possibility of a bowel obstruction. Plain-film X-ray is usually adequate to make the diagnosis of bowel obstruction, but a CT scan is useful in determining the exact etiology and for planning further interventions.

The ureterointestinal anastomosis is a common site of postoperative difficulties. Urine leaks may develop after the operations. They usually manifest themselves as protracted

periods of drainage output. If the origin of the drainage fluid is in doubt, a sample may be sent for creatinine assay. The routine use of ureteral stents and catheter drainage until the suture lines have healed may avert this problem.

The ureteral-intestinal anastomosis may also stricture. The likelihood of this occurring depends on several factors, including the technical expertise of the surgeon, as well as the type of anastomosis performed. When a ureteral stricture is suspected, the differential diagnosis should include ischemic stricture, calculus, or tumor recurrence. Patients with anastomotic strictures may be relatively asymptomatic. Some may complain of vague flank pain, whereas others may present with fulminant pyelonephritis. If the ureteral anastomosis is of the refluxing variety, contrast injection under pressure into the pouch or conduit should promptly reflux into the renal collecting system. Failure to observe reflux suggests an anastomotic stricture. In reservoirs or conduits that do not employ refluxing anastomoses, an IVP may be used to make the diagnosis. Spiral noncontrast CT scan may be considered as an initial diagnostic imaging option in those patients with significant renal insufficiency. Renal ultrasound may also be useful in this setting, but this modality has limited abilities to image the entire ureter in adults.

The choice of treatments for anastomotic strictures depends on the etiology of the stricture and its length. Fibrotic benign strictures have acceptable cure rates with percutaneous balloon dilation or endoscopic incision (31-33). Longer strictures are less likely to have favorable long-term results with these methods, however, and may require open reimplantation.

Stricture of the urethral-reservoir anastomosis is a complication unique to orthotopic neobladders. Simple dilation or internal urethrotomy may be sufficient treatment in the majority of cases. Likewise, continent diversions such as the Indiana pouch may form strictures in the efferent limb from either ischemia or infrequent catheterization.

A life-threatening long-term complication of continent reservoirs is pouch rupture, which nearly always occurs because of over-distension in patients who are noncompliant with a catheterization schedule. The rupture of a continent reservoir must be considered in the differential diagnosis formulation for any patient who presents with signs and symptoms of an acute abdomen. The diagnosis is made by contrast instillation into the pouch, which will reveal intraperitoneal extravasation if rupture has occurred. Treatment almost always requires emergent open repair and abdominal irrigation.

Calculi

Patients with urinary diversions fashioned from bowel are at increased likelihood for calculus formation, both in the diversion itself, as well as in the upper tracts of the urinary system (10,34,35). The reasons for this increased risk are multifactorial and are not completely understood. Bacterial colonization with urease-producting organisms, lithogenic mucous production, increased calcium excretion, and dehydration all likely play a role in the etiology of stones in this population. The stones' compositions are unpredictable and heterogeneous, but usually consist of struvite, calcium oxalate, or calcium phosphate. Preventative measures for stone formation include the avoidance of nonabsorbable sutures or staples during reservoir construction, eradication of ureasplitting organisms when diagnosed, and irrigation of excess mucous.

The treatment of upper tract stones in patients with urinary diversions is challenging. Obtaining retrograde access to the upper tracts via the diversion is often difficult. These stones are sometimes amenable to extracorporeal shockwave lithotripsy therapy, but frequently require antegrade percutaneous renal access (*36*).

SUMMARY

- 1. Urinary diversions constructed from bowel are relatively common urological procedures.
- 2. Proper patient selection, improvements in surgical techniques, and recognition of potential long-term complications have minimized their risks.
- 3. Although the creation of the ideal bladder substitute perhaps awaits the availability of laboratory-grown tissues and organs, thousands of patients will continue to benefit from the available bowel-based technology in the foreseeable future.

REFERENCES

- 1. Bachor R, Hautmann R. Options in urinary diversion: a review and critical assessment. Semin Urol 1993;11:235–250.
- 2. Frazier H, Robertson J, Paulson D. Complications of radical cystectomy and urinary diversion: a retro spective review of 675 cases in 2 decades. J Urol 1992;148:1401–1405.
- 3. Sullivan J, Grabstald H, Whitmore W. Complications of ureteroileal conduit with radical cystectomy: review of 336 cases. J Urol 1980;124:797–801.
- 4. Hensle T, Dean G. Complications of urinary tract reconstruction. Urol Clin N Am 1991;18:755-764.
- 5. Boyd S, Feinberg S, Skinner D, et al. Quality of life survey of urinary diversion patients: comparison of ileal conduits versus continent Koch ileal reservoirs. J Urol 1987;138:1386–1389.
- 6. Bjerre B, Johansen C, Steven K. Health-related quality of life after cystectomy: bladder substitution compared with ileal conduit diversion. A questionnaire survey. Brit J Urol 1995;75:200–205.
- 7. Navon J, Wong A, Weinberg A, et al. A comparative study of postoperative complications associated with the modified Indiana pouch in elderly versus younger patients. J Urol 1995;154:1325–1328.
- 8. De Kernion J, Trapasso J. Urinary diversion and continent reservoir. In: Gillenwater J, Grayhack J, Howards S, Duckett J, eds. Adult and Pediatric Urology. Vol 2, 3rd ed, Mosby, St. Louis, 1996.
- 9. Bachor R , Hautmann R. Options in urinary diversion: a review and critical assessment. Sem Urol 1993;11:235–250.
- 10. McDougal W. Metabolic complications of urinary intestinal diversion. J Urol 1992;147:1199–1206.
- 11. Treiger B, Marshall F. Carcinogenesis and the use of intestinal segments in the urinary tract. Urol Clin N Am 1991;18:737–742.
- 12. Bricker E. Bladder after pelvic evisceration. Surg Clin N Am 1950;30:511-518.
- 13. Fujusawa M, Isotani S, Gotoh A, et al. Health-related quality of life with orthtopic neobladder versus ileal conduit according to the SF-36 survey. Urology 2000;55:862–865.
- 14. Katimura H, Miyao N, Yanase M, et al. Quality of life in patients having an ileal conduit, continent reservoir or orthotopic neobladder after cystectomy for bladder carcinoma. Int J Urol 1999;6:393–399.
- 15. Gilchrist R, Merricks J, Hamlin H, et al. Construction of a substitute bladder and urethra. Surg Gynecol Obstet 1950;90:752–760.
- 16. Rowland RG, Mitchell ME, Bihrle R, et al. Indiana continent urinary reservoir. J Urol 1987;137:1136–1139.
- 17. Rowland RG, Mitchell ME, Bihrle R. The caecoileal continent urinary reservoir. World J Urol 1985;3:185–190.
- 18. Mitrofanoff P. Cystostomie continente trans-appendiculaire dans le traitement des vessies neurologiques. Chir Ped 1980;21:297.
- 19. Studer U, Danuser H, Merz V, et al. Experience in 100 patients with an ileal low pressure bladder substitute combined with an afferent tubular isoperistaltic segment. J Urol 1995;154:49–56.
- 20. Bejany D, Politano V. Modified ileocolonic bladder: 5 years experience. J Urol 1993;149:1441–1444.
- Light J, Englemann U. Le bag: Total replacement of the bladder using an ileocolonic pouch. J Urol 1986;136:27–31.
- 22. Hautmann R, Egghart G, Frohnberg D, et al. The ileal neobladder. J Urol 1988;139:39-42.
- 23. Reddy P, Lange P, Fraley E. Total bladder replacement using detubularized sigmoid colon: technique and results. J Urol 1991;145:51–55.
- 24. Skinner D, Boyd S, Lieskovsky G, et al. Lower urinary tract reconstruction following cystectomy: experience and results in 126 patients using the Kock ileal reservoir with bilateral ureteroileal ure-throstomy. J Urol 1991;146:756–760.
- 25. Iwakiri J, Gill H, Anderson R, et al. Functional and urodynamic characteristics of an ileal neobladder. J Urol 1993;149:1072–1076.

- Steiner M, Morton R, Marshall F. Vitamin B12 deficiency in patients with ileocolic neobladders. J Urol 1993;149:255–257.
- 27. Roth S, Semjonow A, Waldner M, et al. Risk of bowel dysfunction with diarrhea after continent urinary diversion with ileal and ileocecal segments. J Urol 1995;154:1696–1699.
- Gianni S, Nobile N, Sartori L, et al. Bone density and skeletal metabolism in patients with orthotopic ileal neobladder. J Am Soc Nephrol 1997;8:1553–1559.
- 29. Davidson T, Lindergard B, Obrant K, et al. Long-term metabolic effects of urinary diversion on skeletal bone: histomorphometric and mineralogic analysis. Urology 1995;46:328–333.
- Kawakita M, Arai Y, Shigeno C, et al. Bone demineralization following intestinal diversion assessed by urinary pyridinium cross-links and dual energy X-ray absorptiometry. J Urol 1996;156:355-359.
- 31. Kabalin J. Acucise incision of ureteroenteric strictures after urinary diversion. J Endourol 1997;11:37-40.
- 32. Ravery V, de laTaille A, Hoffmann P, et al. Balloon catheter dilatation in the treatment of ureteral and ureteroenteric stricture. J Endourol 1998;12:335–340.
- Stuart Wolf S, Elashry O, Clayman R. Long-term results of endoureterotomy for benign ureteral and ureteroenteric strictures. J Urol 1997;158:759–764.
- Turk T, Koleski F, Albala D. Incidence of urolithiasis in cystectomy patients after intestinal conduit or continent urinary diversion. World J Urol 1999;305–307.
- Dretler S. The pathogenesis of urinary tract calculi occurring after ileal conduit diversion: I. Clinical study. II. Conduit study. III. Prevention. J Urol 1973;109:204–209.
- 36. Cass A, Lee J, Aliabadi H. Extracorporeal shock wave lithotripsy and endoscopic management of renal calculi with urinary diversions. J Urol 1992;148:1123–1125.

IV LARGE BOWEL SURGERY

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CONTENTS

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INTRODUCTION

Segmental colon resection is a relatively common general surgical operation. It is the standard of care for colon cancer and is indicated for a variety of benign conditions to be discussed later.

Patients requiring elective colonic resection will undergo a bowel prep that will be performed at home, commencing 1 or 2 d preoperatively. Patients may expect a 4–10 d hospital stay. The precise length of the hospital stay depends on a number of variables including, operative technique, overall medical condition, motivation, and presence or absence of complications.

When discussing colon resection with patients, two questions are commonly asked by patients or family members. One is: will the patient require a colostomy? Second, what effect will the operation have on the function of the bowel? The colostomy issue will be addressed in the Subheading, "Indications." Regarding the effect of colectomy on bowel function, consider the following. The colon does absorb water, thus, converting liquid stool in the right colon to solid stool by the time it reaches the descending and sigmoid colon. The distal colon and rectum serve as "reservoirs" that allow storage of waste until there is an acceptable time to defecate. Physiologic changes associated with colonic resection will be discussed in subsequent Subheadings.

Most colon resections performed today are segmental collectomies involving removal of 1-2 ft of colon. This is in contrast to a formal "hemicolectomy" where half of the colon

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Fig. 1. "Normal" configuration of colon. (**A**) Ascending colon. (**B**) Transverse colon. (**C**) Descending colon. (**D**) Sigmoid colon. (**E**) Rectum. Some patients have significant redundancy in colon length, with the length of the sigmoid colon having the greatest variation.

Common Indication for Colectomy		
Benign Conditions	Malignant Conditions	
 Diverticulitis GI hemorrhage (AVM or Diverticular) Ulcerative colitis Sigmoid volvulus Foreign body perforation Penetrating injury to colon Ischemic colitis 	 Adenocarcinoma All other malignancies (lymphoma, sarcoma, and so on are rare) 	

Table 1Common Indication for Colectomy

is removed. Anatomical differences between various resections will be described and diagrammed (Fig. 1).

INDICATIONS FOR PROCEDURE

Indications for colectomy include benign and malignant diseases (Table 1). The most common benign conditions are diverticulitis, lower gastrointestinal (GI) hemorrhage, ulcerative colitis, sigmoid volvulus, and penetrating trauma. The most common malignant condition is adenocarcinoma of the colon.

Colectomy is the standard of care for adenocarcinoma of the colon. Most segmental colectomies for carcinoma are performed with intent to cure the patient. Even with advanced (metastatic) disease, colectomy may be required to palliate bleeding or obstruction.

When colectomy is being considered for benign conditions many variables are considered prior to recommending surgery. For example, in cases of sigmoid diverticulitis or sigmoid volvulus, recurrence rates are important. After one episode of sigmoid diverticulitis, the risk of a second episode is about 15–20%. However, if a patient has a second episode, the risk of subsequent attacks of diverticulitis rises to about 50%. Therefore, most surgeons recommend elective segmental colectomy after resolution of a second episode.

Conversely, with sigmoid volvulus recurrence rate after a first episode is 40–80%. Therefore, if a patients is fit enough to tolerate surgery, sigmoid resection is recommended after one episode of sigmoid volvulus. The general medical condition of the patient is important in all of these decisions.

In patients over 50 yr of age, significant lower GI hemorrhage is frequently a result of a bleeding colonic diverticula or a vascular ectasia (1). Bleeding from the right colon (ascending colon and cecum) is more common than bleeding from the left colon (descending and sigmoid colon). The bleeding stops spontaneously in more than 70% of patients. Efforts are made to identify the precise area of hemorrhage. In patients where hemorrhage has stopped, colonoscopy may identify the pathology. In patients with ongoing bleeding, a tagged RBC nuclear scan +/or angiography are utilized to localize the bleeding. Localization is important in order to limit the extent of colonic resection, should the patient come to operation. Patients with active bleeding are considered for surgery. Patients with lower GI bleeding who are in prohibitive medical condition for surgery may be considered for embolization therapy via a selective mesenteric angiogram.

Ulcerative colitis is a surgically curable disease via total colectomy. The patient may then have a permanent ileostomy (see chapter on small intestine) or an ileo-anal anastomosis. Unlike ulcerative colitis, granulomatous colitis may be appropriately treated by segmental colectomy (2).

Urgent colonic surgery may not allow for bowel preparation. Examples would include massive lower GI hemorrhage or colonic obstruction. In the former case, some "bowel preparation" has been accomplished by the cathartic effect of blood in the GI tract. With colonic obstruction however, the surgeon faces a colon full of solid stool with the highest bacterial counts. With urgent colon surgery on unprepped colon, a surgeon may need to perform a colostomy in order to prevent infectious complications, particularly leakage of an anastomosis. The patient should be forewarned of this distinct probability.

CONTRAINDICATIONS

The main contraindication to colon surgery would be medical instability. In other words, the surgeon must ask, can this patient tolerate a general anesthetic and the physiologic stress of major surgery? Examples of medical conditions, which would deter the surgeon are recent myocardial infarction, recent stroke, severe COPD, poorly controlled hypertension, and poorly controlled diabetes mellitus. All of these contraindications must be balanced against the indication for colon resection.

Purely elective colon surgery for a benign condition could be postponed for long periods while medical issues are addressed. Conversely, a patient with a life-threatening colonic condition, e.g., perforated diverticulitis with generalized peritonitis may require urgent surgery despite the patient's fragile condition.
It is not uncommon to encounter patients taking anticoagulant medications for a variety of conditions. The most common example would be patients taking coumadin for atrial fibrillation or for deep venous thrombosis or pulmonary embolism. In such cases, the physicians who care for the patient must determine a perioperative plan for the patient's anticoagulation. If the indication for the anticoagulation is questionable, the anticoagulant may be stopped indefinitely. If however, anticoagulation is a necessity, as in protection of a prosthetic heart valve; the following procedure is frequently followed.

The patient is instructed to stop taking coumadin 1 or 2 d prior to admission. The patient is admitted the day prior to surgery and is given intravenous-iv-heparin. The purpose of this regimen is to convert from anticoagulation, which is slow to reverse (coumadin) to anticoagulation, which is rapidly reversible (heparin).

The intravenous heparin is then stopped about 2 h prior to surgery. The heparin is restarted 4–8 h postoperatively depending on the magnitude of the operation. Finally, the patient resumes his/her coumadin prior to discharge.

PROCEDURE

Elective colon surgery requires bowel preparation. The goal of bowel preparation is to diminish the bacterial load logarithmically. The mechanical portion of the prep is accomplished by oral laxatives, which have replaced old-fashioned enema preps. The oral prep may be performed with a high volume solution of polyethylene glycol plus electrolytes, with Fleets phospho-soda solution, or with magnesium citrate. Additional antimicrobial preparation is achieved via the oral intake of poorly absorbed antibiotics such as neomycin and erythromycin base. Preoperative bowel preparation has lowered infectious complications of colon surgery from double-digit rates to singledigit rates.

As with many operations, a picture or a diagram may be worth a thousand words for understanding the operation. The following description will allow better understanding of the diagrams. Most colon surgery is performed via a vertical midline incision. The colon receives its blood supply from arteries, which originate from the anterior surface of the aorta. The arteries are the superior mesenteric and the inferior mesenteric arteries. The SMA branches supply 80–90% of the colon with arterial blood and the IMA 10–20%. The anastomosis between SMA and IMA branches occurs along the left side of the colon. The ascending and descending colon are fixed by peritoneal attachments. The transverse colon and sigmoid colon are mobile. The blood supply and lymphatics to the colon are contained in a sheet of fibrofatty tissue known as the mesocolon.

Any colon resection involves mobilization of the colon. To mobilize the ascending or the descending colon involves dividing peritoneal attachments laterally and lifting the colon into a midline position, with the colon still attached to the aorta by the mesocolon (3).

The resection margins are selected. At this point, the surgeon may choose to divide the colon at the proximal and distal resection margin or to divide the mesocolon first. The division of the mesocolon involves clamping and tying off branches of the mesenteric arteries and veins. The colon may be divided at resection margins by use of a linear stapler or using a scalpel between bowel clamps. Once these two steps are accomplished, intestinal continuity is reestablished by using suturing or stapling tech-



Fig. 2. (A) Schematic diagram of an end colostomy. (B) A mucus fistula.

niques. Regardless of technique chosen, there are three technical requirements for a successful colonic anastomosis:

- 1. An adequate lumen.
- 2. An adequate blood supply.
- 3. Lack of tension on the anastomosis.

Attention to these technical requirements during the operation may prevent postoperative complications such as leakage and stricture formation.

If the colonic resection is an emergency procedure and the colon is not "prepped" as aformentioned, or there exist extraordinary intraoperative problems, a colostomy may need to be performed. A colostomy involves bringing the colon to the anterior abdominal wall. A hole is created in the anterior abdominal wall. There are two common ways to create the colostomy. One way is to divide the colon. With this technique, the proximal end is brought through the hole in the abdominal wall as an "end colostomy" (Fig. 2A). In these cases, the distal colon is either closed and dropped back in the abdomen "the Hartman procedure" (Fig. 3A and B), or, it also is brought through the abdominal wall as a mucous fistula (Fig. 2B).

The second type of colostomy is a "loop colostomy." With a loop colostomy, the colon is not divided. Instead, a loop of colon is brought through a hole in the anterior abdominal



Fig. 3. (A) The Hartman operation. Resection of tumor containing bowel. (B) Creation of a colostomy, and oversewn blind rectal stump (Adapted from Shackelford's Surgery of the Alimentary Tract, Vol IV, 5th ed. WB Saunders, Philadelphia, PA, 2002).



Fig. 4. Schematic diagram of a loop colostomy.



Fig. 5. Anatomy after right hemicolectomy. The terminal ileum is then anastomosed to the transverse colon.



Fig. 6. Anatomy after sigmoid colectomy. The descending colon is then anastomosed to the rectum.

wall (Fig. 4). The anterior surface of the loop is opened allowing egress of stool. With either type of colostomy, an appliance is placed over the ostomy for collection of stool. As with colectomy with primary anastomosis, colectomy with colostomy may be associated with complications (4). The normal configuration of the colon is seen below in Fig. 1. The anatomy after right hemicolectomy and sigmoid collectomy are seen in Fig. 5 and 6, respectively. The anatomy after left hemicolectomy is seen in Fig. 7.

COMPLICATIONS

Complications of colonic surgery may be considered in two groups, generic complications and complications specific to intestinal surgery. Generic complications include atelectasis, pneumonia, deep venous thrombosis, urinary retention, wound infection, fascial dehiscence, and myocardial infarction. The incidence of any of these complications varies according to risk factors such as age, cigarette smoking, obesity, and presence of comorbid conditions such as diabetes mellitus. Some complications are preventable with proper perioperative care. Examples include proper bowel preparation and prophylactic antibiotic use to reduce the risk of wound infection. Another example is the use of mechanical compression stockings and/or mini-dose heparin to prevent deep venous thrombosis. A detailed discussion of generic complications is beyond the scope of this chapter.

Complications specific to bowel surgery include anastomotic leak and anastomotic stricture. Anastomotic leak can be caused by a variety of factors, most commonly errors in technique (5). Leaks may also be caused by an inadequate blood supply or by undue tension on the anastomosis. Anastomotic leaks are generally serious complications and usually present with signs of sepsis. These signs include oliguria, tachycardia, fever, and leukocytosis. The patient may also develop abdominal or pelvic pain beyond expected postoperative pain.

If an anastomotic leak is suspected, it may be confirmed with a gastrografin enema or with a CT scan with rectal contrast. Minor leaks that have sealed may be treated with percutaneous drainage, a nasogastric tube, and iv antibiotics. However, most commonly the patient will require diversion of the fecal stream with a proximal colostomy or ileostomy plus drainage of any abscess cavity (6). The use of an ostomy under these circumstances is usually temporary. The patient may then have restoration of the GI tract with colostomy takedown 6 wk–3 mo postoperatively. Anastomotic leaks occur in approx 5% of colonic resections. The leak rate is higher with rectal (low-pelvic) anastomoses.

Anastomotic stricture is a late complication, presenting 6 mo to years postoperatively. These strictures are usually caused by low-grade ischemia at the anastomosis or a subclinical leak. In the latter case, the inflammatory response results in fibrosis over time strictures present with constipation, cramping discomfort, bloating, or narrow caliber stools. Strictures occur in less than 10% of colonic resections. They may be treated with endoscopic dilatation in some cases. Significant strictures often require surgical revision of the anastomosis.

The complication rates for colectomy are very well established and are reflected in Table 2.

All other complications such as myocardial infarction, deep venous thrombosis and pulmonary embolism occur in only 1-3% of cases. The 30-d postoperative mortality for colonic resection is 1-6% depending on the series reviewed.

Table 2
Common Complications
After Colon Resection

Generic Complications in a Large Series of Colonic Resections

Prolonged ileus	7.5%
Pneumonia	6.2%
Respiratory failure	5.7%
Urinary tract infection	5.0%

From Ref. 5.

Table 3	
Bowel Function After Coletomy	y

Extent of Resection	Early (1–3 mo) Bowel Changes	Long-Term Bowel Changes
Segmental Colectomy	2-4 BMs/d may be "loose"	No discernible change
Hemicolectomy (1/2 of colon)	2–4 BMs/d may be liquid.	1–3 BMs/d
Subtotal Colectomy (only rectum left)	Diarrhea in form & frequency.	2–4 BMs/d
Total Colectomy with ileo-anal anastomosis	Diarrhea with potential incontinence.	4–8 BMs/d

There are two complications specific to colostomy, parastomal hernia and colostomy prolapse. Parastomal hernias occur in about 10% of cases. Colostomy prolapse is slightly less common. Both of these complications may require reoperation for correction of the problem (4).

Changes in Physiology and Potential Side Effects Caused by the Procedure

Patients vary greatly in what they consider normal bowel function. Some patients regularly have one bowel movement per day. Others go 3–4 d without a movement. As expected then, the physiologic outcome from patient to patient after colectomy is variable (7). Table 3 provides a rough outline of expectations based on experience with hundreds of patients. Patients will undergo physiologic accommodation to the resection for weeks to months. Patients with long-standing diarrhea may get relief with the use of bulking agents, adjustments in diet, or antidiarrheal medications.

ALTERNATIVE PROCEDURES

Colonic resections have been performed laparoscopically since the early 1990s. The technical limitations of the procedure have largely been overcome. The procedure is performed with four or five trocars placed through the abdominal wall. Some resections can be preformed completely laparoscopically. Others can be performed "hand-assisted."



Fig. 7. Schematic diagram of anatomy after a sigmoid colectomy. The left or descending colon is then anastomosed to the rectum.

With the hand-assisted approach, a 5–8-cm incision is made that allows the surgeon to introduce one hand into peritoneal cavity. The procedure is still viewed entirely via the intraperitoneal camera.

Surgical authorities accept laparoscopic colectomy for benign indications. There have been two major concerns voiced regarding laparoscopic colectomy for cancer. First are the many reports of trocar site recurrences of cancer in the abdominal wall. As larger volumes of data have been examined, this concern has waned. The second concern is focused on the question of lymph node dissection. Specifically, can a surgeon safely and routinely resect as much mesocolon (containing lymph nodes) using laparoscopic techniques compared to standard open resection. There is at least one study showing equivalence of lymph nodal resection between open and laparoscopic colectomy.

Ongoing prospective trials will answer the most important question. That is, are survival rates ultimately different using laparoscopic compared to open techniques? Preliminary reports suggest equal survival rates. It is the author's opinion that laparoscopic colectomy will supplant the standard operation by 2005.

COSTS

The upfront costs for laparoscopic colectomy are greater than for the standard open operations. This is because of the many disposable instruments used in laparoscopic surgery. Disposable instruments may range in cost from a \$50 trocar to a \$500 intestinal stapler. Total disposable costs could reach \$2000–\$3000 for a major procedure. This increase in upfront costs may be overcome by a diminished hospital length of stay in patients undergoing laparoscopic surgery. If an 8-d stay is converted to a 4-d stay, the

entire disposable instrument cost could be overcome. Analysis of cost to society would have to include consideration of time out of work. In theory, minimally invasive procedures will allow for an earlier return to work.

SUMMARY

- 1. The majority of colonic resections are performed for adenocarcinoma of the colon.
- 2. Colon resections are major operations, which require general anesthesia. Patients require bowel preparation preoperatively. The hospital stay ranges from 4–10 d based on a number of variables.
- 3. Because the colon is involved primarily in water absorption and waste storage, patients adapt well to resections of portions of the colon.
- 4. As a trend, laparoscopic colectomy will probably replace open colonic surgery during the current decade.

REFERENCES

- 1. Zuckerman GR, Prakash C. Acute lower intestinal bleeding. Gastrointest Endosc 1999;49:228-238.
- 2. Allan A, Andrews H, Hilton CJ, et al. Segmental colonic resection is an appropriate operation for short skip lesions due to Crohn's disease in the colon. World J Surg 1989;13:611–614.
- 3. Zollinger RM. Atlas of Surgical Operations. McGraw-Hill, New York, NY, 1993.
- 4. Allen-Mersh TG, Thompson JP. Surgical treatment of colostomy complications. Br J Surg 1988;75: 416–418.
- 5. LongoWE, Virgo KS, Johnson FE, et al. Risk factors for morbidity and mortality after colectomy for colon cancer. Dis Colon Rectum 2000;43:83–91.
- 6. Mileski WJ, Joehl RJ, Rege RV, et al. Treatment of anastomotic leakage following low anterior colon resection. Arch Surg 1988;123:968–971.
- 7. Desai TK, Kinzie JL, Silverman AL, et al. (1988) Life after colectomy. Gastro Clin North Am 1988;17: 905–915.

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CONTENTS

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INTRODUCTION

Surgery of the rectum and anus has evolved significantly and is quite commonly performed. The following procedures are representative of the more commonly performed operations in this area.

LOW ANTERIOR RESECTION

Low anterior resection (LAR) is generally performed for carcinoma of the mid-and proximal rectum. With the training of more surgeons specializing in operations on the colon and rectum, the extent of resection has been extended to some lesions in the lower third of the rectum, less than 6 cm from the anal verge. The operation does not require a special center *per se*, but Rosen (1) has shown that better outcomes are obtained when it is performed by a surgeon trained in colon and rectal surgery.

Indications

Apart from rectal carcinoma, complicated diverticular disease or high rectovaginal fistula secondary to radiation may occasionally require LAR.

From: *Clinical Gastroenterology: An Internist's Illustrated Guide to Gastrointestinal Surgery* Edited by: George Y. Wu, Khalid Aziz, and Giles F. Whalen © Humana Press Inc., Totowa, NJ

Contraindications

Contraindications to performance of LAR include poor general medical condition, which makes the patient an unacceptable risk for major abdominal surgery and unresectability of the primary disease.

Procedure

Prior to surgery, bowel preparation is required. Appropriate preoperative mechanical cleansing of the colon and antibiotic administration has been shown to significantly reduce the incidence of postoperative infectious complications (2).

Mechanical preparation can be achieved with laxatives, enemas, or lavage solutions. Oral antibiotics, given on the day preoperatively, usually consist of three doses of neomycin (1 g) and erythromycin base (500 mg) given at 1 PM, 2 PM, and 11 PM (3). Many surgeons have substituted metronidazole 500 mg for erythromycin base because of the unpleasant GI side effects of the latter. Most surgeons also elect to administer a single iv dose of a long-lasting cephalosporin immediately pre-operatively.

The patient undergoes general anesthesia and is then placed in the low lithotomy position utilizing Lloyd-Davies or Allyn stirrups. The abdomen and perineum is prepared with an antiseptic solution and the abdomen is entered through a lower midline incision. Full exploration is carried out to determine both resectability and the presence or absence of metastases. Once resectability has been determined, the sigmoid colon is mobilized by dividing the lateral peritoneal reflection. This incision is carried down into the pelvis to the level of cul de sac. Immediately, a similar incision is created. The ureters are identified at the level of the pelvic brim and protected. The superior hemorrhoidal artery, as a continuation of the inferior mesenteric artery, is ligated at its origin with concomitant ligation of the inferior mesenteric vein. The colon is divided at the level defined by vascular ligation, as is its mesentery. The technique of total mesorectal excision as defined by Heald (4) is then utilized to complete the pelvic dissection. The space between the mesorectum and the posterior and lateral pelvic parietal peritoneum is entered and sharp dissection is used to carry this dissection to the level of the pelvic floor. The rectum is divided at this level, using a linear stapler, and the specimen is removed. The anastomosis is carried out with a circular stapler, which places a double or triple row of staples circumferentially and then cuts out the center tissue. Fecal diversion with a proximal ostomy is rarely required (Fig. 1).

A word regarding total mesorectal excision is warranted. This technical advance has been shown to lower local recurrence rates to less than 10%, a marked improvement over historical rates of greater than 30%. Though technically demanding, it should be used in all cases of rectal cancer operated on for cure.

Complications and Management

The rate of complications following LAR has been reported as high as 41% (5). Most of these are common to most major abdominal procedures and would include atelectasis, urinary tract infection, wound infection, and deep venous thrombosis. Significant complications specific to LAR include anastomotic leakage, anastomotic stricture, and imperfections of continence or bowel habit. Leakage from the anastomosis after LAR



Fig. 1. Use of a circular stapler to create an anastomosis. (A) Resection of bowel containing a low lying tumor. (B) Positioning of the device in the rectal remnant and apposition of the bowel. (C) Completed anastomosis by a stapler that places a double or triple row of staples circumferentially, and then cuts out the center tissue.

is more common than from other colonic anastomoses because of the deep pelvic dissection and the inherent technical difficulty of the anastomosis. Rates of leakage have traditionally been reported in the range of 10% (6), though more recently rates less than 3% are seen (7). Many factors have been implicated in increased leakage rates. Chief among them being anemia, diabetes, local atherosclerotic disease, and prior pelvic irradiation. Anastomotic leakage leads to pelvic abscess and possibly sepsis, and requires drainage (in either an opened or closed CT-guided fashion) and usually temporary proximal diversion with either a colostomy or ileostomy. Anastomotic stricture may be the result of an anastomotic leak or may occur de novo with fibrosis at the anastomotic site. It is a late complication and can usually be managed with dilatation. It may require a local procedure or, less commonly, reresection. Imperfections of continence and irregularities of bowel habit are not uncommon and are generally related to loss of the fecal reservoir with rectal resection. The majority of these problems resolve within six mo without intervention.

Alternative Procedure

The alternative to LAR is complete abdomino-perineal resection with permanent colostomy, to be discussed later. While giving equivalent oncological results, LAR enables sphincter sparing in nearly all cases of midrectal cancer and now in some cases of distal rectal cancer.

Cost

The cost of this procedure, predicated on a 6-d hospitalization and including surgeon's fee, is approx \$11,300.

Summary

- 1. LAR can be performed safely by a surgical team performing the operation frequently.
- 2. It results in sphincter preservation, with an improved quality of life for the patient and, utilizing the technique of total mesorectal excision, affords excellent oncologic results.

ABDOMINO-PERINEAL RESECTION

This most radical operation for carcinoma of the rectum was first described by Ernest Miles in 1908 and is performed in much the same fashion today. Several modifications have lowered complication rates and improved cure rates. It should, again, be performed by a surgical team that undertakes the procedure relatively frequently and in an institution offering access to an enterostomal therapy nurse.

Indications

Abdomino-perineal resection is indicated for resectable carcinoma of the distal rectum, i.e., located less than 7 cm from the anal verge.

Contraindications

Contraindications to abdomino-perineal resection include unresectable metastatic disease and conditions making colostomy care difficult or impossible (blindness, severe arthritis), unless no other options are available.

Procedure

The patient undergoes preoperative bowel preparation as aforementioned. The procedure is performed as a two-team operation, with the patient positioned in lithotomy position. As described for LAR, the abdominal operator makes a lower midline incision. Exploration is carried out to ensure both resectability and the absence of metastatic disease, and then mobilization to the level of the pelvic floor is carried out exactly as described for LAR. The rectum is, however, not divided with a stapler distally. The perineal operator, having previously placed a purse-string suture around the anus, creates a wide perineal incision elliptically around the anus. The incision is deepened into the ischio-rectal fossae bilaterally, dividing the inferior hemorrhoidal vessels, and to the coccyx posteriorly. The presacral space is entered just anterior to the coccyx, usually with the tips of the dissecting scissors. The levator muscles are then divided sharply from posterior to anterior. The anterior dissection is carried out last, dividing the transverse perinei muscles and carefully separating the rectum from the posterior vaginal wall in women and from the prostate and seminal vesicles in men. The specimen is then delivered in its entirety to the perineal operator and removed from the operative field. The perineal wound is then closed and a drain is inserted. The abdominal operator, meanwhile, has created a sigmoid colostomy in the left lower quadrant and effected abdominal wound closure.

Complications and Management

Complications following abdomino-perineal resection have been reported as high as 61% (8). Most of these can generally be prevented by appropriate pre-operative evaluation and careful operative technique. Postoperative sexual dysfunction can occur in both men and women, including a significant percentage of men with impotence. These complications are more common with advancing age and are somewhat unavoidable. Management, when indicated, can consist of counseling, medication, and implantation of prosthetic devices, or reconstructive surgery. The problems of colostomy management are discussed in detail in Chapter 15.

Alternative Procedure

Alternatives to abdomino-perineal resection for rectal cancer include local procedures, such as transanal excision or electrocoagulation, or brachytherapy with high-dose local radiation therapy. Though effective for early stage rectal stage rectal cancer, none of the local procedures can be performed reliably for cure in carcinoma.

Cost

This operation generally requires a 6-d hospital stay and the cost, again including both surgeon and hospital payments, is \$12,900.

Summary

1. Abdomino-perineal resection can be performed safely by a surgical team well versed in its technique, with excellent oncologic results and acceptable complication rates.

2. With greater application of sphincter-saving procedures, such as LAR, the numbers of patients undergoing this operation will continue to decrease.

TOTAL PROCTOCOLECTOMY WITH END-ILEOSTOMY

Total proctocolectomy with end-ileostomy (TPC) refers to the removal of the entire colon and rectum with permanent ileostomy. Though it does not require a specialized center, it does require a surgical team skilled particularly in rectal resection. The availability of an enterostomal therapy specialty nurse for both pre- and postoperative teaching and stoma consultation is desirable.

Indications

TPC has traditionally been the operation of choice for patients with ulcerative colitis requiring elective operation, though in recent years it has been supplanted by restorative proctocolectomy with ileoanal pouch, to be discussed in the next section. Because of its proven reliability in patients with ulcerative colitis, it is still the standard against which the results of newer operations are judged. TPC is also performed in patients with Crohn's colitis also having rectal involvement.

Contraindications

TPC should not be performed in the emergent or urgent case. In patients who require surgery under such conditions, total abdominal colectomy with ileostomy should be performed, but proctectomy should be deferred to a later date. A relative contraindication is the presence in the patient of severe arthritis involving the hands, blindness, or another disability, which would make stoma care by the patient impossible.

Procedure

Prior to surgery, the patient undergoes a full mechanical bowel preparation, again as aforementioned. Parenteral steroids are administered when indicated. After undergoing general anesthesia, the patient is placed in the lithotomy position. A midline incision, extending from the symphysis pubis to the supraumbilical region is generally utilized. Exploration of the abdomen is carried out with particular attention to the small intestine, looking for any signs of Crohn's disease involving that organ. The ileum is first divided close to the ileocecal valve, preserving as much small bowel length as possible. The right colon, transverse colon, and left colon are then mobilized from their lateral peritoneal and omental attachments. Care is taken to avoid injury to the duodenum, when mobilizing the hepatic flexure, and the spleen when mobilizing the splenic flexure. The sigmoid colon is then carefully dissected free from the left iliac fossa, with care taken to identify and protect the left ureter. The mesentery to all of the above segments of colon is then divided along with the vascular supply, including the ileocolic, right colic, middle colic, left colic, and sigmoid vessels. As dissection proceeds into the pelvis, a second surgical team begins with a perineal dissection. The abdominal surgeon carefully enters the pelvis, sharply dissecting the rectum and its mesentery from their posterior and lateral attachments. The sympathetic and parasympathetic nerves are preserved to the extent possible in the lateral and posterior dissections. Anteriorly, in men, the seminal vesicles are identified and retracted. Dissection close to the rectum is

carried out to the level of the levator muscles. The technique of the proctectomy differs from that performed with cancer in that it is carried out in the intersphincteric plane, very close to the anal canal and rectum. This technique has been shown to significantly lower the incidence of nonhealing of the perineal wound. Once the dissection of the perineal and abdominal operators meet, the entire colon and rectum are removed from the operating field. While the perineal operator is closing the perineal wound, the abdominal operator creates an end ileostomy as described in the previous chapter and closes the abdominal wound.

Complications and Management

Aside from the complications inherent to abdominal procedures in general, several complications are relatively specific to this operation including sexual dysfunction, nonhealing of the perineal wound, and complications related to the ileostomy stoma itself. Sexual dysfunction (erectile dysfunction or retrograde ejaculation in men and dyspareunia in women) has been reported in up to 11% of men undergoing proctectomy for inflammatory bowel disease (9) and up to 50% of women (10). Even with the use of intersphincteric proctectomy, nonhealing of the perineal wound remains a significant problem, occurring in 11% of patients operated on for ulcerative colitis and 33% of those operated on for Crohn's disease (11). Complications related to the ileostomy are reviewed earlier.

Alternative Procedures

The alternative to TPC is restorative proctocolectomy with an ileoanal pouch, to be discussed in the next section. This operation has the advantage of avoiding a permanent ileostomy but generally requires at least two stages and has an increased rate of complications.

Cost

Payments to the hospital and surgeon for this operation generally total approx \$12,700. The cost of stoma appliances on a permanent basis is difficult to estimate and is not always reimbursed by insurance companies.

Summary

- 1. Total proctocolectomy with end ileostomy can be safely performed for patients with ulcerative colitis and Crohn's disease with rectal involvement.
- 2. The use of the intersphincteric technique for proctectomy is important and the availability of an enterostomal therapy nurse is advisable.

TOTAL PROCTOCOLECTOMY WITH ILEO-ANAL ANASTOMOSIS

Total proctocolectomy with ileal pouch-anal anastomosis (TPC-IPAA) was initially popularized in the late 1970s as a sphincter-saving alternative to total proctocolectomy with ileostomy in the operative treatment of ulcerative colitis and familial adenomatous polyposis (12,13). Since that time, this operation, with its avoidance of a permanent ileostomy, has become the preferred procedure in the elective treatment of both of the above diseases. It is a technically demanding procedure and should only be performed

by surgeons well trained in its technique and in centers performing a substantial number of such procedures.

Indications

TPC-IPAA is indicated in the elective surgical treatment of patients with ulcerative colitis and familial adenomatous polyposis. If a patient requires urgent operation, sub-total colectomy should be performed with later elective restorative surgery.

Contraindications

Contraindications include the need for emergency surgery, Crohn's disease, the presence of invasive cancer, anal incontinence, morbid obesity, psychological instability, and advanced age. The operation is usually performed in two stages. At the first operation, a total colectomy is performed as described in the previous section, with rectal resection being carried down to approx 2–3 cm from the anal verge. In the past, the rectal mucosa was stripped from the remaining rectal stump, but, in general, this is no longer done. The rectum is divided with a stapler at that level and a reservoir/pouch is then constructed from the distal 30 cm of terminal ileum. The most popular configuration of the pouch is a "J" shape, but "S"-shaped and "W"-shaped pouches have also been used. Following formation of the pouch, a circular stapled anastomosis is created between the apex of the pouch and a short rectal cuff. A proximal diverting ileostomy is then performed. The ileostomy is subsequently closed, as a second stage operation, in 8–12 wk after radiological confirmation of pouch integrity and anastomotic healing is obtained (Fig. 2).

Complications and Management

There are numerous complications to TPC-IPAA. In addition to those reported with most major intestinal resections, a number of complications are specific to this procedure. These include small bowel obstruction, particularly related to the temporary diverting ileostomy, pelvic sepsis, pouch-vaginal or pouch-anal fistulas, incontinence, pouch-anal stricture, and pouchitis (14). The most common long-term side effect is an increased stool frequency, occasionally associated with dehydration. An increased stool frequency is also seen with episodes of pouchitis, a poorly understood nonspecific inflammation of the pouch. Whereas stool frequency can generally be controlled well by the use of bulk-forming agents, diet, and the judicious use of antimotility agents, the presence of pouchitis usually requires a course of antibiotics, most commonly metronidazole. Uncommon complications include urinary or sexual dysfunction and, in patients operated on for familial adenomatous polyposis, the formation of intraabdominal desmoid tumors.

Alternative Procedure

As previously noted, the alternative procedure to TPC-IPAA for both ulcerative colitis and familial adenomatous polyposis is total proctocolectomy with permanent ileostomy. The major advantage of TPC-IPAA is its avoidance of the permanent ileostomy. The disadvantages are the need for a second-stage operation (ileostomy closure) and the higher complication rate.



Fig. 2. Ileo J-pouch and anal anastomosis. (A) The rectum is divided and a reservoir/pouch is then constructed from the distal 30 cm of terminal ileum. (B) Following formation of the pouch, a circular stapled anastomosis is created between the apex of the pouch, and a short rectal cuff. The ileostomy is subsequently closed, as a second stage operation, in 8-12 wk and anastomotic healing is obtained.

Cost

The cost for the first stage of this operation is approx \$13,200. In most cases, a second stage procedure of ileostomy closure will be required and this additional procedure and hospitalization will add \$7600, making the total cost \$20,800.

Summary

- 1. TPA-IPAA is currently the preferred procedure for definitive surgical treatment of ulcerative colitis and familial adenomatous polyposis.
- 2. Though technically more demanding than the traditional TPC-permanent ileostomy, it avoids the necessity for a permanent ileostomy and is, therefore, more widely accepted by patients. Success rates range from 94–97%.

SURGERY FOR RECTAL PROLAPSE

Rectal prolapse is an uncommon condition defined as complete protrusion of the entire thickness of the rectal wall through the anus. It is seen far more commonly in women than in men and generally after the age of 40 (15). Pathologic defects noted are a diastasis of the levator ani muscles, an abnormally deep cul de sac, an elongated sigmoid colon, and loss of the rectal fixation to the sacrum. Prolapse can secondarily result in incontinence caused by a patulous anus. Numerous procedures have been described for correction of rectal prolapse, including both abdominal and perineal approaches. Neither approach requires specialized facilities and the choice of approach is generally determined by patient risk factors. One of the most common abdominal operations employed is the Ripstein procedure. It is indicated for the repair of complete rectal prolapse in a patient considered being an acceptable risk for abdominal surgery. Contraindications include an excessively redundant sigmoid colon in a patient with high risk for postoperative mortality and morbidity from an abdominal procedure.



Fig. 3. Ripstein procedure for rectal prolapse. A 5-cm band of synthetic plastic mesh is then sutured to the sacrum with nonabsorbable sutures. The rectum is then pulled out of the pelvis, and the mesh is sutured to the rectum in a fully encircling.

Procedure

The technique, as described by Ripstein, involves a formal laparotomy through a lower abdominal incision, the rectum is mobilized from the hollow of the sacrum and from the pelvic sidewall to the level of the coccyx. A 5-cm band of synthetic plastic mesh is then sutured to the sacrum with nonabsorbable sutures. The rectum is then pulled taut out of the pelvis and the mesh is sutured to the rectum in a fully encircling fashion following which the pelvic peritoneum is closed (Fig. 3). Some modifications of the procedure use a mesh wrap that is not completely circumferential to avoid possible rectal narrowing.

Complications and Management

A review of 1111 procedures performed by members of the American Society of Colon and Rectal Surgeons found a prolapse recurrence rate of 2.3% and a complication rate of 16.5% (*16*). The most common complication, fecal impaction/constipation is likely related to the circumferential placement of the mesh. Modification of the procedure to a partial rather than complete mesh wrap has decreased this complication. The judicious use of stool softeners has also proven successful.

Alternate Procedure

For patients considered a poor risk for abdominal surgery, a perineal approach to repair of rectal prolapse is indicated. Perineal rectosigmoidectomy, originally proposed by Altmeier (17) and modified by Prasad (18) is the procedure most often utilized. It is performed with the patient again in lithotomy position and either regional or general anesthesia can be utilized. Because it avoids laparotomy, hospital stay and postoperative



Fig. 4. Perineal rectopexy. The prolapse is reproduced, and a full-thickness circumferential incision is created through the rectal wall. Once the colon has been maximally delivered, it is divided along with its mesentery, and a one-layer anastomosis is created to the distal rectal cuff. Prior to anastomosis, plication of the levator ani muscles is performed and posterior suture of the mesentery to the presacral fascia are accomplished for further fixation.

morbidity are minimized. With the patient in the lithotomy position, the prolapse is reproduced and a full-thickness circumferential incision is created through the rectal wall approx 1 cm proximal to the dentate line. The rectosigmoid colon can then be further prolapsed. Once the colon has been maximally delivered, it is divided along with its mesentery and a one-layer anastomosis is created to the distal rectal cuff. Prior to anastomosis, plication of the levator ani muscles is performed and posterior suture of the mesentery to the presacral fascia are accomplished for further fixation (Fig. 4). Postoperatively, patients resume a diet immediately and are discharged within 1–2 d.

Complications

Complications, including anastomotic stricture or dehiscence and constipation are unusual with this operation. Recurrence rates are generally reported at around 10% (18). Improvement in pre-operative incontinence is unpredictable.

Cost

A 4–5-d hospitalization is usually necessary and the total hospital/surgeon cost is about \$8000.

Summary

- 1. The Ripstein procedure offers patients who are suitable candidates for an abdominal operation a procedure with low recurrence rates and acceptable morbidity.
- 2. The alternative procedure of perineal rectosigmoidectomy has a higher recurrence rate but far lower morbidity and is ideally suited to the older, higher-risk patient.

ANORECTAL PROCEDURES

Anorectal afflictions have troubled the human race for millennia, but remain somewhat of an enigma to a majority of both physicians and laypersons. First described formally in the Chester Beatty Medical Papyrus, written about 1250 BC and further defined by Hippocrates around 400 BC(19), the treatment of these disorders has progressively improved with the wider dissemination of knowledge regarding them and the development of an increasing number of physicians trained specifically in their care (two of the most common anorectal conditions seen in the clinician's office are anal fissure and hemorrhoids). They are not uncommonly confused with one another as both can present with rectal bleeding. Their proper differentiation is crucial to the selection of the appropriate treatment modalities.

LATERAL INTERNAL SPHINCTEROTOMY

Lateral internal sphincterotomy (LIS) is the current appropriate treatment for anal fissure not responsive to nonoperative intervention (20). The previously performed posterior midline sphincterotomy, with or without fissurectomy, has been supplanted by this approach because of the unacceptably high incontinence rate with the former procedure. The procedure is performed in an outpatient ambulatory surgery unit and can be done with general, regional, or local anesthetic techniques.

Indications

The primary indication for the performance of a LIS is a midline anal fissure, which has not responded to dietary manipulation, bulk-forming agents, stool softeners, and topical agents. It is occasionally performed as treatment for symptomatic internal hemorrhoids, though this indication is far less well accepted.

Contraindications

Contraindications include fissures located off the midline, which may indicate a systemic illness (Crohn's disease, ulcerative colitis, tuberculosis, leukemia, syphilis, LGV) and fissures with atypical appearance (broad-based, deep, large, and/or edematous tags), which can be associated with anal carcinoma or HIV/AIDS. The presence of any degree of fecal incontinence in the patient is an absolute contraindication (we prefer to perform a lateral internal sphincterotomy with the patient placed in the prone jackknife position).

Procedure

A single phospho-soda enema is given preoperatively. After preparing the perineum with an antiseptic solution, a local anesthetic solution with epinephrine is injected as a



Fig. 5. Lateral sphincterotomy. (**A**) Visualization of a fissure. (**B**) A superficial incision is created in the lateral position at the anal verge. (**C**) The portion of the internal sphincter distal to the dentate line is divided. (**D**) The wound can then either be closed with an absorbable suture or left open.

perianal block. This is utilized even with a general or regional anesthetic, as it promotes excellent hemostasis. With an appropriate operating anal retractor in place, a superficial incision is created in the lateral position at the anal verge. The distal portion of the internal sphincter (that part distal to the dentate line) is divided with scalpel or cautery, and the wound can then either be closed with an absorbable suture or left open. No specific intervention on the fissure itself is generally required (Fig. 5).

Complications

Significant complications of LIS include infection/abscess, recurrence or failure to heal, and incontinence. In a consecutive series of 53 patients, we noted a 1.8% incidence of abscess formation, a 5.7% incidence of recurrence or failure to heal, and a 1.8% incidence of incontinence to flatus. These figures are consistent with published figures in the literature (21,22). Abscess is easily managed in the office with drainage or superficial fistulotomy. A recurrent or nonhealing fissure will, in most cases, respond to repeat lateral internal sphincterotomy, usually on the opposite side. Anal incontinence, usually to flatus, is a difficult complication commonly related to division of the internal sphincter repair.

Alternative Procedures

Alternative procedures for treatment of anal fissure include excision of the fissure with or without a midline sphincterotomy, an advancement flap repair/anoplasty utiliz-

ing the perianal tissue, and the anal stretch. These are limited by requirement for inhospital stay, technical difficulty, and increased rates of incontinence.

Cost

The cost for this procedure, performed in the Ambulatory Surgery Center, is about \$860 including both surgeon and facility costs.

Summary

- 1. Lateral internal sphincterotomy can be safely performed in the outpatient setting for treatment of chronic anal fissure with low recurrence and complication rates.
- 2. Care must be taken to limit the extent of the sphincterotomy and patients must certainly be counseled regarding the potential for partial incontinence.

HEMORRHOIDECTOMY/BANDING

Operative hemorrhoidectomy and rubber band ligation are the two most-common interventions for symptomatic hemorrhoids today. Both are highly effective when utilized properly. Hemorrhoidectomy refers to the operative excision of the hemorrhoids, usually in the outpatient surgical suite, whereas rubber band ligation is performed in the office setting. Hemorrhoids are generally symptomatic with either bleeding (typically bright red, painless, and commonly dripping into the toilet bowl) or protrusion (occasionally associated with discomfort, itching, or irritation and burning). Pain is usually not a symptom of hemorrhoids unless thrombosis or strangulation has occurred.

RUBBER BAND LIGATION

Rubber band ligation is performed for internal hemorrhoids with bleeding or minor degrees of protrusion. It is not performed for external hemorrhoids in patients with coagulopathies, or generally in patients taking anticoagulants (banding is performed in the office or the outpatient clinic and requires no specific preparation). The patient is placed in the knee-chest or lateral position, an anoscope is inserted, and the hemorrhoidal group to be ligated is visualized. Using a ligator placed through the anoscope, the redundant portion of the mucosa at the upper portion of the hemorrhoid is grasped and a constricting elastic band is placed around it. If the band is placed lower, significant pain may result (Fig. 6). The hemorrhoid will slough in 7–10 d leaving a small, ulcerated area to heal.

Complications

Complications occur in less than 2% of patients and can include pain, vasovagal reaction, thrombosis of external hemorrhoids, massive hemorrhage, and sepsis (23). Pain is generally secondary to a band placed in too low a position, close to the dentate line, and may require removal of the band. Vasovagal reaction is transient and patients respond to reassurance and being placed in the supine position generally. External hemorrhoidal thrombosis is easily managed by excision under local anesthesia. Massive bleeding occurs in 0.5% of patients and is seen at 7–10 d postligation. It must be attended to urgently and may require cauterization or suture ligation. Postligation sepsis (24) is



Fig. 6. Band hemorrhoidectomy. Suction of a hemorrhoid into a banding device introduced through an anoscope, is followed by release of a rubber band around the hemorrhoid resulting in ligation.

a potentially life-threatening complication and when occurring generally occurs 3–4 d postligation. It is generally heralded by fever, increasing pelvi-perineal pain, and urinary retention. Prompt hospitalization, broad-spectrum antibiotics, examination under anesthesia, and debridement of necrotic tissue are required. A colostomy may also be required. Though rare, this complication can be devastating.

OPERATIVE HEMORRHOIDECTOMY

Operative hemorrhoidectomy is performed for hemorrhoids with significant protrusion, large external components, unremitting bleeding, or edematous prolapse. There are no specific contraindications to its use.

Procedure

The procedure is performed in the outpatient ambulatory surgery unit under local, regional, or general anesthesia. We prefer positioning the patient in the prone, jackknife position, the buttocks being taped apart. Regardless of the anesthetic agent, a local anesthetic with epinephrine is used to promote hemostasis. The hemorrhoid is dissected free from the underlying sphincter mechanism, beginning on the anoderm externally and proceeding to the apex of the hemorrhoid internally. The wound is then closed with an absorbable running suture. The process is repeated for each of the three major hemorrhoidal groups, taking care to preserve anoderm between each excision site (Fig. 7).

Complications

Complications include urinary retention, urinary tract infection, bleeding, anal stenosis, incontinence, fecal impaction, infection, and fistula formation. Local infection and fistula formation are quite uncommon, easily recognized, and generally treated in the office with drainage or superficial fistulotomy. Urinary retention is generally secondary to both local pain and the amount of intravenous fluids received intraoperatively. Rates



Fig. 7. Surgical hemorroidectomy. (**A**) Exposure of the hemorrhoid. (**B**) Excision of the hemorrhoid and exposure of submucosal vascular plexuses. (**C**) Dissection of the hemorrhoidal plexuses. (**D**) Wound closure. (**E**) Completed procedure.

up to 17% have been reported (25), but the incidence can be minimized by limiting intraoperative fluids to less than 300 cm³. Bleeding can be immediate, i.e., in the recovery room, or delayed, at between 7–16 d postoperatively (25). If hemodynamically significant, this must be addressed in an operating room with suture ligation or cautery. Anal stenosis and incontinence are both the result of overzealous excision-stenosis from the failure to preserve native tissue between excision sites and incontinence from excision of sphincter muscle fibers with the hemorrhoidal cushions. Anal stenosis, if present, may require subsequent anoplasty. Incontinence is very difficult to correct and is generally addressed nonoperatively. Fecal impaction is generally avoided by the institution of bulking agents and stool softeners but can also be treated with enemas. Rarely, disimpaction under general anesthesia may be required.

Alternative Procedures

Alternative procedures to banding and operative hemorrhoidectomy include sclerosis, cryosurgery, infrared coagulation, and laser hemorrhoidectomy. These procedures have similar success rates but slightly higher incidence of discomfort or requirement for additional expensive equipment. Laser hemorrhoidectomy has been shown to offer no benefit over standard operative therapy (26), but requires much more expensive operating room equipment.

Cost

Operative hemorrhoidectomy results in a cost of approx \$2200, including both a surgical and out-patient facility fee. Rubber band ligation of internal hemorrhoids, usually requiring three bands and performed in the office or clinic, costs about \$500.

Summary

- 1. LIS, rubber band ligation, and operative hemorrhoidectomy are all outpatient treatments and, although costs vary widely according to region, represent low-cost options for treatment of anal fissures and symptomatic hemorrhoids.
- 2. When carefully performed by an experienced operator, success rates are high with concomitantly low complication rates.

REFERENCES

- Rosen L, Stasik JJ, Reed JF, et al. Variations in colon and rectal surgical mortality. Comparison of specialties with a state-legislated database. Dis Colon Rectum 1996;39:129–135.
- 2. Le TH, Timmcke AE, Gathright JB, et al. Outpatient bowel preparation for elective colon resection. South Med J 1997;90:526–530.
- 3. Nichols RL, Condon RE. Preoperative preparation of the colon. Surg Gynecol Obstet 1971;132:323-337.
- 4. Heald RJ, Ryall RD. Recurrence and survival after total mesorectal excision for rectal cancer. Lancet 1986;1:1479–1482.
- 5. Manson PN, Corman ML, Coller JA, et al. Anterior resection for adenocarcinoma. Lahey Clinic experience from 1963 through 1969. Am J Surg 1976;131:434–441.
- 6. Max E, Sweeney WB, Bailey HR, et al. Results of 1000 single-layer continuous polypropylene intestinal anastomoses. Am J Surg 1991;162:461–467.
- 7. Griffen FD, Knight CD, Whitaker JM, Knight CD Jr. The double stapling technique for low anterior resection. Results, modifications, and observations. Ann Surg 1990;211:745–751.
- 8. Rosen L, Veidenheimer MC, Coller JA, Corman ML. Mortality, morbidity, and patterns of recurrence after abdominoperineal resection for cancer of the rectum. Dis Colon Rectum 1982;25:202–208.
- 9. Fazio VW, Fletcher J, Montague D. Prospective study of the effect of resection of the rectum on male sexual function. World J Surg 1980;4:149–152.
- Metcalf AM, Dozois RR, Kelly KA. Sexual function in women after proctocolectomy. Ann Surg 1986; 204:624–627.
- 11. Corman ML, Veidenheimer MC, Coller JA, Ross VH. Perineal wound healing after proctectomy for inflammatory bowel disease. Dis Colon Rectum 1978;21:155–159.
- 12. Martin LW, LeCoultre C, Schubert WK. Total colectomy and mucosal proctectomy with preservation of continence in ulcerative colitis. Ann Surg 1977;186:477–480.
- Parks AG, Nicholls RJ, Belliveau P. Proctocolectomy with ileal reservoir and anal anastomosis. Br J Surg 1980;67:533–538.
- DeLaurier GA and Nelson J. Ileal pouch-anal anastomosis. In: Hicks T, Beck D, Opelka F, Timmke A, eds. Complications of Colon and Rectal Surgery. Williams and Wilkins, Baltimore, MD, 1996, p. 339.
- 15. Goldberg SM, Gordon PH, Nivatvongs S. Rectal Prolapse. In: Essentials of Anorectal Surgery, JB Lippincott, Philadelphia, PA, 1980, p. 248.
- 16. Gordon PH, Hoexter B. Complications of the Ripstein Procedure. Dis Colon Rectum 1978;21:277–280.
- 17. Altmeier WA, Culbertson WR, Schowengerdt C, et al. Nineteen years' experience with the one-stage perineal repair of rectal prolapse. Ann Surg 1971;173:993–1006.
- Prasad ML, Pearl R, Abcarian H, et al. Perineal proctectomy, posterior rectopexy and post-anal levator repair for treatment of rectal prolapse. Dis Colon Rectum 1986;29:547–552.
- 19. Bernstein WC. Foreword: The History of Anorectal Surgery. In: Goldberg SM, Gordon PH, Nivatvongs S., eds. Essentials of Anorectal Surgery. JB Lippincott, Philadelphia, PA, 1980, pp. ix-xv.
- Eisenhammer S. The evaluation of internal anal sphincterotomy-operation with special reference to anal fissure. Surg Gynecol Obstet 1959;109:583–590.
- 21. Hsu TC, MacKeigan JM. Surgical treatment of chronic anal fissure: a retrospective study of 1753 cases. Dis Colon Rectum 1984;27:475–478.

- Jensen SL, Lund F, Nielsen OV, Tange G. Lateral subcutaneous sphincterotomy versus anal dilatation in the treatment of fissure-in-ano in outpatients: a prospective randomized study. Br Med J 1984;289: 528–530.
- Rothberg R, Rubin RJ, Eisenstat T, Salvati EP. Rubber band ligation hemorrhoidectomy: long term results. Am Surg 1983;49:167.
- 24. Russell TR, Donahue JH. Hemorrhoidal banding: a warning. Dis Colon Rectum 1985;28:291-293.
- 25. Bleday R, Pena JP, Rothenberger DA, et al. Symptomatic Hemorrhoids: current incidence and complications of operative therapy. Dis Colon Rectum 1992;35:477–481.
- Senagore A, Jazier WP, Luchtefeld MA, MacKeigan JM, Wengert T. Treatment of advanced hemorrhoidal disease: a prospective randomized comparison of cold scalpel vs contact Nd:YAG laser. Dis Colon Rectum 1993;36:1042–1049.
- 27. Feil, W. Atlas of Surgical Stapling. Johann Ambrosius Barth, Heidelberg, Germany, 2000.
- 28. Shackelford S. Surgery of the Alimentary Tract, Volume 4, Fifth Edition, WB Saunders, Philadelphia, PA, 2002.
- 29. Keighley, MR. Atlas of Colorectal Surgery. Churchill Livingston, New York, 1996.
- 30. Goldberg, SL et al. Essentials of Anorectal Surgery. JB Lippincott, Philadelphia, PA, 1980.



HEPATIC AND BILIARY SURGERY

17 Hepatic Resection

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INTRODUCTION

The liver is the only organ in the human body capable of regenerating functional tissue: an ability for which it is celebrated in the rather grisly myth of Prometheus. Modern hepatic resections are done to remove inflammatory and neoplastic tumors situated in the parenchyma, occasionally for trauma, and when the portal blood supply to a section of liver is sacrificed during resection of cholangiocarcinoma of the proximal bile ducts. It is reasonable to expect that the remaining liver will grow back to a normal size over the first 3 mo postoperatively, assuming the remaining liver is normal. It may do so even more quickly than that (1-3). Growth of hepatic grafts in transplanted patients suggests that a normal liver volume in an adult is approx 25 mL per kg of body weight (4,5). Determining that hepatocellular mass adequate to maintain normal liver function remains or develops soon after hepatectomy is one of the most challenging and important perioperative medical calculations for these patients.

SURGICAL ANATOMY

The liver has a dual blood supply, which enters it as a single portal "cable." The portal vein normally supplies approx 80% of the blood flow to the hepatocytes. The hepatic artery supplies a smaller percentage, but is an important source of blood flow to the biliary tree. Venous drainage occurs via the hepatic veins, which empty into the inferior

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Fig. 1. Hepatobiliary anatomy and nomenclature. Note the relatively longer and extrahepatic course of the left hepatic duct, and its entry into the hepatic substance at the umbilical fissure. The common duct is named the common hepatic duct above the junction of the cystic duct, and the common bile duct below this junction. The common bile duct passes beneath the first portion of the duodenum to run in the pancreas and duodenal wall until it empties into the second portion of the duodenum at the ampulla of Vater.

vena cava (IVC) just below the heart at the top of the liver. The human liver is divided into right and left lobes based on the bifurcation of the portal structures (bile ducts, portal vein, and hepatic artery all within a sheath of Glisson's capsule) at the hepatic hilum, rather than on external appearances. Although the anatomy of the portal structures in hepatic hilum may be quite variable, the main right portal branch typically bifurcates again quickly into anterior and posterior segments within the substance of the liver. The main left portal branch is longer and runs outside the liver before it dives in at the falciform ligament; then it branches to the medial and lateral segments of the left lobe (Fig. 1). The falciform ligament is a fatty structure that runs from the umbilicus to the umbilical fissure in the liver and is a conspicuous feature both on inspection of the external surface of the liver and on computed tomography (CT) scan. Consequently, the falciform is often misapprehended as dividing the larger right lobe from a smaller left lobe. In fact, the two main lobes are roughly equivalent in size and the falciform marks a division between the medial and lateral segments of the left lobe. A line (Cantlie's line) between the gallbladder and the IVC marks the parenchymal plane separating the left and right lobes of the liver. It is also known as the main portal fissure. This plane is also inhabited by the main trunk of the middle hepatic vein. The main trunk of the right hepatic vein runs in a plane between the anterior and posterior segments of the right lobe of the liver, and the left hepatic vein runs in the plane between the medial and lateral segments of the left lobe. The caudate lobe, which is also called segment 1 in the Hepp-Couinad classification, sits underneath the portal structures and on top of the IVC. Using this classification, eight segments are identified. The left lobe contains segments 2, 3, and 4. The right lobe contains segments 5, 6, 7, and 8 (Fig. 2).



Fig. 2. Segmental structure of the liver and it blood supply.

TYPES OF HEPATECTOMY

Resections

Although 75% of a normal liver may be resected with a reasonable expectation that enough hepatic function will remain to support regeneration of the hepatic remnant, this percentage is significantly reduced when the liver parenchyma is diffusely diseased. A larger hepatic remnant will be necessary to support regeneration and reduce the risk of fulminant hepatic insufficiency. Typically, when more than 50% of the functional capacity of the liver is removed, the possibility of liver failure becomes a threat. For these reasons, judgments about the advisability of a major resection are based upon both estimates of the normalcy of the residual hepatic cell mass (e.g., no cirrhosis) as well as how much of the functioning tissue will remain after the resection. For example, a right trisegmentectomy may be done relatively safely for a large tumor that has replaced the right lobe and medial segment of the left lobe because most hepatic function will already have shifted to the left lateral segment. However, if a trisegmentectomy is done for a small tumor that simply straddles the right lobe and medial segment of the left lobe, a relatively large amount of functional parenchyma is sacrificed and the risk of liver failure increases proportionally. Often, the presence or absence of hypertrophy of the putative hepatic remnant can be appreciated grossly either by CT scan or at operation.

Hepatic resections are frequently termed "anatomic" when an entire lobe (Fig. 3A,C) or segment (Fig. 3D) has been removed, or "nonanatomic" when a piece of liver tissue has been taken without regard for segmental anatomy described earlier. Excisional wedge biopsies of small lesions in the liver are an example of a nonanatomic resection (Fig. 3E). "Extended" resections refer to resections that include more than just a right- or left hemi-hepatectomy; usually another segment. The most common extended hepatectomy is the right trisegmentectomy, which includes the medial segment of the left lobe in addition to the right lobe (Fig. 3B); the left lateral segment (segments 2 and 3) remains. A left trisegmentectomy is harder to perform and is called for infrequently. The easiest anatomic resection to perform is a left lateral segment-ectomy (Fig. 3D). For really large tumors requiring an extended resection, the plane of resection is often dictated by the configuration of the tumor.



Fig. 3. Anatomical landmarks for hepatic resections. (A) Right hepatectomy. (B) Right trisegmentectomy. (C) Left hepatectomy. (D) Left lateral segmentectomy. (E) Wedge resection.

Enucleation

Some benign tumors can be shelled out of the hepatic parenchyma by working in the pseudocapsule of compressed normal liver tissue adjacent to the expanding tumor. This technique is called enucleation and its great advantage is that large tumors can be removed without sacrificing much normal liver tissue or crossing major vascular channels. The main disadvantage is that there is no margin of normal tissue taken around the tumor, so that the technique is generally not employed for malignant tumors. However, for some benign tumors such as hemangiomas, it may be the ideal approach.

Cryoablation and Radiofrequency Ablation

There are several other nonresectional techniques for obliterating tumors in the hepatic parenchyma. These modern ablative procedures include cryoablation and radiofrequency ablation (RFA). In both cases, a major operation is typically required, although both may be employed laparscopically and even percutaneously in select circumstances. In both cases, a probe is placed into the tumor, typically under ultrasound guidance. For RFA, microwave energy is passed down the probe into the tumor; cooking it to death. For cryoablation, liquid nitrogen is passed down the probe creating an ice ball; freezing the tumor to death. It is easier to follow the growth of the ice ball in relation to tumor margin with ultrasound than it is to follow the RFA coagulative lesion in real time, so the security of the margins may theoretically be better with cryoablation. However, RFA is more

widely available. In both cryoabalation and RFA, the frozen or coagulated tissue is typically left *in situ*; to be gradually resorbed later. These thermal ablation techniques are more akin to nonanatomic resections in that they may be undertaken without regard for segmental anatomy.

There are limitations, however. Tumors adjacent to the major vascular channels (portal or hepatic vein or IVC) may not be reliably ablated because the blood flow acts as a thermal sink, protecting the malignant cells on the vessel wall from the applied temperature extremes. The great advantage of these techniques is that they can destroy tumor in disparate parts of the liver and preserve intervening parenchyma. They are, therefore, ideally suited to smaller tumors. Indeed, the larger the amount of dead (frozen or coagulated) left in the liver, the more likely are adverse systemic consequences. These adverse consequences are a variant of a systemic inflammatory response and include thrombocytopenia, myoglobinuria, ATN, and noncardiogenic pulmonary edema. They are increasingly prevalent as the volume of ablated tissue passes 30% of the liver volume, and appear to be more prevalent following cryoabalation than following RFA (6).

INDICATIONS FOR LIVER RESECTION

Unless a piece of normal liver is being removed for transplantation, the indications for resection of the liver revolve around the presence of an abnormality in the hepatic parenchyma or bile ducts, which can and should be removed. Neoplasms are removed either because they are or could be malignant, or because they are causing symptoms that will be improved by hepatectomy. Other lesions, such as abscesses, are removed because specific circumstances suggest that resection, rather than drainage, is the least morbid treatment. Often, the circumstances in which resection is chosen over drainage for these patients involve anatomic abnormalities such as localized biliary strictures that have provoked the infectious problem. Intrahepatic biliary strictures may be caused by devascularizing events such as previous surgery or following trauma. They can also occur as a result of several other problems such as stones, or Caroli's disease, or even parasitic infections like Clonorchis (Oriental Cholangiohepatitis). Occasionally, a piece of devitalized liver is resected along anatomic planes as a posttraumatic debridement or to control bleeding.

The most common reason tumors are removed from the liver is to attempt a cure for a patient with a malignant neoplasm. Primary hepatocellular cancer, or hepatoma, is the most common malignant solid tumor worldwide. However, it is much less common in the United States. Here, the most common malignant neoplasms of the liver are metastatic deposits from lung, breast, and gastrointestinal (GI) primary sites. Some of these patients can be cured by removing the metastases from the liver. However, they are a highly selected group who have a small number of isolated colorectal or neuroendocrine metastases in the liver. Patients with metastatic lung, pancreatic, breast or gastric cancer are not reliably salvaged by resectional or ablative strategies for the liver metastases. Similarly, a minority of patients with hepatoma can be cured by resection and/or ablation because these tumors tend to present at a late stage or in a cirrhotic liver that will not tolerate a resection. Nevertheless, there is no other curative treatment for these cancers and so if resection or ablation is feasible, it should be undertaken.

For both primary hepatocellular cancer and metastatic colorectal cancer, there is currently no demonstrable benefit to "debulking" the cancer. Consequently, operative resection or ablation should be undertaken only if a complete resection with clear margins can be contemplated by preoperative review of the scans. The key to technical success within the liver is a clear margin on the target tumor and it does not matter whether these are achieved by anatomic or nonanatomic resections or ablations. These basic principles are valid for a number of other more uncommon cancers such as peripheral cholangiocarcinomas and sarcomas. The major exception is when the tumor is hormonally active as happens with some neuroendocrine cancers. In these cases resectional or ablative maneuvers that only debulk the patient can have a significant palliative impact and should be contemplated despite expectations that the disease will recur locally.

Benign tumors are removed when there is doubt about their benign nature as may happen with any tumor, but particularly with hemangiomas, which should not be biopsied because of the possibility of precipitating uncontrolled bleeding, and hepatocellular adenomas, which may be confused with well differentiated hepatoma. They are also removed when they threaten other complications such as hemorrhage. Interestingly, the threat of spontaneous rupture and bleeding is from adenomas, not hemangiomas as was commonly feared (7,8). Finally, benign tumors are removed when they cause symptoms such as pain, or symptoms consistent with a mass effect (satiety, breathlessness, distention, and so on), or rare systemic problems such as the consumptive coagulopathy that can occur with giant hemangiomas. Cysts are operated on for the same sorts of reasons: doubt about their benign nature (e.g., complex cysts) or because they are symptomatic. In the latter case, the operation performed is a fenestration; the top of the cyst is excised to allow it to drain freely into the peritoneal cavity.

A major hepatic resection is sometimes required to remove cholangiocarcinomas of the proximal bile duct. These tumors, which frequently involve the bifurcation of bile ducts, are also known as Klatskin's tumors. The cancer often extends proximally and distally along the bile ducts so that achieving a clear luminal margin, particularly on the hepatic side, with a segmental resection of the bile duct can be problematic. The solution is to take the liver as well as the bile duct. Achieving clear radial margins can also be a problem because the bile ducts run as one component of a "cable" made up of the portal veins and hepatic arteries (Fig. 4). Consequently, achieving a clear radial margin may involve the sacrifice of these vascular structures and the hepatic parenchyma they subtend.

COMPLICATIONS

For purposes of discussion, the acute complications of hepatic resections and ablations may be lumped into three categories. Those occurring as a result of an underlying disease process that provoked the need for operation in the first place, those occurring as a result of any major upper abdominal procedure, and those related to the resection or destruction of liver tissue. Of course, in any individual patient these may all be active at the same time. General prognostic markers for the risk of complications and mortality following hepatectomy include the volume of liver resected, volume of blood loss, and need for transfusion, cirrhosis, jaundice and cholangitis particularly, renal insufficiency, and the need for caval resections (9,10).

In addition to cardiovascular difficulties that can occur with any large operation, particular complications of major upper abdominal surgery include perioperative hemorrhage, intrabdominal collections, ileus, pancreatitis, significant incisional pain with



Fig. 4. Glissonian Cable at the hilus of the liver extending into the liver substance. Glisson's capsule also surrounds the liver.

consequent atelectasis and fever, wound infections, and dehiscence. Patients are also at risk for acute tubular necrosis and renal insufficiency if isotonic replacement of third space losses in the perioperative period is inadequate. Diabetic patients commonly become transiently more glucose intolerant from the stress of the operation; and all patients should be supported nutritionally if they are unable to use their GI tract within in 5 or 6 d of operation. Many of these problems can be anticipated and steps taken to either avoid them or lessen their impact. When the major upper abdominal procedure is a hepatectomy or an ablation, there is perhaps a greater likelihood of sympathetic pleural effusions because subdiaphragmatic surface are routinely irritated by the operation and healing processes. Also, the large raw surfaces of liver and empty subdiaphragmatic spaces may result in a slightly greater likelihood of bile and infected fluid collections forming postoperatively than for example happens after gastrectomy. Bile leaks can develop when clips or sutures on the divided liver slough off, or when a section of bile duct necroses following a cryoablation or radiofrequency ablation. The problem typically becomes manifest 5–10 d postoperatively, although it is not uncommon for the collection to present with a vague symptom complex best described as "failure to thrive" at 3–5 wk out from operation. When these collections occur, they can usually be handled by percutaneous drainage. Interestingly, leaving a drain in the operative field at the time of surgery does not appear to decrease the incidence of this complication, or the need for percutaneous drainage (11).

When the liver has been resected or ablated, a variable amount of dead or devascularized tissue may be left *in situ*. This can be inadvertent such as occurs at the margins of a formal hepatectomy, when more of the liver may be devascularized than is resected. However, a volume of dead and devascularized tissue encompassing the entire tumor and surrounding margin of normal parenchyma is routinely left after ablation procedures. As noted earlier, this necrotic tissue may provoke a systemic inflammatory
response in addition to the complications noted above. It is important to realize that the necrotic tissue need not be infected to provoke this syndrome. A CT scan obtained in the first 10 d after a cryoablation may be misleading in this regard because it can show air even in a normally resorbing cryolesion (12). Another particular complication of cryoablation and RFA is the accidental, unrecognized ablation of a structure abutting the liver while the ablation lesion within the liver is being carefully and safely monitored with ultrasound. The structures at risk for this complication are the diaphragm and lung, the gallbladder, the hepatic flexure of the colon, duodenum, and any adherent small intestine.

After a significant hepatic resection, the liver begins to regenerate within 12–36 h (2,4,13-15). This blessed event is often heralded by a precipitous drop in serum phosphorus and an exacerbation of the mild hepatic insufficiency, which accompanies removal of a large amount of functional liver. The reason is that hepatocytes use large amounts of ATP as their task changes from differentiated hepatic synthetic and excretory function to cell division (13,16). It is important to keep the patient hydrated during this period, to replete phosphorus, and not let the prothrombin time get too prolonged (> 16 sec) because that may lead to a delayed bleed in the operative field. The hepatic insufficiency is usually mild and transitory; clearing by postoperative day 5.

The most feared liver-specific complication following hepatectomy is liver failure. This can be provoked straightforwardly by removing or devascularizing too much functional tissue. It can also develop more insidiously in the postoperative period when the liver fails to regenerate. The reasons why this happens are varied. Infection is certainly one culprit and occult infections should be sought and aggressively treated if the problem develops. Hepatotoxic drugs are another cause. Therefore, the patient's medex should be scrutinized and modified. Thrombosis of either the portal vein or hepatic artery does occur in the postoperative period and can lead to this problem. Consequently, these vessels, and the remaining hepatic veins, should be studied, typically by Doppler ultrasound first, if the patient develops hepatic failure. Another reason patients slide into hepatic failure postoperatively is that their remaining liver tissue was not normal before the resection and is incapable of the amount of regeneration required for survival. Although this may be a result of longstanding biliary obstruction or extensive fatty infiltration of the liver, by far and away the most common reason is cirrhosis. Also, it is difficult to judge how much hepatic reserve exists in a patient with cirrhosis, before the resection. As a result, even a minor resection in these patients may turn out to have a functional impact equivalent to an extended resection in a normal patient. For these reasons, major resections (more than a segment) are undertaken very hesitantly in cirrhotic patients, and then mostly in Child's Class A patients if they don't have portal hypertension (17). Indeed, partial hepatectomy in cirrhotic patients worsens portal hypertension acutely; increasing the likelihood of problems with ascites and variceal hemorrhage.

LONG-TERM COMPLICATIONS OF HEPATIC RESECTION

Despite the myriad of possible acute complications associated with hepatic resection, there are few long-term sequelae once the liver has regenerated. There is no particular evidence that the regenerated hepatic parenchyma is more fragile or susceptible to hepatotoxic drugs. Repeat hepatic resection may even be done safely if the indications and circumstances warrant it (18–20). Patients certainly can develop wound problems such as hernias or chronic pain. They infrequently develop biliary strictures as a result of chronic inflammation, iatrogenic low-grade ischemia, or intrarterial chemotherapy. The clinician caring for these patients should be aware that the orientation of portal structures in the hilum is frequently rotated following a major resection and regeneration, as this knowledge can be helpful interpreting radiological studies.

Still, the most common late problem encountered by patients who have undergone some form of hepatectomy is a recurrence of the disease that precipitated the need for the original hepatectomy. For example, approx 30% of equivalently selected patients who have undergone either hepatectomy or ablations for colorectal liver metastases will have the first recurrence of their tumor confined to the liver (21-23). Patients who have undergone a hepatectomy for hepatoma usually face not only the risk of recurrence of their tumor, but also the progression of cirrhosis and complications of portal hypertension. Nevertheless, the risk of tumor reappearance in the liver of patients with severely cirrhotic livers is very high (24). Many of these "recurrences" may really be new tumors arising in the damaged field, but their appearance within 5 yr of successful resection or ablation is unfortunately quite reliable and ultimately lethal.

Patients are often followed for recurrence of their tumors with serum markers (CEA for colorectal, CA 19-9 for biliary, and alpha fetoprotein for hepatocellular cancer) and with CT scans or MRI. Patients who have undergone an ablation of their tumor should have a new "baseline" CT or MRI obtained at 6–8 wk postoperatively. Subsequent scans should confirm that the ablation lesion is either the same size or smaller. Growth of the ablation lesion suggests a local recurrence at that site as opposed to the growth of other, previously unappreciated, hepatic metastases. PET scans will probably be a worthwhile way to evaluate suspicious ablation sites in the near future. The value of aggressive radiological follow-up depends to some extent on what can be done about a recurrence of the tumor when it is found.

CONTRAINDICATIONS

For all the reasons detailed earlier, the major contraindication to hepatic resection is cirrhosis or evidence of compromised hepatic function. Although patients should be medically fit enough to undergo a major operative procedure, age *per se* has not been shown to be a contraindication for elective hepatic surgery. This is an important point because many patients presenting with hepatic malignancies are in the seventh and eighth decades of life.

COST

A major hepatic resection with a relatively uncomplicated postoperative course generates between \$24,000–\$30,000 in hospital charges. Professional charges by surgeons, anesthetists, and other consultants run in the range of \$8000–\$10,000.

SUMMARY

- 1. The liver can grow back after it is resected; so that as much as 75% can be surgically removed as long as that remaining works well.
- 2. The more surrounding normal liver parenchyma is removed in a resection of a tumor, the more likely is liver failure.

- 3. Cirrhotic livers do not regenerate or tolerate resection as well as noncirrhotic livers and so resections and ablations in these circumstances should be approached cautiously.
- 4. Remediable causes of liver failure after hepatic surgery include drugs, infections, complications such as bile leaks and other intrabdominal leaks, and vascular problems such as portal vein thrombosis.
- 5. The goal of resection and ablations of malignant hepatic tumors is complete extirpation; debulking does not improve outcome except as palliation for functional endocrine metastases.

REFERENCES

- 1. Ezaki T, Koyanagi N, Toyomasu T, et al. Natural history of hepatectomy regarding liver function: a study of both normal livers and livers with chronic hepatitis and cirrhosis. Hepatogastroenterology 1998;45:1795–1801.
- 2. Marcos A, Fisher RA, Ham JM, et al. Liver regeneration and function in donor and recipient after right lobe adult to adult living donor liver transplantation. Transplantation 2000;69:1375–1379.
- 3. Zoli M, Marchesini G, Melli A, et al. Evaluation of liver volume and liver function following hepatic resection in man. Liver 1986;6:286–291.
- 4. Kawasaki S, Makuuchi M, Ishizone S, et al. Liver regeneration in recipients and donors after transplantation. Lancet 1992;339:580–581.
- 5. Chari R.S, Baker M.E, Sue SR, et al. Regeneration of a transplanted liver after right hepatic lobectomy. Liver Transpl Surg 1996;2:233–234.
- 6. Chapman WC, Debelak JP, Blackwell TS, et al. Hepatic cryoablation-induced acute lung injury: pulmonary hemodynamic and permeability effects in a sheep model. Arch Surg 2000;135:667–672; discussion 672–673.
- 7. Lise M, Feltrin G, Da Pian PP, et al. Giant cavernous hemangiomas: diagnosis and surgical strategies. World J Surg 1992;16:516–520.
- Trastek VF, van Heerden JA, Sheedy PF, et al. Cavernous hemangiomas of the liver: resect or observe? Am J Surg 1983;145:49–53.
- 9. Melendez J, Ferri E, Zwillman M, et al. Extended hepatic resection: a 6-year retrospective study of risk factors for perioperative mortality. J Am Coll Surg 2001;192:47–53.
- Gozzetti G, Mazziotti A, Grazi GL, et al. Liver resection without blood transfusion. Brit J Surg 1995; 82:1105–1110.
- 11. Fong Y, Brennan MF, Brown K, et al. Drainage is unnecessary after elective liver resection. Am J Surg 1996;171:158–162.
- 12. Kuszyk BS, Choti MA, Urban BA, et al. Hepatic tumors treated by cryosurgery: normal CT appearance. AJR Am J Roentgenol 1996;166:363–368.
- Farghali H, Rilo H, Zhang W, et al. Liver regeneration after partial hepatectomy in the rat. Sequential events monitored by 31P-nuclear magnetic resonance spectroscopy and biochemical studies. Lab Invest 1994;70:418–425.
- Miyazaki S, Takasaki K, Yamamoto M, et al. Liver regeneration and restoration of liver function after partial hepatectomy: the relation of fibrosis of the liver parenchyma. Hepatogastroenterology 1999;46: 2919–2924.
- 15. Nagasue N, Yukaya H, Ogawa Y, et al. Human liver regeneration after major hepatic resection. A study of normal liver and livers with chronic hepatitis and cirrhosis. Ann Surg 1987;206:30–39.
- 16. George R, Shiu MH. Hypophosphatemia after major hepatic resection. Surgery 1992;111:281-286.
- 17. Fong Y, Sun RL, Jarnagin W, et al. An analysis of 412 cases of hepatocellular carcinoma at a Western center. Ann Surg 1999;229:790–799; discussion 799–800.
- 18. Elias D, Lasser P, Hoang JM, et al. Repeat hepatectomy for cancer. Br J Surg 1993;80:1557–1562.
- 19. Adam R, Bismuth H, Castaing D, et al. Repeat hepatectomy for colorectal liver metastases. Ann Surg 1997;225:51–60; discussion 60–62.
- Bismuth H, Adam R, Navarro F, et al. Re-resection for colorectal liver metastasis. Surg Oncol Clin N Am 1996;5:353–364.
- 21. Hughes KS, Rosenstein RB, Songhorabodi S, et al. Resection of the liver for colorectal carcinoma metastases. A multi- institutional study of long-term survivors. Dis Colon Rectum 1988;1:1–4.

- 22. Seifert, JK, Morris D.L. Prognostic factors after cryotherapy for hepatic metastases from colorectal cancer. Ann Surg 1998;228:201–208.
- 23. Curley SA, Izzo F, Delrio P, et al. Radiofrequency ablation of unresectable primary and metastatic hepatic malignancies: results in 123 patients [see comments]. Ann Surg 1999;230:1–8.
- 24. Bilimoria MM, Lauwers GY, Doherty D, et al. Underlying liver disease, not tumor factors, predicts long-term survival after resection of hepatocellular carcinoma. Arch Surg 2001;136:528–535.

Bypass and Reconstruction of Bile Ducts

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BILIARY-ENTERIC ANASTOMOSIS INDICATIONS CHOICE OF BILIARY-ENTERIC ANASTOMOSIS COMPLICATIONS OF BILIARY-ENTERIC ANASTOMOSIS COST SUMMARY REFERENCES

BILIARY-ENTERIC ANASTOMOSIS

When the bile duct is obstructed, it may be surgically bypassed. When resected, it must be surgically reconstructed. In both cases, the small intestine is anastomosed to the biliary tree. The only variation is which piece of small intestine is used for the anastomosis and how it is brought up to the biliary tree. The names of these procedures are based on these variations and the level of the anastomosis on the biliary tree. For example, a side-to-side choledochoduodenostomy refers to an anastomosis between the common bile duct and the second portion of the duodenum (Fig. 1A). This is one of the simplest biliary bypasses to perform and is occasionally done in an end-to-side fashion (Fig. 1B). Similarly, a hepaticojejunostomy refers to an anastomosis between the hepatic duct (common and above) and jejunum. Although these anastomoses may be done with a loop of jejunum (e.g., cholecystojejunostomy), the workhorse is a Roux-Y limb of jejunum. The principle recommending a Roux limb is that peristalsis remains directed downstream for the limb and the gastrointestinal (GI) tract (Fig. 2). Consequently, no food will be brought to the biliary tree by the gut as a matter of normal function, and better drainage is expected. Again, Roux-en Y anastomoses are named for which part of the biliary tree is anastomosed to the end of divided jejunum; "Choledochojejunostomy" and "Cholehepaticojejunostomy," or just plain "hepaticojejunostomy" if the anastomosis is at or above the bifurcation of the bile ducts. The Roux limb should be 40–70 cm in length and constructed from proximal jejunum. Accidental use of more distal intestine risks diarrhea and malabsorption.

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Fig. 1. Diagrams of choledochoduodenostomies. (A) Side-to side. (B) End-to-side.



Fig. 2. Roux-en-Y jejunal limb. Arrow depict the direction of peristaltic movement.



Fig. 3. Bile duct blood supply. Note the rich network of blood vessels from the pancreas and duodenum in the infraduodenal portions, and from the right and left hepatic arteries at the bifurcation. In between, the blood supply is axial.

INDICATIONS

The usual indications for a bypass are obstruction by a benign or a malignant stricture. Benign strictures are particularly likely to occur in the distal bile duct as a result of stones, or chronic pancreatitis. Benign strictures in the middle of the of the common duct, up to and including the bifurcation can also be caused by stones, parasitic infestations, autoimmune inflammation of the ducts (e.g., sclerosing cholangitis), congenital problems (e.g., Caroli's disease), or even trauma. However, the most common reason is iatrogenic: the most notorious of these in modern times being an operative injury during laparoscopic cholecystectomy. The injury may occur by transaction, inadvertent clip placement, cauterization or indirectly by devascularization of the duct. Because the blood supply to the mid-bile duct is axial (Fig. 3), it is relatively susceptible to devascularization. The injury may or may not be recognized at the original surgery. If it is recognized and repaired simply by reapproximating the divided duct, the repair often fails; resulting either in bile leakage, which occurs early, or stricture formation, which occurs several months to years after the surgery. Rarely, late bile duct stricture may develop because of neuroma formation around the cystic duct stump following an apparently uncomplicated operation (1). In addition to laparoscopic surgery, several other operations and therapeutic maneuvers can result in bile duct injury with subsequent benign stricture as a complication. These include misadventures during operations on the common bile duct, hepatic resections, duodenal and pancreatic operations, and gastric operations for peptic ulcer disease. Endoscopic retrograde cholangiopancreatography (ERCP) and sphincterotomy can damage the bile duct as well as infusions of chemotherapy into the hepatic artery, and misadventures during percutaneous trans-hepatic procedures (e.g., stent placements, cholangiograms, biopsies, and so on). In the absence of an obvious cause for a bile duct stricture, it can be difficult to distinguish benign and malignant strictures, and this uncertainty often animates therapeutic choices.

Malignant obstructions or strictures can also occur at any level in the biliary tree, but most commonly involve the distal end as a result of a ductal adenocarcinoma of the head of the pancreas. Adenocarcinoma of the ampulla of Vater, the duodenum, and even the bile duct (cholangiocarcinoma) all occur in the same region and can be virtually impossible to distinguish from cancer of the head of the pancreas preoperatively. When resectable, these tumors are all handled by a Whipple procedure (*see* Chapter 20). Primary cholangiocarcinoma arising in the mid-bile duct is also occasionally treatable with a segmental resection of the bile duct, and this should be done if it is feasible. More commonly, proximal cholangiocarcinomas involve the bifurcation of the bile ducts (Klatskin's tumors). To achieve clear margins for these tumors often involves hepatic parenchymal resection too as was aforementioned. The bile duct may finally be obstructed by malignant ingrowths from other adjacent organs. This happens most commonly with locally advanced carcinoma of the gallbladder, but can occur from nodal metastases from gastric or colon cancer, or even lymphomas involving the portal lymph nodes in the hepatoduodenal ligament.

Another indication for biliary-enteric procedures is biliary atresia in infants. These infants may have only vestigal remnants of their biliary tree. The ideal situation for a biliary-enteric anastomosis is one in which the intrahepatic ducts are normal and only the extrahepatic ducts are atretic. When there are no obvious dilated intrahepatic ducts and the condition is recognized before liver failure and cirrhosis supervene, the infant may undergo a procedure called the Kasai portoenterostomy. This operation also involves reconstruction of the extrahepatic biliary tree with a Roux-Y limb up to the hepatic hilum where the intrahepatic ducts are supposed to be. The area where the atretic ducts are above the portal vein bifurcation is cored out with hepatic parenchyma, and the jejunum is sewn to the liver there in anticipation of bile drainage from microscopic bile ductules. Success depends upon performing this operation soon enough to avoid the complications of liver failure, and on how much of the biliary tree is atretic. If there are no intrahepatic ducts at all, the Kasai procedure will fail and liver transplant must be performed then. After one of these operations, the effluent into the roux limb is carefully monitored for bile because a remarkable number (40%) of these infants will survive if bile flow develops and their jaundice clears. The incidence of repetitive episodes cholangitis in survivors is quite high and surviving children must be watched for the later development of cirrhosis and its attendant complications. Liver transplant can salvage these patients even after a Kasai portoenterostomy (2,3).

Finally, patients may undergo either a bypass or reconstruction after a resection of a type 1 choledochal cyst. In this congenital biliary abnormality, the extrahepatic bile duct balloons out. Patients suffer repetitive attacks of cholangitis and are subsequently at higher risk for the development of cholangiocarcinoma in the cyst. Because it more effectively prevents subsequent attacks of cholangitis in this condition, and because of the threat of malignancy, resection of the cyst and reconstruction with a Roux-Y limb is preferred over simple bypass. During resection the surgeon must be particularly aware of a frequent anomaly: high entry of the pancreatic duct into the bile duct. If this duct is inadvertently oversewn during the closure of the distal duct, particularly severe pancreatitis may be precipitated.



Fig. 4. Sphincteroplasty. The common wall between the bile duct and duodenum is opened from the ampulla going proximally (done from inside the duodenum after opening the duodenum opposite the ampulla). Then, the duodenum is sewn to the distal common bile duct as shown, creating essentially a side-to-side anastomosis that leaves a much larger opening.

CHOICE OF BILIARY-ENTERIC ANASTOMOSIS

Once a decision has been made to proceed with surgical relief of the obstructed bile duct, the choice of the appropriate operative procedure revolves around whether a resection is being done and the natural history of the problem causing the stricture. In this regard, the ultimate fate of the duodenum and lower bile duct figures prominently. By and large, resections of the bile duct are reconstructed with a Roux-en-Y cholehepaticojejunostomy, or a hepaticojejunostomy if the anastomosis is up in the liver. Although it seems logical to bypass all benign strictures, a stricture situated high in the bile duct may be best handled by resection. The anastomosis is then performed in normal tissue above the scar. The reason is that a resection sometimes provides better exposure of the structures the surgeon wishes to preserve (portal vein and hepatic artery) while seeking more normal bile duct tissue. Conversely, benign strictures in the distal bile duct are often most expeditiously dealt with by bypassing them because normal tissue above the stricture is usually easily accessible. If the process causing a distal stricture is not expected to obstruct the duodenum (e.g., pancreatitis or multiple common bile duct stones), a choledochoduodenostomy is a very reasonable choice. If the stricture is at the ampulla, for example from an impacted gallstone, a sphincteroplasty is often done. Although this is not technically a "bypass," it does involve anastomosing a section of bile duct (the slitopen intraduodenal portion) with the duodenum (Fig. 4). An older literature suggested that sphincteroplasty, with mucosa to mucosa approximation by suture, had a lower restricture rate than an open sphincterotomy (4).

Malignant obstructions are resected when appropriate and bypassed when that should not be done. Resections are appropriate when the therapeutic aim is to try to cure the patient of their cancer and the patient can tolerate that magnitude of operative insult, or when circumstances paradoxically suggest that resection would be the least morbid way to deal with the patient's problem. The only real opportunity to surgically bypass a malignant obstruction at the bifurcation of the bile duct is at the base of the round ligament where the ducts to segments 2 and 3 may be exposed at a little distance from the hepatic hilum. Malignant obstruction of the distal bile duct is typically bypassed with either a Roux-Y choledochojejunostomy or a cholecystojejunostomy, which may be done as a loop and is an easier operation to perform. Which of these two is the best palliative option for patients with an irresectable cancer at the head of the pancreas has been a longstanding surgical controversy. Whereas each operation has its proponents, all agree that a normal gallbladder with a patent cystic duct that does not enter the common bile duct too low down—near where the cancer is obstructing—is a prerequisite for successful cholecystojejunostomy. There has been a recent resurgence in interest in the loop cholecystojejunostomy because this operation can be accomplished laparoscopically relatively easily. As a practical matter, the usual consideration is whether placement of a stent across the malignant obstruction by endoscopic or trans hepatic percutaneous techniques offers the same degree of palliation with less overall morbidity than a surgical bypass. The smaller a stent and the longer it is in place, the more likely is a patient to experience stent occlusion and repetitive episodes of cholangitis. Consequently, surgical bypass in these situations becomes more attractive as the life expectancy of the patient increases beyond 6 mo (5).

COMPLICATIONS OF BILIARY-ENTERIC ANATOMOSIS

The acute complications of these procedures are related to the magnitude of the upperabdominal operation to accomplish them (outlined in the section on liver resection) as well as bile leaks.

Bile leaks can occur not only from the anastomosis itself, but also from unappreciated ducts in the liver. This latter problem occurs almost exclusively in the setting of an acute repair of a bile duct injury when an injured segmental duct joins the injured bile duct at or below the bifurcation and is simply missed when the biliary-enteric anastomosis is performed. Most anastomotic bile leaks can be handled with the judicious use of percutaneous drainage and/or transhepatic stents. Leaks from missed ducts usually require reoperation.

Later complications of these operations revolve around progression of the disease that precipitated the need for the original operation (e.g., pancreatic cancer), complications associated with any upper abdominal operation (e.g., wound pains, hernias, adhesive bowel obstructions), and stricture of the biliary enteric anastomosis. Stricture of these anastomoses generally leads to episodes of cholangitis, and even frank obstructive jaundice. In repetitive and neglected cases this can progress to cirrhosis and portal hypertension; a development that greatly complicates subsequent therapeutic maneuvers. Although early problems with the anastomosis can presage later failure, recurrence of a benign stricture may take 10 yr to develop (6). So these patients must be followed with periodic checks of their liver function tests (particularly alkaline phosphatase) for years. Whether prolonged perioperative stenting of biliary-enteric anastomoses decreases the chance of later stricture formation is a minor surgical controversy. Although there is no definitive data, most surgeons no longer stent their anastomoses beyond the first few weeks postoperatively if they have achieved a good mucosa-to-mucosa anastomosis in relatively normal, nonsclerotic duct. Many do, however, fashion the roux limb for easy percutaneous access to the biliary tree in case that becomes necessary (Fig. 5). An early advantage of having percutaneous tubes across the biliary-enteric anastomosis is the ease of radiographic study it if things are not going well or if a leak needs to be managed.

Radiological studies of patients with biliary-enteric anastomoses will frequently show air in the biliary tree. This is often seen even after an ERCP and sphincterotomy. It is not



Fig. 5. Roux limb tip to abdominal wall after biliary-enteric anastomosis to facilitate future access to the bile ducts.

necessarily a pathologic finding. However, obstruction of the GI tract in these patients can cause abnormalities of liver function. Occasionally, a bowel obstruction will lead directly to cholangitis by virtue of the concomitant obstruction of the biliary tract. Therefore, it is prudent to consider antibiotic coverage in these patients if they do develop even a partial bowel obstruction. Episodes of cholangitis in the absence of bowel obstruction should precipitate a search for strictures in the biliary tree or at the anastomosis, or at jejunojejunostomy of the Roux limb. Unless the bypass is a spincteroplasty or a choledochoduodenostomy it may be difficult to reach the duct or the duct-enteric anastomosis with an endoscope. Usually, a percutaneous transhepatic cholangiogram of some sort must be done. Often these studies can be combined with balloon dilation of any strictures that are found; a maneuver that can either be temporizing or result in a more durable solution.

COST

The hospital charges for these sorts of procedures vary widely; depending on the underlying condition of the patient that has precipitated the need for a biliary bypass and the magnitude of the operation required to accomplish it. Professional charges for these operations (excluding pancreatectomy and hepatectomy) run in the range of \$3000 to \$6000.

SUMMARY

- 1. The biliary tree may be anastomosed to the proximal intestine to deal with all sorts of biliary obstructions, and the most common construction is with a Roux-Y jejunal limb.
- 2. Bile leak is the main early complication and can be from the anatomosis or a missed duct.
- Stricture of these reconstructions is one of the main late complications and is often heralded by cholangitis.

4. Patients with these reconstructions may also develop an element of cholangitis with subsequent distal bowel obstructions.

REFERENCES

- 1. Nagafuchi Y, Katuki M, Hisatome K, et al. A traumatic neuroma associated with obstructive jaundice after laparoscopic cholecystectomy. Hepatogastroenterology 1998;45:424–427.
- 2. Grosfeld, J. Is there a place for the Kasai procedure in biliary atresia? Curr Opin Gen Surg 1994:168–172.
- 3. Stewart BA, Hall RJ, Lilly JR. Liver transplantation and the Kasai operation in biliary atresia. J Pediatr Surg 1988;23:623–626.
- 4. Choi TK, Wong J, Lam KH, et al. Late result of sphincteroplasty in the treatment of primary cholangitis. Arch Surg 1981;116:1173–1175.
- 5. van den Bosch RP, van der Schelling GP, Klinkenbijl JH, et al. Guidelines for the application of surgery and endoprostheses in the palliation of obstructive jaundice inadvanced cancer of the pancreas. Ann Surg 1994;219:18–24.
- 6. Pitt HA, Miyamoto T, Parapatis SK, et al. Factors influencing outcome in patients with postoperative biliary strictures. Am J Surg 1982;144:14–21.

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INTRODUCTION

Cholecystectomy is one of the most frequently performed abdominal surgeries in the United States. Between 400,000 and 500,000 patients undergo the procedure annually most for problems caused by gallstones (1). The prevalence of cholelithiasis in the general population is estimated to be between 10–15% and is positively influenced by many other factors including age, female gender, family history, central obesity, rapid weight loss, distal small bowel disease, TPN, estrogen replacement therapy, and diabetes mellitus. When subsets of the population are analyzed, the prevalence is even higher. For example, 25% of Caucasian women in the United States over the age of 50 are estimated to have gallstones. Although the prevalence of cholelithiasis is high, the rate of symptom development in asymptomatic patients is relatively low at an estimated 1.5% annually (2).

A new era of gallbladder surgery began in 1987 when the first laparoscopic cholecystectomy was performed in Lyon, France. Although the treatment principles of gallbladder disease have remained unchanged, the techniques and tools used by surgeons, radiologists, and endoscopists have evolved substantially over the past 15 yr.

SURGICAL ANATOMY

The gallbladder is a hollow, pear-shaped organ located on the undersurface of the liver and externally marks a boundary between the anatomic right and left lobes of the liver

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(Cantlie's line). The gallbladder is composed of four parts-the fundus, body, neck and infundibulum. The fundus is the dome of the gallbladder and typically falls below the inferior margin of the liver. The fundus is susceptible to ischemia, necrosis, and perforation during inflammatory processes because it is far from the main body of the cystic artery and is supplied by end arteries. The body of the gallbladder is in contact with the duodenum and colon and can involve these structures during inflammatory processes. The neck of the gallbladder is significantly narrower than the body and can impact gallstones. There is frequently an outpouching of the gallbladder at the proximal portion of the gallbladder neck, which is referred to as the gallbladder infundibulum, or Hartmann Pouch. The infundibulum can overlap the cystic duct obscuring it during surgery and can impinge on the common bile duct to the point of obstruction (the often described and seldom seen "Mirizzi's syndrome") (3). The cystic artery supplies the gallbladder and typically arises from the right hepatic artery. Venous drainage does not mirror arterial supply. Instead, multiple cystic veins drain into the right branch of the portal vein and into the quadrate lobe of the liver. Lymphatics drain the gallbladder into the liver and into lymph nodes along the cystic duct. There is typically one main node located at the junction of the gallbladder and cystic duct. The extrahepatic biliary system begins with the right and left hepatic ducts that join to form the common hepatic duct. The common hepatic duct courses in the hepatoduodenal ligament for 2-4cm and joins the cystic duct forming the common bile duct. The cystic duct is typically 2-4cm long, but multiple anatomic variations exist in both its length and course to join the common hepatic duct (Fig. 1A). The common bile duct (CBD), after originating from the junction of the cystic duct and common hepatic duct, continues in the hepatoduodenal ligament anterior to the portal vein and to the right of the right hepatic artery (Fig. 1B). The CBD is typically 5-15 cm long and runs behind the first portion of the duodenum down to the pancreas, then behind or through the pancreas into the second portion of the duodenum. The proximal portion of the duodenum is shaped in a "C" loop, which allows for it to be in proximity to the CBD twice during its course. The head of the pancreas lies in the "C" loop. It is imperative for the surgeon to recognize anatomic variations of the biliary tree at the time of surgery so that injury to the common hepatic and common bile duct is avoided. Failure to correctly identify the cystic duct at the time of surgery is responsible for most of the serious morbidity following cholecystectomy. Two anatomic triangles are classically used to describe and facilitate identification of the anatomy of extrahepatic biliary tree at its junction with the gallbladder-the hepatocystic triangle and Calot's triangle. The hepatocystic triangle is formed by the margin of the liver, the common hepatic duct and the cystic duct. Calot's triangle is defined by the cystic artery, CHD, and cystic duct.

GALLBLADDER AND BILIARY IMAGING

Ultrasound and cholescintigraphy are the preferred imaging methods for the routine evaluation and diagnosis of gallbladder pathology and each offers unique advantages and limitations. Ultrasound is used most frequently for several reasons. First, the typical right upper quadrant ultrasound exam can be performed quickly and takes about 15 min for the experienced technician to complete. Second, other abdominal organs such as the liver, pancreas, kidneys, and spleen can be visualized and other sources of pain and symptoms can be diagnosed when the gallbladder is normal. Third, the possibility of other gallbladder pathology, cancer for example, may be evaluated. Finally, ultrasound can identify gallstones, thickening of the gallbladder wall, pericholecystic fluid and



Fig. 1. Anatomy around the gallbladder and hepatoduodenal ligament. (A) The structures are covered by the fat and peritoneum at initial visualization. (B) Anatomy within the hepatoduodenal ligament. (C) Anatomy after cholecystectomy.

tenderness when the ultrasound probe presses down directly over the gallbladder (sonographic Murphy sign). This constellation of ultrasound findings is highly sensitive and specific for the diagnosis of acute cholecystitis (>90%) (4). Diagnostic problems arise when only one or two of these ultrasound findings are present or the clinical presentation is atypical for acute cholecystitis. Cholescintigraphy is performed by intravenous injection of a radioactive contrast media (HIDA, DISIDA) that is rapidly taken up by the liver and excreted into bile. The flow of bile into the biliary tree, duodenum, and gallbladder can then be imaged. If bile does not enter the gallbladder but is seen entering the duodenum, cystic duct obstruction is highly likely. Because the cystic duct is usually obstructed in acute calculous cholecystitis, the scintographic finding of a nonfilling gallbladder is considered diagnostic of acute cholecystitis. This test is of limited value when the gallbladder is already filled to capacity (e.g., during prolonged starvation) or is already surgically absent. The test has poor sensitivity for the identification of chronic cholecystitis, nonobstructing gallstones or other pathology.

TYPES OF CHOLECYSTECTOMY

Cholecystectomy consists of two basic steps—dissection of the gallbladder from the liver and identification and division of the cystic duct and artery (Fig. 1C). The procedure may be performed either by an open or laparoscopic technique. In the open procedure, the abdomen is entered through a right subcostal incision and the gallbladder is dissected from the liver bed "from top down," that is from fundus to the neck. As the cystic duct and cystic artery are reached, they are both ligated and divided and the gallbladder is removed. In this fashion, the likelihood of mistaking the common bile duct for the cystic duct is minimized. During laparoscopic cholecystectomy, the abdomen is accessed through four ports—one adjacent to the umbilicus, one in the epigastrium, and two in the lateral right upper quadrant. The cystic duct and artery are identified and divided first followed by dissection of the gallbladder "from bottom up;" from the neck to the fundus.

Laparoscopy is the preferred approach for routine cholecystectomy although several relative contraindications exist. These include a failed endoscopic stone extraction/ papillotomy with known large common bile duct stones, suspected gallbladder cancer, and a history of multiple right upper quadrant surgeries. Additionally, laparoscopy is poorly tolerated unless general anesthesia is used and depends on carbon dioxide pnuemoperitoneum for exposure. Patients who are a poor general anesthesia risk or who cannot clear the carbon dioxide that is absorbed systemically should be considered for the open procedure. Some patients may not tolerate the decreased venous return to the heart caused by the increased abdominal pressure during pnuemoperitoneum unless aggressive monitoring is used to guide intraoperative patient support (5). Open cholecystectomy may be performed with regional anesthesia if the patient risks warrant this approach but general anesthesia is preferred. Laparoscopic cholecystectomy should be abandoned in favor of the open technique when the biliary anatomy is unclear or when an iatrogenic injury is suspected. Despite some of the potential drawbacks of laparoscopy, it has been clearly demonstrated to reduce postoperative pain and pulmonary dysfunction significantly when compared to open cholecystectomy (6).

In selected patients with gallbladder cancer, a radical cholecystectomy is performed. The gallbladder is resected in continuity with a underlying normal liver tissue. Lymph nodes along the celiac axis, hepatic artery, pancreas, and retroperitoneum are also removed. Because gallbladder cancer is usually detected at an advanced stage, the opportunity to cure the patient by radical cholecystectomy presents itself infrequently. In fact, curable gallbladder cancer is most commonly encountered accidentally; when the gallbladder is removed for other reasons and an unexpected cancer is discovered within the gallbladder several days later upon pathologic examination. It appears that survival in this situation is not affected by whether the gallbladder is removed by an open or laparoscopic technique (7).

INDICATIONS

Complications of gallstone disease are the most common indication for cholecystectomy. The estimated prevalence of gallstones in the US population is 15% (8). Gallstones are classified by composition and three types are recognized—cholesterol, pigment and mixed cholesterol/pigment gallstones. Cholesterol and mixed stones are more common in the US while pigmented stones are more common worldwide. Cholesterol stones form when the primary organic solutes of bile—bile salts, cholesterol and phospholipid—are in a molar ratio such that cholesterol crystals form and agglomerate. Pigmented stones contain a high concentration of bilirubin and arise in the setting of hemolytic disorders, long-term TPN and biliary infection (9).

As a generalization, only patients with symptoms or complications from their gallstones should undergo cholecystectomy. "Asymptomatic" gallstones are not treated in most patients because the rate of symptom development and serious complications are low. Only 1.4% of patients with known gallstones will develop symptoms each year (2,10). Exceptions to this rule are rare but include children and patients with sickle cell anemia. Children are believed to have a higher rate of symptom development than adults because they live longer and cholecystectomy is therefore recommended. In addition, the symptoms of gallbladder pathology can mimic those of a sickle crisis thus cholecystectomy may prevent diagnostic confusion during the evaluation of abdominal pain in a patient with sickle cell disease.

Intermittent biliary colic is the most common complication of gallstones. It occurs when the cystic duct is transiently obstructed by a gallstone, which either then disimpacts from the neck and falls back into the body of the gallbladder or passes down the extrahepatic biliary tree and into the duodenum. This produces a constant pain in the epigastrium, is often preceded by a fatty meal, and usually resolves within several hours. These patients are offered elective cholecystectomy based on history and the demonstration of gallstones by ultrasound.

The more serious complications of gallstones include acute cholecystitis, gallstone pancreatitis and ascending cholangitis. The treatment of these complications usually requires hospitalization. Acute cholecystitis occurs when the cystic duct is obstructed by a gallstone followed by bacterial overgrowth in the static bile pool of the gallbladder and gallbladder wall inflammation. Antibiotics and cholecystectomy are the preferred management. Laparoscopic cholecystectomy is more easily accomplished early in the setting of acute cholecystitis. Ideally, it should be done within 3 d. As acute inflammation and scarring progress, the likelihood that the procedure can be accomplished laparoscopically decreases. Conversion to an open operation may be necessary in as many as 20-30% patients in this circumstance (11). Early cholecystectomy also avoids the possible complication of gallbladder wall necrosis and perforation (12). Although this is not a common scenario, it should be a concern in debilitated patients and those who have symptom

progression despite antibiotics and cessation of diet. Acute cholecystitis can be managed with percutaneous gallbladder drainage under ultrasound or computed tomography (CT) guidance (13). This therapeutic option is typically reserved for high-risk patients, or those in whom the inflammation is so severe and established that it seems unlikely that cholecystectomy can be accomplished safely. Cholecystectomy can be performed at a later date when the patient is a more suitable surgical candidate or when the inflammation has subsided.

The initial goal of treatment for patients with gallstone pancreatitis and ascending cholangitis is resuscitation and stabilization, not cholecystectomy. Patients with gallstone pancreatitis usually improve with supportive care and cholecystectomy is delayed during the hospital admission until abdominal symptoms resolve and amylase/lipase levels in the blood return to normal. Cholecystectomy performed before symptom resolution carries a higher morbidity risk than when pancreatitis has settled down (14). Nevertheless, cholecystectomy during the same admission after symptom resolution is recommended since the recurrence rate of pancreatitis may be as high as 75% in the first 6 wk following discharge in patients who do not undergo cholecystectomy (15). Ascending cholangitis occurs when the biliary tree is obstructed, pressure in the biliary system rises, and bacterial and inflammatory mediators reflux into the systemic circulation through the hepatic sinusoids. Emergent drainage of the biliary tree is necessary if patients worsen despite intravenous (iv) antibiotics and fluid resuscitation. This is a lifethreatening emergency. Drainage of the biliary tree can performed either by ERCP or percutaneous transhepatic biliary cannulation. If these modalities are unavailable, the obstructed bile duct should be drained surgically with placement of a T tube. Once the sepsis resolves and the etiology of the obstruction and the biliary anatomy have been defined, a cholecystectomy may be performed.

Not all gallbladder problems are due to gallstones. Acute acalculous cholecystitis (acute cholecystitis with a patent cystic duct) is a recognized but uncommon entity. It represents approx 5% of the diagnoses of acute cholecystitis (16). Acalculous cholecystitis is a disease of intensive care units and elderly men with vascular disease. It typically has a fulminant progression to gangrenous necrosis of the gallbladder if unrecognized (17). More recently, an indolent variety of acalculous cholecystitis has been seen in HIV positive patients and may be related to Cryptosporidium and CMV infection (18). Another uncommon diagnosis unrelated to gallstones is gallbladder dyskinesia. The diagnosis is a controversial and other causes of abdominal pain must be excluded first. In the absence of other reasonable causes for the patient's complaints, a diagnosis is made when there are classic biliary colic symptoms and abnormal gallbladder emptying as measured by an ejection fraction less than 35% on cholescintigraphy. Cholecystectomy may relieve symptoms in these patients (19).

Patients who have cirrhosis and complications of gallstone disease can be difficult management problems because of the risks of cholecystectomy are increased. The mortality rate following emergent cholecystectomy in patients with advanced cirrhosis may be 24% or higher (20, 21). Child classification, elevated prothrombin time, elevated bilirubin, and ascites serve as clinical markers of increased risk. Percutaneous cholecystostomy may be considered for the treatment of acute cholecystitis in these patients. If a cholecystectomy is undertaken, subtotal cholecystectomy may be a way out of a difficult situation. In this operation the posterior gallbladder wall is left attached to the liver and the cystic duct is closed from within the gallbladder neck with a suture. Although messy,

this approach may decrease bleeding from the liver bed and avoids the potentially hazardous task of identifying the cystic duct in an inflammatory mass.

CONTRAINDICATIONS

As aforementioned, cirrhosis is a relative contraindication to cholecystectomy because bleeding from the diseased liver parenchyma as the gallbladder is dissected can be difficult to control. This bleeding problem is compounded by portal venous hypertension and coagulation abnormalities from reduced liver synthetic function. In the event that bleeding from the gallbladder fossa cannot be satisfactorily controlled, the only option may be to decrease portal hypertension with a portal-systemic shunt of some sort (usually a TIPS).

Surgical treatment of biliary colic in pregnant patients is usually deferred until the postpartum period unless symptoms are too severe or there is gestational weight loss. When cholecystectomy is to be undertaken, the second trimester is typically the preferred time. Miscarriage rates are lower in the second than the first trimester and preterm labor rates are lower in the second than third trimester. Modern series of pregnant patients with biliary pancreatitis, however, have challenged the notion that the second trimester should be the preferred time for biliary surgery or that it should carry significant maternal or fetal risk. Cholecystectomy may prove to be a safe procedure at any time during pregnancy so long as obstetric involvement is obtained early and fetal monitoring is performed (22).

EARLY COMPLICATIONS

Although, complications such as pancreatitis, or bowel perforations from cautery injuries can occur after cholecystectomy, bile leaks and bile duct obstruction are the notorious perioperative procedure-related complications. The most threatening morbidity of cholecystectomy comes from damaging the main bile duct. Patients who present with abdominal pain, fever, chills, leukocytosis, or jaundice should be evaluated for either of these complications. Liver function tests and ultrasound are chosen to determine if the etiology is caused by biliary obstruction or leak. A bile leak on ultrasound will manifest as a fluid collection in the right upper quadrant while an obstruction is diagnosed by dilated intrahepatic or extrahepatic bile ducts. Whereas in-depth management of complications following cholecystectomy is beyond the scope of this chapter, several tests and procedures are undertaken in each of these circumstances. Abdominal bile collections are drained percutaneously under radiological guidance. The location of the leak in the biliary system is defined with ERCP. If the leak is from the cystic duct stump, then papillotomy to ensure easy flow of bile through the ampulla of Vater can be performed and most cystic duct stump leaks will spontaneously close. Biliary obstruction following cholecystectomy is most often caused by a gallstone retained in the common bile duct that was unrecognized at the time of surgery. Most gallstones can be extracted from the common bile duct with ERCP. Iatrogenic injuries to the common hepatic or common bile duct recognized after cholecystectomy often require complex treatment. These injuries are best referred to tertiary centers with experience in major hepatobiliary surgery. When these serious injuries are suspected, biliary imaging and drainage can be accomplished even if definitive care is referred.

LATE COMPLICATIONS

Some patients report an increase in the number or looseness of bowel movements in the first few weeks to months after cholecystectomy. This is often mistakenly blamed on steatorrhea but is probably a result of bile salt malabsorption. Bile salt malabsorption is thought to result from the constant secretion of bile into the intestine once the reservoir function of the gallbladder is lost after cholecystectomy. In most patients, this resolves spontaneously within several months. Steatorrhea should not occur after cholecystectomy in healthy patients because the quantity of bile in the intestine remains above the minimal amount necessary for emulsification and absorption of fat.

The possibility of a relationship between increased colon cancer risk and gallstone disease has been entertained for years. It is believed that the same environmental, dietary and genetic factors that predispose to gallstones may also increase risk for colorectal cancer. The postcholecystectomy state itself, however, does not appear to change the risk of colon cancer (23).

The most frustrating problem that faces the surgeon is the patient who has undergone cholecystectomy and returns with symptoms identical to those prior to surgery. The return of episodic upper abdominal pain after cholecystectomy may indicate other underlying pathology such as peptic ulcer disease or pancreatitis, or even angina. A search for other conditions should be undertaken. There is also the possibility that the patient has common bile duct stones. These can certainly mimic the symptoms that precipitated the cholecystectomy in the first place. Common bile duct stones that develop in the bile duct after cholecystectomy are known as primary common bile duct stones. These are typically soft brown crumbly stones that occur in an abnormal, poorly emptying duct. More commonly, post cholecystectomy common bile duct stones are "retained" stones; stones that came from the gallbladder before it was removed.

When there is no explanation for the patient's biliary complaints, the problem may be described as a "postcholcystectomy syndrome." Many theories have been advanced to explain the etiology of this pain syndrome and to provide insight into treatment strategies. At present, there is no convincing evidence of a common etiology. Much work has focused on sphincter of Oddi dysfunction and biliary dysmotility. Imaging and diagnostic techniques to demonstrate these abnormalities include MRCP, ERCP, sphincter of Oddi manometry, and dynamic scintigraphy. There appears to growing evidence that certain abnormalities of the biliary tree or sphincter of Oddi could be responsible for these pain syndromes (24-26). Still, many patients with postcholecystectomy pain have no demonstrable abnormality.

Patients who have persistent pain after cholecystectomy often did not have histories consistent with symptomatic gallstone disease in the first place, even though they had gallstones on ultrasound. In such patients, there has probably been a failure of proper patient selection rather than a complication of the operation. Overall, cholecystectomy is a very reliable and safe procedure. Moreover, the addition of laparoscopy has significantly reduced postoperative pain and shortened the recovery period.

COST

Individual costs have also decreased as a result of laparoscopic techniques. Currently, the hospital charges for an uncomplicated laparoscopic cholecystectomy run in the range of \$5000–\$6000 depending on how long the patient stays at hospital. Professional charges

(surgeon, anesthesia, and consultant fees) are approx \$3600. However, the overall costs for the management of symptomatic gallstones appears to have been increased by this technology because a greater percentage of patients and physicians have been opting for surgical management than they did when open surgery was their only option (27).

SUMMARY

- 1. The rate of symptom development in asymptomatic patients with gallstones is low and no intervention is usually required. However, symptomatic patients have a high incidence of recurrant symptoms and complications and cholecystectomy is generally indicated.
- 2. Laproscopic cholecystectomy is preferred therapy in such patients. Open cholecystectomy is indicated if patient is found to have large CBD stone, which cannot be removed endoscopically, or if there is a suspicion of gallbladder cancer.
- 3. Laproscopic cholecystectomy is well tolerated and has a low incidence of early and late complications. However, elderly patients with multiple medical problems and patients with advanced cirrhosis and HIV have a higher complication rate.
- 4. Bile duct obstruction and bile leak are the most serious procedur-related complications of cholecystectomy and patients that present with abdominal pain, fever, chills, leuko-cytosis, or jaundice need evaluation for these possible complications.
- 5. Patients with acute cholecystitis who are poor surgical candidates may be treated with percutaneous cholecystostomy tube placement and gallbladder drainage.

REFERENCES

- 1. 1987 summary: National Hospital Discharge Survey. Adv Data 1988. 1-14.
- 2. Friedman GD. Natural history of asymptomatic and symptomatic gallstones. Am J Surg 1993;165:399-404.
- 3. Toscano RL, Taylor PH, Peters J, et al. Mirizzi syndrome. Am Surg 1994;60:889-891.
- Ralls PW, Colletti PM, Lapin SA, et al. Real-time sonography in suspected acute cholecystitis. Prospective evaluation of primary and secondary signs. Radiology 1985;155:767–771.
- 5. Safran D, Sgambati S, Orlando R. Laparoscopy in high-risk cardiac patients. Surg Gynecol Obstet 1993;176:548–554.
- Schauer PR, Luna J, Ghiatas AA, et al. Pulmonary function after laparoscopic cholecystectomy. Surgery 1993;114:389–397; discussion 397–399.
- 7. Whalen GF, Bird I, Tanski W, et al. Laparoscopic cholecystectomy does not demonstrably decrease survival of patients with serendipitously treated gallbladder cancer. J Am Coll Surg 2001;192:189–195.
- Diehl AK. Epidemiology and natural history of gallstone disease. Gastroenterol Clin North Am 1991; 20:1–19.
- 9. Carey MC. Pathogenesis of gallstones. Am J Surg 1993;165:410-419.
- 10. Johnston DE, Kaplan MM. Pathogenesis and treatment of gallstones. N Engl J Med 1993;328:412-421.
- 11. Fried GM, Barkun JS, Sigman HH, et al. Factors determining conversion to laparotomy in patients undergoing laparoscopic cholecystectomy. Am J Surg 1994;167:35–39; discussion 39–41.
- 12. Sharp KW. Acute cholecystitis. Surg Clin North Am 1988;68:269-279.
- 13. Boland GW, Lee MJ, Leung J, et al. Percutaneous cholecystostomy in critically ill patients: early response and final outcome in 82 patients. AJR Am J Roentgenol 1994;163:339–342.
- Schwesinger WH, Page CP, Sirinek KR, et al. Biliary pancreatitis. Operative outcome with a selective approach. Arch Surg 1991;126:836–839; discussion 839.
- Soper NJ, Brunt LM, Callery MP, et al. Role of laparoscopic cholecystectomy in the management of acute gallstone pancreatitis. Am J Surg 1994;167:42–50; discussion 50,51.
- 16. Frazee RC, Nagorney DM, Mucha P. Acute acalculous cholecystitis. Mayo Clin Proc 1989;64:163–167.
- 17. Babb RR. Acute acalculous cholecystitis. A review. J Clin Gastroenterol 1992;15:238-241.
- Keshavjee SH, Magee LA, Mullen BJ, et al. Acalculous cholecystitis associated with cytomegalo virus and sclerosing cholangitis in a patient with acquired immunodeficiency syndrome. Can J Surg 1993;36: 321–325.

- Yost F, Margenthaler J, Presti M, et al. Cholecystectomy is an effective treatment for biliary dyskinesia. Am J Surg 1999;178:462–465.
- 20. Doberneck RC, Sterling WA, Allison DC. Morbidity and mortality after operation in nonbleeding cirrhotic patients. Am J Surg 1983;146:306–309.
- 21. Bloch RS, Allaben RD, Walt AJ. Cholecystectomy in patients with cirrhosis. A surgical challenge. Arch Surg 1985;120:669–672.
- Cosenza CA, Saffari B, Jabbour N, et al. Surgical management of biliary gallstone disease during pregnancy. Am J Surg 1999;178:545–548.
- 23. Jorgensen T, Rafaelsen S. Gallstones and colorectal cancer—there is a relationship, but it is hardly due to cholecystectomy. Dis Colon Rectum 1992;35:24–28.
- 24. Chuttani R, Carr-Locke DL. (1993) Pathophysiology of the sphincter of Oddi. Surg Clin North Am 1993;73:1311–1322.
- 25. Lichtenstein GR, Dabezies MA. Biliary Tract Dysmotility. Curr Treat Options Gastroenterol 1998;1: 27–34.
- 26. Rubini G, Dimonte M. Postcholecystectomy syndrome: evaluation by biliary cholescintigraphy and MR cholangiopancreatography. Clin Nucl Med 1999;24:784–788.
- Orlando R, Russell JC, Lynch J, et al. Laparoscopic cholecystectomy. A statewide experience. The Connecticut Laparoscopic Cholecystectomy Registry. Arch Surg 1993;128:49449–8; discussion 498–499.

VI PANCREATIC SURGERY

20 Pancreatic Surgery

Janette U. Gaw, MD and Dana K. Andersen, MD

CONTENTS

INTRODUCTION PANCREATIC RESECTION PANCREATIC DECOMPRESSION PSEUDOCYST DRAINAGE REFERENCES

INTRODUCTION

Operations on the pancreas are very serious operations, for several reasons. First, the conditions for which operations are done are usually difficult medical problems. Most operations, for example, are for pancreatic tumors. Although some of these tumors can have a relatively benign natural history (neuroendocrine, cystadenomas, and cystodenocarcinoma), ductal adenocarcinoma of the pancreas, the most common tumor, is difficult to cure with even the best surgery, and multimodality therapy. Operations are also done for significant abdominal trauma that disrupts the pancreas, and for complications of either severe or intractable pancreatitis.

Second, operations on the pancreas usually involve the manipulation and dissection of many anatomic structures deep in the back of the upper abdomen. They are, therefore, often "large operations" from the standpoint of physiologic insult.

Finally, there is always the specter of pancreas-specific surgical complications. These are the result of leaks from the pancreatic duct, from enteric anastomoses to the pancreatic duct, or from pancreatitis stimulated by the surgery. These complications are the primary source of concern in the early postoperative period, and require experienced teams to deal with them. Late complications are usually the result of pancreatic exocrine, and endocrine insufficiency caused by the underlying conditions, the operation, or both.

Pancreatic surgery may be broadly categorized as either pancreatic resection or pancreatic drainage or both. This chapter discusses some of the common operations performed on the pancreas, indications, contraindications, as well as short- and longterm complications of these operations.

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PANCREATIC RESECTION

Proximal Pancreaticoduoenectomy (Whipple Procedure)

Although pancreatic resection has been promoted by several surgeons in the last 60 yr, it was Kausch in Germany and Allen Whipple in the United States who successfully performed, and popularized the surgery for periampullary, and pancreatic head carcinoma in 1909 and 1935, respectively. Since then, it has become the standard operation for carcinoma of head of pancreas. Pancreaticoduodenectomy and "Whipple procedure" are used synonymously, however, it is far more extensive surgery than the name pancreaticoduodenectomy because it involves resection of the head, the neck and uncinate process of the pancreas; the distal bile duct and gall bladder; distal stomach, the entire duodenum and proximal jejunum, followed by reconstruction (Fig. 1A). The reconstruction generally consists of a pancreaticojejunostomy as the most proximal anastomosis on the jejunal limb, followed by a choledochojejunostomy, and last, a gastrojejunostomy. A variation of the Whipple procedure is the pyloric sparing Whipple procedure in which the distal stomach is preserved to maintain normal gastric physiology.

Indications and Contraindications

The most common indication for a pancreaticoduodenectomy is resection of a tumor of the head, neck, or uncinate process of the pancreas or periampullary tumors. Another indication is chronic pancreatitis with imflammatory changes localized to the head of the pancreas. Preoperative evaluation includes imaging for diagnosis, as well as staging and determination of resectability.

The most common imaging modalities used include transabdominal ultrasound, endoscopic ultrasound, computed tomography (CT) scan, magnetic resonance imaging (MRI), and endoscopic retrograde cholargiopancreatography ERCP. CT scan is useful both for the diagnosis and staging of pancreatic cancer. Endoscopic ultrasound is most sensitive for evaluation of the local extent of the primary tumor.

Metastatic disease to the liver, peritoneum, and local invasion are contraindications for a pancreaticoduodenctomy. However, various centers have different levels of aggressiveness regarding portomesenteric vessel invasion and resection.

Another important aspect of preoperative evaluation is the determination of a patient's ability to survive the procedure. Debilitating acute or chronic diseases make the risk of surgery unacceptable.

STANDARD WHIPPLE PROCEDURE

The procedure begins with an exploration of the abdomen for evidence of metastatic disease or advanced local disease. This exploration may be initiated with a laparoscope. The peritoneal cavity is inspected for ascitic fluid and peritoneal implantation of metastasis. The liver surface is inspected. Following this, the extent of the tumor is determined by mobilizing the head of the pancreas and the duodenum. The lesser sac is entered, and the body and tail of the pancreas are examined, as well as the area of the superior mesenteric vein, which passes under the neck of the pancreas. Invasion of adjacent organs, superior mesenteric vessels, or portal vein is a contraindication for resection in most centers. Once a determination has been made to resect the tumor, the stomach is divided approx 5–7 cm from the pylorus. The biliary tract is then divided at the common hepatic duct level. A cholecystectomy is performed. Next, the pancreas



Fig. 1. Whipple procedure. (A) Standard Whipple procedure. (B) Pylorus sparing Whipple procedure.

is transected at the level of the superior mesenteric vein. Transection of the jejunum 10–15 cm distal to the ligament of Treitz completes the resection. Reconstruction is achieved by bringing the jejunal limb either anterior to the transverse colon (antecolic) or through the mesocolon (retrocolic). A pancreatico-jejunostomy is performed first, followed by a choledacojejunostomy. Finally, a gastrojejunostomy is performed.

Given that most complications arise from the pancreaticojejunostomy, several different techniques have been described. As an alternative to the pancreaticojejunostomy reconstruction, some surgeons perform a pancreaticogastrostomy. Both techniques yield similar results.

Pylorus Sparing Whipple Procedure

A modification of the standard Whipple procedure involves preservation of the pylorus and the proximal 2 cm of the duodenum (Fig. 1B). The advantages over the classical Whipple procedure include preservation of the stomach reservoir and the pylorus, and theoretically, maintaining a more normal gastric emptying, and hormonal control. The reconstruction is similar to the classical pancreaticoduodenctomy except that instead of a gastrojejunostomy, a duodenojejunostomy is performed. The theoretical disadvantage is the inadequacy of margins during a resection for cancer. Randomized trials of the pylorus-sparing and standard Whipple procedure have failed to reveal any differences in outcome or morbidity.

Complications

The postoperative mortality rate is about 2-5%. The morbidity rate is about 20-50% (1-3). Advanced age is no longer a contraindication, and recent series have increasingly included octogenarians. Complications not specific to the procedure include cardiopulmonary events, postoperative bleeding, and infectious complications. Because of the complex nature of the resection and reconstruction, complications can originate from

each of the different anastomoses. Leakage from the choledochojejunal anastomosis occurs in 5-8% of patients, but is usually managed nonoperatively as most of these resolve spontaneously. The pancreaticojejunal anastomosis leak presents a more challenging problem. The incidence of pancreatic fistula is reported to be between 3-20%, and can result in postoperative death caused by infection and bowel injury. A reoperation can be prevented if there is adequate drainage of the leak. Supportive care is initiated with hyperalimentation and administration of octreotide.

Another problem seen postoperatively is delayed gastric emptying. This has been postulated to be secondary to a deficit in an enteric hormone, motilin, in pancreatic cancer. Erythromycin, a drug with motilin-like activity, has been recommended to manage this problem.

Late complications of the procedure include alkaline reflux gastritis, marginal ulcers, dumping syndrome, gastric outlet obstruction, and pancreatic fistula.

Cost

Average hospital cost is between 20,000 and 72,000 (4–7). It has been shown that centers that perform pancreaticoduodenectomies more frequently incur a lower hospital cost, shorter stay, and lower morbidity.

Summary

- 1. The reconstruction involves three anastomoses: pancreaticojejunostomy, gastrojejunostomy, and choledochojejunostomy. Leakage from these anastomoses is associated with significant morbidity and mortality.
- 2. Whipple's procedure is indicated for pancreatic carcinoma and other malignancies of the pancreas as well as benign diseases such as chronic pancreatitis.
- 3. Although the morbidity and mortality is low with advancement in the technical and supportive care for the critically ill patients, the 5-yr survival rate for carcinoma head of pancreas is still low as the diagnosis is often delayed.

Distal Pancreatectomy

Distal pancreatectomy involves resection of the body and tail of the pancreas (Fig. 2). It is a less-morbid procedure than proximal pancreatic resection and is performed for either a benign or a malignant lesion of the body and tail of the pancreas.

Indications and Contraindications

Distal pancreatectomy is indicated for lesions at the body and tail of the pancreas. If the lesion is malignant, it must be ascertained whether or not the lesion is metastatic. In addition, a locally advanced lesion usually precludes a resection. This includes invasion into the duodenum, involvement of the celiac axis, common hepatic artery or portal vein.

Adenocarcinoma of the pancreas occurs less frequently at the tail of the pancreas, however, most of the tumors are large and advanced at diagnosis and less likely to be resectable, as they do not cause any obstructive symptoms. Cystic lesions and islet cell tumors of the body and tail of the pancreas are more typically amenable to distal resection.

Distal pancreatectomy may also be indicated for chronic pancreatitis in selected cases where the disease is clearly limited to the body and tail. This may result following abdominal trauma with partial disruption of the pancreatic duct at the neck.



Fig. 2. Distal pancreatectomy. (**A**) Abdominal exploration for resectability. (**B**) Splenic mobilization and ligation of splenic artery and vein. (**C**) Pancreatic resection and pancreatic stump closure. (**D**) Step 4. Splenic sparing distal pancreatectomy.

Management of a pseudocyst at the tail of the pancreas may also involve a distal pancreatectomy. This is done when the cyst is small, and lies farther to the left. The advantage of a resection includes the removal of the diseased gland associated with the pseudocyst. However, a pseudocyst that is large or that is located fairly midline is more amenable to a drainage procedure. A cyst that is suspicious for a cystic neoplasm should be resected.

Trauma to the pancreas that involves the body and tail can also be managed with a distal pancreatectomy.

Distal pancreatectomy may be performed with en-bloc splenectomy or without splenectomy depending upon the involvement of the spleen and the nature of the disease (benign vs neoplastic).

EN-BLOC SPLENECTOMY

The abdomen is first explored for any evidence of metastatic disease. Also, the local extent of the tumor is determined. Invasion of the duodenum or encasement of the major vessels precludes resection (Fig. 2A). Once it is determined the resection is possible, the splenic artery and vein, and the short gastric vessels are ligated, and the spleen is mobilized from the retroperitoneum (Fig. 2B). This facilitates the mobilization of the tail of the pancreas. The pancreas is then divided at the neck and the margin is sent for a frozen section. If the margins still contain tumor, further resection is performed. The pancreatic

stump is then closed using either sutures or a stapler (Fig. 2C). No anastomosis is performed if the proximal pancreatic duct is patent.

SPLENIC-SPARING DISTAL PANCREATECTOMY

The spleen may be preserved if the procedure is performed for benign disease (Fig. 2D). A spleen-sparing distal pancreatectomy may be performed laparoscopically or by an open technique. The procedure requires dissecting the distal pancreas from the splenic vessels, which may prove to be difficult. Conservation of the spleen may not be possible because of the bleeding that may be encountered from the splenic vessels.

Complications

The mortality of the procedure is low, 0-5% (8–10). The complications include hemorrhage, infection, and pancreatic fistula, which occur in about 5% of the cases (11). Late complications are secondary to the endocrine and exocrine insufficiency, however, risk of diabetes is less than for proximal pancreatectomy.

Summary

- 1. Distal pancreatectomy involves resection of the body and tail of the pancreas and may be performed with or without splenectomy.
- 2. It is well tolerated and has a low mortality and morbidity rate.
- 3. The spleen may be preserved in surgery for benign diseases.

Total/Subtotal Pancreatectomy

The first total pancreatectomy was performed in the 1940s although a near total pancreatectomy is attributed to Billroth in 1884. It was advocated in the 1950s because of the belief that pancreatic cancer is a multicentric disease, and a curative resection requires a total pancreatectomy. It also includes a more extensive lymphadenectomy which theorectically decreases the risk of local recurrence. In addition, problems with the pancreaticojejunal anastomosis are eliminated, but with the added cost of significant metabolic disorders, exocrine insufficiency, and diabetes in 100% of cases.

A total pancreatectomy involves removal of the entire gland, the duodenum, distal stomach, distal bile duct, spleen, and the greater omentum (Fig. 3). This procedure was largely abandoned after a high mortality rate was observed both early and late. The metabolic changes that ensue are also challenging to control. As many as 50% of all of the late deaths that occur after total pancreatectomy are a result of "iatrogenic hypoglycemia." Moreover, a survival benefit over the Whipple procedure has not been demonstrated for similar stage tumors of the proximal pancreas. Hence, the indication for a total pancreatectomy currently is the finding of carcinoma in the margin of a proximal pancreatectomy in a patient who can tolerate the metabolic demands of a complete resection.

Indications and Contraindications

Total pancreatectomy may be indicated in cases where there is obvious tumor along the main pancreatic duct, and disease-free margins cannot be obtained. Rarely, a giant cystadenocarcinoma or sarcoma extends along the whole gland, and requires a complete resection. It may also be performed if the pancreatic remnant is friable, and will not hold sutures for a safe reconstruction. Total pancreatectomy may also be required for diffuse



Fig. 3. Total pancreatecctomy. (A) Mobilization of pancreas and duodenum with antrectomy. (B) Cholecystectomy, transection of common hepatic duct, jejunum and splenic resection. (C) Reconstruction with choledochojejunostomy and gastrojejunostomy.

intraductal papillary mucinous tumor of the pancreatic duct. Additionally, it may be used to control a postoperative pancreaticojejunostomy leak.

Total pancreatectomy may also be considered for symptomatic chronic pancreatitis, which is refractory to medical therapy. Because the latter is a benign disease, a modification (near total or subtotal pancreatectomy) is advocated by many surgeons, which involves preserving the duodenum and the spleen. However, a subtotal pancreatectomy should not be the procedure of choice if the duodenum or the distal common bile duct are involved in the inflammatory process. As will be discussed later, different types of parenchyma-preserving surgery are performed for chronic pancreatitis. However, total pancreatectomy may be indicated in cases where a partial pancreatectomy has failed. In addition, some surgeons recommend a total pancreatectomy in patients with diffuse parenchymal disease who already have pancreatic endocrine and exocrine insufficiency. At some centers, total or subtotal pancreatectomy may be combined with autologous islet transfusion to prevent diabetes.

Procedure

For total pancreatectomy, the resectability of the tumor is first determined after the peritoneal cavity is entered. The duodenum and head of the pancreas are mobilized using the Kocher maneuver as described for the Whipple procedure. An antrectomy is performed (Fig. 3A), or the pylorus is preserved, as for the Whipple procedure. A cholecys-

tectomy is also performed, and the common hepatic duct is transected. The jejunum is transected distal to the ligament of Treitz. The spleen is mobilized, and is included in the enbloc resection (Fig. 3B). Reconstruction involves a choledochojejunostomy and either a gastrojejunostomy or a duodeuojejunectomy if the pylorus is preserved (Fig. 3C).

A subtotal pancreatectomy involves resection of 95% of the pancreas, and preserves the duodenum (Fig. 4). The tail and body of the pancreas are removed as described in the Distal Pancreatectomy section. As the dissection approaches the ampullary region, the common bile duct is identified, and preserved. The pancreatic duct at the ampulla is divided and oversewn.

Complications

Historically, total pancreatectomy carries a high mortality, in the range of 25%. More recently, the mortality in experienced hands has been reported at about 2-5% (1-5,12,13). The morbidity is reported to be around 30-50% (12-14). Intraoperative and postoperative hemorrhage is a common complication with total pancreatectomy although the incidence is reduced if performed by an experienced surgeon. Patients can also develop infection complications from an intraabdominal source. Anastomotic leaks from the gastrojejunostomy or choledochojejunostomy can also occur. Other complications include cardiopulmonary complications, delayed gastric emptying, gastrointestinal bleeding, and "brittle" diabetes.

A large part of the morbidity from total pancreatectomy, however, arises from the metabolic derangements that result from the procedure. Both hyper- and hypoglycemia can be life threatening. During the immediate postoperative period, patients will benefit from frequent blood glucose measurements, and an insulin drip for tight control of the blood sugar. Sliding-scale doses of insulin are discouraged initially because the response to insulin administration can be erratic.

Patients will develop malabsorption and steatorrhea after total pancreatectomy, and will require pancreatic enzyme replacement with each meal and snack. After several months, the requirement should stabilize, although continued surveillance for the maintenance of body weight and evidence of hypoglycemia is mandatory.

Long-term management of diabetes can be particularly difficult. Hypoglycemic episodes can be frequent as a result of enhanced peripheral sensitivity to insulin, and the loss of pancreatic glucagon. However, ketogenic episodes are rare. Patients should be instructed to eat frequent small meals regularly. They should also carry snacks with them in case any symptoms of hypoglycemia should occur.

Summary

- 1. Total pancreatectomy has a historically high mortality and morbidity.
- 2. Even though the mortality has improved, the physiologic derangements that follow make it a highly morbid procedure.
- 3. At this time, there are limited indications for total pancratectomy. These include a diffuse malignancy, or as a last resort, for chronic pancreatitis. When performed for benign disease, it may be combined with autologous islet transplantation.

Duodenum-Sparing Proximal Pancreatic Resection

Up to one-third of patients with chronic pancreatitis can develop an inflammatory mass predominantly at the head of the pancreas. The pancreatic head becomes enlarged,



Fig. 4. Subtotal pancreatectomy.

and develops parenchymal calcifications, ductal calculi, and necrosis. When resection is considered, some centers advocate a pancreaticoduodenectomy. However, given that chronic pancreatitis is a non-malignant disease, a Whipple procedure may be excessive. The duodenum-sparing pancreatic head resection spares the stomach, duodenum, and the biliary tree, and results in decreased morbidity and mortality, but allows removal of the central portion of the diseased pancreatic head.

Indications and Contraindications

The most common indication for a duodenum-sparing pancreatic head resection is intractable pain caused by chronic pancreatitis. However, this operation is also performed for premalignant ductal, cystic, or solid lesions of the head of the pancreas, or for endocrine tumors of the pancreatic head. Several variations of this operation are discussed here.

DUODENUM-PRESERVING PANCREATIC HEAD RESECTION (BEGER PROCEDURE)

First described by Hans Beger in 1972, the procedure involves exposure of the pancreatic head, and mobilization of the pancreatic neck. The pancreas is transected at the level of the neck, and a subtotal resection of the pancreatic head is performed (Fig. 5A). The uncinate process may be included in the resection, and the intrapancreatic common bile duct is exposed and preserved. A small rim of pancreas is left on the duodenum, as well as the posterior branch of the gastroduodenal artery to preserve the blood supply of the duodenum. Reconstruction involves one or two Roux-en-Y pancreaticojejunal anastomoses, one to the neck of the pancreas, and usually another to the rim of pancreatic head (Fig. 5B). In cases where there is a stenosis of the common bile duct, a choledochojejunostomy can also be performed. If the main pancreatic duct is dilated and multiple stenosis exists, then a longitudinal decompression with a side-to-side anastomosis is also performed.

Complications

The perioperative mortality is reported to be 1% in a large series (15). Early complications include bleeding, anastomotic leakage, and abscess. Ischemia of the duodenum



Fig. 5. Beger procedure. (A) Pancreatic head resection. (B) Reconstruction with Roux-en-Y pancreaticojejunal anastomosis.

may occur as well, and the rate for reexploration is about 5%. Late mortality after a median of 6-yr follow-up was 9%, when performed for chronic pancreatitis.

A Beger procedure results in the removal of about 20% of the pancreatic mass. Only 2% of patients had a deterioration of their diabetes and 9% had some improvement compared to preoperative glucose tolerance tests.

LONGITUDINAL PANCREATICOJEJUNOSTOMY WITH EXCAVATION OF THE PANCREATIC HEAD (FREY PROCEDURE)

Another duodenum-sparing approach is the Frey procedure, described in 1987. There are two versions of this procedure. One involves mostly a decompressive-type procedure, which resects the pancreatic parenchyma overlying the pancreatic duct in the head of the pancreas. The other involves excavation of the head of the pancreas, and resecting the main duct and its branches while leaving a rim of pancreatic tissue posteriorly (Fig. 6A,B). This differs from the Beger procedure in that the pancreas is not transected at the neck, and only a single side-to-side pancreaticojejunostomy is required for reconstruction (Fig. 6C).

Complications

The postoperative mortality was less than 2% (16). The late mortality was reported to be 10% (17), owing to progression of disease, and complications of chronic pancreatitis. Progression of diabetes occurred in 11%. However, complete pain relief was reported at 75%, improvement in 13%, and no improvement in another 13%. Because less pancreatic parenchyma is removed than for a Beger or Whipple procedure, the Frey procedure has a low incidence of new postoperative pancreatic insufficiency.

Physiological Changes

The physiological changes that occur after duodenum-sparing proximal pancreatectomy are directly related to the amount of pancreatic tissue resected, and the amount of functional pancreatic mass remaining. The percent of resection is estimated by the superior mesenteric vessels, which divides the pancreatic mass into two equal halves.



Fig. 6. Frey procedure. (A) Excavation of head of pancreas. (B) Creation of enterostomy. (C) Side-toside pancreaticojejunostomy.

Usually, patients with normal pancreatic function preoperatively can tolerate an 80% pancreatic resection without significant physiological changes. On the other hand, patients with diffuse parenchymal disease as seen with chronic pancreatitis may not tolerate a 50% resection metabolic deficiency (19).

The metabolic changes that occur are a result of exocrine, and endocrine insufficiency. Total pancreatectomy results in the most extreme insult. Patients will develop malabsorption and steatorrhea unless maintained on adequate enzyme replacement therapy.

The diabetes that develops after total pancreatectomy is particularly difficult to manage, however. Patients are exquisitely sensitive to insulin because of enhanced peripheral insulin sensitivity. Hypoglycemic episodes can be frequent, secondary to the lack of glucagon, and the counter-regulation it provides for a fall in blood glucose. Fasting and postprandial hyperglycemia is common because of unsuppressed hepatic glucose production. The paradox of hepatic resistance to insulin, and enhanced peripheral sensitivity to insulin causes difficulty in the management of postoperative diabetes. The duodenum-preserving pancreatic head resections have a lower incident of postoperative diabetes, and may actually result in improved glucose tolerance. This observation sug-
gests that the preservation of the duodenum, and a small amount of the pancreatic head may have a profound benefit on the postoperative course of the patient.

Alternative Treatments

Pancreatic resection is the only treatment modality which offers the possibility for cure in pancreatic cancer. However, distant metastases, and advanced local disease are contraindications to resection. Also, resection should be avoided in patients with acute or chronic diseases that may make the risk of surgery and anesthesia prohibitive. Tissue diagnosis may be obtained in these cases through percutaneous methods, by ERCP, or EUS. Biliary and duodenal obstruction may be treated with either surgical bypass or with endoscopically placed stents.

In contrast, chronic pancreatitis is primarily a medically managed disease. Surgery is indicated when medical treatment fails or when endoscopic methods are unsuccessful in the treatment of an obstructed pancreatic duct. Medical treatment of chronic pancreatitis includes pain management, and patients are encouraged to abstain from alcohol. Not only does this remove the cause of chronic pancreatitis, but alcohol is also a secretagog, and can stimulate an already compromised organ. In order to avoid overstimulating the pancreas, small meals containing low amounts of fat and protein are advised. To further rest the pancreas, some have prescribed acid-suppressing agents, pancreatic enzymes, and octreotide, a somatostatin analog. Although these measures make physiological sense, they have not been definitively proven to be of benefit in the long-term treatment of chronic pancreatitis.

Pain management includes analgesics as well as analgesia-enhancing drugs. NSAIDS and acetaminophen are first used. However, narcotics are usually required for adequate pain control. Celiac plexus block is effective in pancreatic cancer, but is not as effective in chronic pancreatitis because of the reluctance to use permanent neurolytic agents. Procedure-related complications include transient hypotension, nerve root pain, and focal neuropathic damage.

Endoscopic treatment of chronic pancreatitis is also possible. Strictures in the main pancreatic duct may be amenable to pancreatic duct stenting, with an efficacy of 66% reported in some series (20–26). However, this is also associated with its own set of complications including cholangitis, hemobilia, stent occlusion, stent migration, intraductal infection, duodenal erosions, and ductal perforation. Long-term complications include morphologic changes of the pancreatic duct, which can lead to strictures. Stents also need to be replaced, and therefore, do not provide long-term symptomatic relief that a surgical drainage procedure can provide.

Pancreatic ductal stones may also be removed endoscopically. This technique is best when the stones are small and limited to the pancreatic head. Impacted stones may be fragmented first by lithotripsy.

Endoscopic therapy is, therefore, an acceptable short-term treatment of symptoms from chronic pancreatitis. It may be appropriate therapy for patients who are high surgical risks, but further studies are needed to compare medical, endoscopic, and surgical treatment of chronic pancreatitis.

Cost

The cost of duodenal-preserving pancreatic head resection in one series was 23,000 + 16,500. The disease-specific hospital cost decreased after surgery by 57% (18). This

is attributed to reduced pain score and hospital admission rate. Also, the occupational rehabilitation rate is between 68 and 75% (16,18).

Summary

- 1. Duodenal-sparing pancreatic head resection as described by Beger and Frey are very welltolerated procedures with lower morbidity and mortality than the Whipple procedure.
- 2. When compared to a pancreaticoduodenectomy, they appear to be equal in efficacy for providing long-term relief and may result in less pancreatic insufficiency.

PANCREATIC DECOMPRESSION

Pancreaticojejunostomy

LONGITUDINAL (SIDE-TO-SIDE) PANCREATICOJEJUNOSTOMY (PUESTOW PROCEDURE)

Pain in chronic pancreatitis may be caused by obstruction and dilatation of the pancreatic duct. Early surgical approaches developed for decompression included biliary sphincterotomy, and caudal drainage of the pancreas to a loop of jejunum (Duval procedure). However, because multiple strictures and dilatation of the pancreatic duct occurs throughout the ductal system, Puestow advocated a method for wider decompression in 1960. This involved opening the pancreatic duct from the neck of the pancreas to the tail. The entire distal pancreas was then invaginated into a jejunal loop for enteric drainage of the distal gland. This approach was modified by Partington and Rochelle who performed a side-to-side, Roux-en-Y, pancreaticojejunostomy (Fig. 7).

The advantage of this procedure, still known as a Puestow procedure, is that there is no removal of pancreatic parenchyma and, therefore, no risk of additional endocrine or exocrine insufficiency. However, this procedure can only be performed if dilated ducts are present. Long-term follow-up studies show pain improvement in 70–80% of the patients (27-32). A decompression procedure prevents or delays the progression of pancreatic insufficiency when compared to medically treated obstructive chronic pancreatitis (33).

Procedure

The procedure begins with an exploration of the abdomen to rule out a malignancy. The pancreatic duct is then located by palpation and confirmed by needle aspiration of pancreatic fluid. An intraoperative ultrasound may also be used for pancreatic duct localization. Following this, the pancreatic duct is then splayed open from the pancreatic tail to as close to the entry into the head as possible (Fig. 7A). All ductal stones are removed. A jejunal limb is anastomosed to the open pancreatic duct (Fig. 7B) and bowel continuity is re-established in a Roux-en-Y fashion (Fig. 7C).

Complications

The Puestow procedure has a reported mortality rate of 4%, and a complication rate between 10-15% (27–33). Because pancreatic parenchyma is preserved, endocrine and exocrine insufficiency is not exacerbated. Despite the fact that a longitudinal pancreaticojejunostomy is a safe procedure, long-term mortality remains high with a 5-yr survival as low as 40%. This is attributed to continued alcoholism, and comorbid conditions. Recurrent inflammatory changes occur in 15–20% of patients, as a result of obstruction and persistent disease in the pancreatic head.



Fig. 7. Puestow procedure. (A) Opening of pancreatic duct. (B) Anastomosis of pancreatic duct to jejunal limd. (C) Roux-en-Y pancreaticojejunostomy.

LONGITUDINAL PANCREATICOJEJUNOSTOMY WITH EXCAVATION OF THE PANCREATIC HEAD (FREY PROCEDURE)

As the pancreatic duct dives posteriorly into the head of the gland, adequate decompression with a longitudinal pancreaticojejunostomy alone is difficult. The Frey procedure, with excavation of the proximal gland, is used especially in cases where the pancreatic head is enlarged as seen in most cases of chronic pancreatitis.

Procedure

The Frey procedure is a modification of the longitudinal pancreaticojejunostomy procedure, where a duodenum-preserving excavation of the head of the pancreas is also performed. The tissue overlying the ducts of Wirsung and Santorini in the head is resected, and the duct to the uncinate process is resected or opened along its axis. A side-to-side pancreaticojejunostomy with a Roux-en-Y loop of jejunum is performed similar to the Puestow reconstruction (Fig. 6).

Complications

The late mortality is reported to be 10% and progression of diabetes occurred in 11% (17).

Roux-en-Y (Side-to-End) Pancreaticojejunostomy

A Roux-en-Y (side-to-end) pancreaticojejunostomy is used for internal drainage of pancreatic duct leaks that may result from trauma, surgery, or acute pancreatitis. Pancreatic leaks that occur at the body and tail of the pancreas may be treated with a distal pancreatectomy. However, a leak from the pancreatic duct in a chronically inflamed pancreas may be more safely managed with a Roux-en-Y pancreaticojejunostomy because resection of the pancreas in this setting carries a higher risk of morbidity.

The procedure involves creating a Roux-en-Y limb of jejunum, and suturing it to the area of injury or leak on the pancreatic capsule, so as to provide internal (enteric) drainage of the ductal secretions.

Cost

The cost of longitudinal pancreaticojejunostomy is highly variable because of the confounding problems aforementioned. The reported cost in the literature is \$24,000 (27).

Summary

A longitudinal pancreaticojejunostomy is a safe procedure for chronic pancreatitis with low morbidity and mortality in the immediate postoperative period. However, the long-term quality of life may be diminished by alcoholic recidivism or by ongoing pancreatic insufficiency.

PSEUDOCYST DRAINAGE

A pancreatic pseudocyst is a fluid collection within or adjacent to the pancreas with a surrounding wall of fibrous tissue lacking an epithelial lining. Pseudocysts may occur after pancreatitis or pancreatic trauma. They may remain asymptomatic or may cause pain. They may also cause symptoms from gastric or duodenal compression such as early satiety, nausea, and vomiting. Compression of the biliary system may lead to obstructive jaundice. Portal hypertension can result from thrombosis of the splenic vein owing to pseudocyst compression. Additionally, pancreatic pseudocysts may cause hemorrhage either from the inflammatory pseudocyst wall or from erosion of the pseudocyst rupture.

Indications and Contraindications

The indications for surgery are somewhat controversial. Surgical drainage is the preferred method for all symptomatic pseudocysts larger than 5 cm, which are not amenable to endoscopic, transgastric drainage. Surgery probably is the treatment of choice for recurrent pseudocysts, pseudocysts associated with common bile duct stenosis or duodenal stenosis, pseudocysts that penetrate through the transverse mesocolon or extend into the mediastinum or lower abdomen, and for cystic lesions where a cystic neoplasm cannot be ruled out.

There are four techniques described for surgical drainage of a pseudocyst: external drainage, cystogastrostomy, cystoduodenostomy, and cystojejunostomy. The choice of

technique is based on the anatomical position of the pseudocyst. The pseudocyst is generally drained into the segment of the gastrointestinal tract to which it is densely adherent, or by means of a Roux-en-Y limb of jejunum.

EXTERNAL DRAINAGE

External drainage of a pseudocyst involves placing a large bore catheter into the pseudocyst cavity, and draining it out through the skin (Fig. 8). External drainage of a pseudocyst is not generally the treatment of choice during an open procedure. It is associated with a mortality rate of 10%, and a recurrence rate of 18% (34). The risks include hemorrhage from abrasion of the drainage tube, development of a pancreatic fistula, and secondary infection. It is only used when the surgeon finds that the pseudocyst is frankly infected or thin-walled. Either of these would make an anastomosis at risk for dehiscence.

Cystogastrostomy

Cystogastrostomy is utilized when the pseudocyst is adjacent to the posterior gastric wall. It is best used when the pseudocyst is already adhered to the stomach, otherwise, most surgeons would recommend a cystojejunostomy instead. Splenic vein obstruction is also relative contraindication to this procedure because it predisposes to postoperative bleeding.

Cystogastrostomy is performed by making an incision in the anterior wall of the stomach (Fig. 9A). An opening is then made on the combined posterior gastric, and pseudocyst wall (Fig. 9B,C) and gastrostomy is closed (Fig. 9D). The procedure has been described as an open technique, as a laparoscopic method, or as a combined endoscopic and laparoscopic procedure.

Cystoduodenostomy

A cystoduodenostomy is the procedure of choice when the pseudocyst abuts the medial wall of the duodenum (Fig. 10). This is performed by first mobilizing the duodenum and pancreatic head with a Kocher maneuver (Fig. 10A). An incision is made on the lateral wall of the duodenum. If there is less than 1 cm from the medial wall of the duodenum to the pseudocyst, then one can proceed with the cystoduodenostomy. An incision is made on the medial wall of the duodenum, being careful not to injure the ampulla (Fig. 10B). This is carried down to the pseudocyst while avoiding the common bile duct, anterior and posterior gastroduodenal arteries. If there is a substantial amount of pancreatic parenchyma between the medial duodenal wall and the pseudocyst, a cystojejunostomy is usually performed instead (Fig. 10C).

Cystojejunostomy

A cystojejunostomy is performed if the pseudocyst is not adjacent to the stomach or the duodenum. A wide anastomosis is made between the pseudocyst, and the Rouxen-Y jejunal limb (Fig. 11). In all surgical drainage procedures, the contents of the pseudocyst are thoroughly excavated, and a biopsy of the cyst wall is obtained to confirm the diagnosis.

Complications

The mortality rate for the internal drainage procedures are between 0 and 5% (2% for cystogastrostomy, 1.9% for cystojejunostomy, and 0% for cystoduodenostomy) (34). The recurrence rate is 8%.



Fig. 8. External drainage of pancreatic pseudocyst.



Fig. 9. Cystogastrostomy. (A) Anterior gastric wall incision. (B) Posterior gastric wall incision. (C) Creation of cystogastrostomy. (D) Gastrostomy closure.



Fig. 10. Cystoduodenostomy. (A) Duodenum and pancreatic head mobilization. (B) Incision of medial duodenal wall. (C) Creation of cystoduodenostomy.

Alternative Procedures

Ultrasound and CT-guided drainage of pseudocysts provide alternatives to surgical drainage of pseudocysts. Internal drainage with endoscopy has been developed recently (*see* Chapter 21). Pseudocysts can be drained through the stomach, duodenum, or pancreatic duct. Recent reports have encouraging results (*35–38*). However, these are fairly recent developments, and studies are needed to compare endoscopic treatment with surgical drainage. At this time, endoscopic and radiologic the inability to provide drainage may have a higher recurrence rate than surgery because of inadequate debridement, and the inability to provide drainage of proteinaceous contents through small caliber tubes. These less invasive treatments are preferred if the patient is critically ill or chronically debilitated.



Fig. 11. Cystojejunostomy.

Summary

- 1. Pancreatic pseudocysts are amylase rich fluid collections as a result of acute pancreatitis or pancreatic trauma.
- 2. The majority of these cysts resolve without any treatment.
- 3. Treatment is indicated in large symptomatic pseudocysts.
- 4. Several surgical and endoscopic therapies are available for the management of pseudocysts.
- 5. The choice of pseudocyst drainage procedure depends upon the site of pseudocyst, availability of surgical and endoscopic therapy, and general condition of the patient.

REFERENCES

- 1. Pellegrini CA, Heck CF, Raper S, Way LW. An analysis of the reduced mortality and morbidity rates after pancreaticodoudenectomy. Arch Surg. 1989;124:778–781.
- 2. Yeo CJ, Cameron JL, Lillemoe KD, et al. Pancreaticoduodenectomy for cancer of the head of the pancreas. 201 patients. Ann Surg. 1995;221:721–733.
- 3. Fernandez del Castillo C, Rattner DW, Warshaw AL. Standards for pancreatic resection in the 1990s. Arch Surg. 1995;130:295–300.
- 4. Rosemurgy AS, Bloomston M, Serafini FM, et al. Frequency with which surgeons undertake pancreaticoduodenctomy determines length of stay, hospital charges, and in-house mortality. J Gastrointest Surg 2001;5:21–26.
- 5. Cooperman AM, Schwartz ET, Fader A, et al. Safety, efficacy, and cost of pancreaticoduodenal resection in a specialized center based at a community hospital. Arch Surg 1997;132:744–747.
- 6. Porter GA, Pisters PW, Mansyur C, et al. Cost and utilization impact of a clinical pathway for patients undergoing pancreaticoduodenectomy. Ann Surg Onc 2000;7:484–489.
- Holbrook RF, Hargrave K, Traverso LW. A prospective cost analysis of pancreaticoduodenectomy. Am J Surg 1996;171:508–511.
- 8. Nordback IH, Hruban RH, Boitnott JK, et al. Carcinoma of the body and tail of the pancreas. Am J Surg 1992;164:26–31.
- 9. Johnson CD, Schwall G, Flectenmacher J, Trede M. Resection for adenocarcinoma of the body and tail of the pancreas. Br J Surg 1993;80:1177–1179.

- 10. Brennan MF, Moccia RD, Klimstra D. Management of adenocarcinoma of the body and tail of the pancreas. Ann Surg 1996;223:506–512.
- 11. Fabre JM, Houry S, Manddrsheid JC, et al. Surgery for left-sided pancreatic cancer. Br J Surg 1996;83: 1065–1070.
- Sugiyama M, Atomi Y. Pylorus-preserving total pancreatectomy for pancreatic cancer. World J Surg 2000;24:66–70.
- Karpoff HM, Klimstra DS, Brennan MF, et al. Results of total pancreatectomy for adenocarcinoma of the pancreas. Arch Surg 2001;136:44–47.
- Wagner M, Z'graggen K, Vagianos CE, et al. Pylorus-preserving total pancreatectomy. Early and late results. Dig Surg 2001;18:188–195.
- 15. Beger HG, Schlosser W, Friess HM, et al. Duodenum-preserving head resection in chronic pancreatitis changes the natural course of the disease: a single-center 26-year experience. Ann Surg 1999;230: 512–519; discussion 519–23.
- 16. Izbicki JR, Bloechle C, Knoefel WT, et al. Duodenum-preserving resection of the head of the pancreas in chronic pancreatitis. A prospective, randomized trial. Ann Surg. 1995;221:350–358.
- Frey CF, Amikura K. Local resection of the head of the pancreas combined with longitudinal pancreaticojejunostomy in the management of patients with chronic pancreatitis. Ann Surg 1994;220: 492–504; discussion 504–507.
- Howard TJ, Jones JW, Sherman S, et al. Impact of Pancreatic Head Resection on Direct Medical Costs in Patients with Chronic Pancreatitis. Ann Surg 2001;234:661–667.
- Slezak, L, Andersen DK. Pancreatic Resection: Effects on Glucose Metabolism. World J. Surg 2001;25: 452–460.
- 20. McCarthy J, Geenen JE, Hogan WJ. Preliminary experience with endoscopic stent placement in benign pancreatic diseases. Gastrointest Endosc. 1988;34:16.
- 21. Grimm H, Meyer WH, Nam VC, et al. New modalities for treating chronic pancreatitis. Endoscopy 1989;21:70–74.
- 22. Cremer M, Deviere J, Delhaye M, et al. Stenting in severe chronic pancreatitis:results of medium-term follow-up in seventy-six patients. Endoscopy 1991;23:171–176.
- 23. Kozarek RA. Chronic pancreatitis in 1994: is there a role for endoscopic treatment? Endoscopy. 1994; 26:625–628.
- 24. Binmoeller KF, Jue P, Seifert H, et al. Endoscopic pancreatic stent drainage in chronic pancreatitis and a dominant stricture: long-term results. Endoscopy 1995;27:638–644.
- 25. Ponchon T, Bory RM, Hedelius F, et al. Endoscopic stenting for pain relief in chronic pancreatitis: results of a standardized protocol. Gastrointest Endosc 1995;42:452–456.
- 26. Smits ME, Badiga SM, Rauws EA, et al. Long-term results of pancreatic stents in chronic pancreatitis. Gastrointest Endosc 1995;42:461–467.
- 27. Kalady MF, Broome AH, Meyers WC, et al. Immediate and long-term outcomes after lateral pancreaticojejunostomy for chronic pancreatitis. Am Surg 2001;67:478–483.
- Prinz RA, Greenlee HB. Pancreatic duct drainage in 100 patients with chronic pancreatitis. Ann Surg 1981;194:313–320.
- 29. Wilson TG, Hollands MJ, Little JM. Pancreaticojejunostomy for chronic pancreatitis. Aust NZ J Surg 1992;62:111–115.
- 30. Bradley EL. Long-term results of pancreaticojejunostomy in patients with chronic pancreatitis. Am J Surg 1987;153:207–213.
- Hart MJ, Miyashita H, Morita N, et al. Pancreaticojejunostomy report of a 25 year experience. Am J Surg 1983;145:567–570.
- 32. Greenlee HB, Prinz RA, Aranha GV. Long-term results of side-to-side pancreaticojejunosotmy. World J Surg 1990;14:70–76.
- 33. Nealon WH and Thompson JC. Progressive loss of pancreatic function in chronic pancreatitis is delayed by main pancreatic duct decompression. A longitudinal prospective analysis of the modified Puestow proceduure. Ann Surg 1993;217:466–476.
- Bumpers HL, Bradley EL. Treatment of pancreatic pseudocysts. In: Howard J, Idezuki Y, Ihse I, et al., eds. Surgical Diseases of Pancreas. Williams & Wilkins, Baltimore, MD, 1998, pp. 423–432.
- 35. Sharma SS, Bhargawa N, Govil A. Endoscopic management of pancreatic pseudocyst: a lon-term follow-up. Endoscopy 2002;34:203–207.

- 36. Lo SK, Rowe A. Endoscopic management of pancreatic pseudocysts. Gastroenterologist 1997;5:10-25.
- Beckingham IJ, Krige JE, Bomman PC, et al. Endoscopic management of pancreatic pseudocysts.Br J Surg 1997;84:1638–1645.
- 38. Howard J, Idezuki Y, Ihse I, Prinz R, eds. Surgical Diseases of the Pancreas. Williams & Wilkin, Baltimore, MD, 1998.

Endoscopic Management of Pancreatic Pseudocysts

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CONTENTS

Introduction Clinical Presentation Diagnosis Differential Diagnosis Therapy Transpapillary Cystic Drainage (TCD) Transmural or Transenteric Drainage Endoscopic Ultrasound (EUS) Alternative Procedure and Cost Summary References

INTRODUCTION

A pancreatic pseudocyst is defined as an amylase-rich fluid collection enclosed by a nonepithelialized wall arising in or adjacent to the pancreas, as a result of acute or chronic pancreatitis, pancreatic trauma, or pancreatic duct obstruction (1). The reported incidence varies between 1.6% and 69% (2,3). Acute pancreatic collections, which occur in 50% of cases of moderate to severe pancreatitis should not be confused with pancreatic pseudocysts, as more than 50% of these lesions resolve spontaneously (1,4). These occur as a result of an exudative reaction to pancreatic injury and inflammation, and do not communicate with the pancreatic duct, and therefore, do not contain a high concentration of pancreatic enzymes. In addition, they do not possess a well-defined wall, and there is a loss of an interface between the fluid and adjacent organs. Approximately 10–15% may persist beyond 3 wk, at which time they may develop a capsule and may be diagnosed by ultrasound or computed tomography (CT) scan as a pancreatic pseudocyst (5).

CLINICAL PRESENTATION

The majority of pseudocysts are asymptomatic. However, they may present with a variety of clinical symptoms depending on the location and extent of fluid accumulation.

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Upper abdominal pain or distress of insidious onset, often localized to the midepigastrium is the most frequent symptom (4). Expansion of the pseudocyst may likewise result in duodenal or biliary obstruction, vascular occlusion, or fistula formation into adjacent structures such as the viscera, pleura, or pericardium (6). Leakage from the pseudocyst or pancreatic duct with concomitant fistula formation can result in pancreatic ascites or a pleural effusion. Pseudocyst rupture occurs in less than 3% of patients (7), and may be clinically asymptomatic. However, rupture into the peritoneum can present as an acute abdominal event necessitating emergent surgery, which is often fatal (8). Erosion into the gastrointestinal tract may result in hematemesis, melena, or massive hematochezia (9). Massive bleeding into the gastrointestinal tract occurs in approx 5-10% of patients (10,11), and occurs as a result of pseudocyst erosion into a major pancreatic or peripancreatic vessel, leading to free rupture or pseudoaneurysm formation (12). The diagnosis should be suspected in patients with an unexplained drop in hematocrit, recurrent gastrointestinal bleeding or in the setting of an enlarging pulsatile mass or abdominal bruit in patients with increasing abdominal pain. Bolus dynamic CT is the most useful initial diagnostic modality to demonstrate the presence of hemorrhage or a pseudoaneurysm. Subsequent angiography is the procedure of choice for isolating the site of bleeding and directing embolization therapy (11). Surgery is otherwise indicated in patients who are hemodynamically unstable or when embolization is unsuccessful or technically not feasible. Secondary cyst infection occurs in approx 10% of patients (7). A confirmatory diagnosis is established on the basis of a positive Gram stain or bacterial isolate from the cyst aspirate in the context of frank pus or sepsis. Percutaneous drainage is successful in approx 85% of cases and should be the initial treatment of choice (13).

DIAGNOSIS

Ultrasound or CT scan usually makes the diagnosis of a pancreatic pseudocyst. CT scan is by far the most accurate test for detecting pseudocyst with a sensitivity of 90-100% in contrast to ultrasonography that has a sensitivity of 75-90%. Ultrasound is often operator dependent and its use is limited in obese patients. On the other hand, given its convenience and lower cost, ultrasonography is the ideal method for monitoring pseudocyst size (14).

The role of endoscopic retrograde cholangiopancreatography (ERCP) following the diagnosis of a pancreatic pseudocyst remains controversial. Advocates for ERCP have reported a 95% advantage of demonstrating pancreatic ductal abnormalities and an 80% detection rate for duct-pseudocyst communication (15). Additionally, routine ERCP has been found to alter the operative plan for pseudocyst drainage in 24 of 41 patients, with 19 requiring a surgical drainage procedure (16). Furthermore, demonstration of ductal abnormalities, particularly ductal communication or stricturing of the main pancreatic duct plays a major role when considering internal drainage over a percutaneous drainage procedure (17). In contrast, ERCP has been demonstrated to exacerbate acute pancreati-tis, resulting in bacterial seeding of fluid collections, and needlessly increase the extent of operation without a significant advantage in outcome (18,19).

DIFFERENTIAL DIAGNOSIS

Pancreatic pseudocysts comprise approx 75% of all cystic lesions within or adjacent to the pancreas. The remainder of pancreatic cysts consists of retention cysts (10%),

congenital cysts (5%), and cystic neoplasm (20). It is essential to distinguish between a cystic neoplasm, and a pancreatic pseudocyst to determine the specific therapeutic intervention, more specifically, resection rather than a drainage procedure. Several guidelines based on clinical and radiologic criteria have been proposed to differentiate those with a greater risk of a cystic neoplasm. A cystic lesion is considered to be a probable neoplasm if: 1) there is no antecedent history of pancreatitis; 2) the pancreatogram is normal on ERCP; 3) there are multiple cysts or internal septae on CT scan; 4) it is a hypervascular tumor as demonstrated on angiography; 5) it is thin-walled, and not adherent to any surrounding tissue at the time of laparotomy; 6) serum amylase levels are normal; and 7) fluid amylase levels are equal to or lower than serum (21).

Some authors advocate percutaneous aspiration with fluid analysis for viscosity, CA-125, carcinoembryonic antigen (CEA) and cytology. CA-125 and CEA levels have been found to be elevated in neoplastic cysts, and lower in pseudocysts (22). Cytologic analysis has an accuracy of approx 88% for mucinous cysts and its diagnostic value in serous cystadenomas appears to be limited (23).

THERAPY

Majority of pancreatic pseudocysts regress spontaneously. Earlier studies have indicated that cysts less than 6 wk old have a resolution rate of approx 40% in contrast with a resolution rate of 8% for cysts present for 7-12 wk. Beyond 13 wk, no further resolution occurred and the complication rate increased from 20% to 46% and 75%, respectively (5). Based on these findings, surgery has been a widely accepted approach for cysts that persist beyond 6 wk. However, two additional reviews advocate a more conservative approach with expectant follow-up. In a retrospective review of 68 patients with asymptomatic pseudocysts, approx 63% of patients either had spontaneous pseudocyst resolution or remained asymptomatic at a mean follow-up averaging 51 mo. There was a 9% incidence of serious complications including pseudoaneursym formation in three, perforation in two, and spontaneous abscess formation in one. Thirty-five percent of patients underwent operative therapy, generally for cyst enlargement associated with pain or gastric and biliary obstruction (24). In another series of 75 patients, 39 patients underwent surgery for severe abdominal pain; complications or progressive cyst enlargement, whereas the remaining 36 patients were followed conservatively with serial CT scans. Approx 60% of patients in the latter group had complete resolution at 1 yr with only one pseudocystrelated complication of intracystic hemorrhage with no reported mortality (25).

Pseudocyst drainage is indicated in: 1) symptomatic patients; 2) pseudocysts greater than 6 cm in size or in progressively enlarging cysts; and 3) infected pseudocysts (4). Immediate drainage can be accomplished safely in patients with mature cysts walls or with cysts that occur in the setting of chronic pancreatitis (26).

Surgery remains the standard drainage procedure of choice despite the availability of less-invasive procedures. Surgery is associated with a morbidity rate of 10% to 30%, mortality rate of 1% to 5%, and 10% to 20% recurrence rate (27,28). Endoscopic drainage procedures compare favorably to standard operative techniques. Similar success rates of 50% and 52% were reported in a retrospective review evaluating surgical management to endoscopic therapy (29). Comparably, equal rates were found in 71 cases of endoscopic drainage and 73 cases of surgical drainage, with a reported resolution rate of 72%, morbidity of 15%, and mortality of 1% (30). These procedures should, however,

be performed on selected patients in experienced centers where surgical back-up is available.

Endoscopic drainage procedures are extensions of ERCP techniques and consist of: 1) tanspapillary; and 2) transenteric or transmural approaches.

TRANSPAPILLARY CYSTIC DRAINAGE (TCD)

Transpapillary cyst drainage (TCD) is feasible in the presence of a documented communication between the pseudocyst and pancreatic ductal system. Communication between the pseudocyst and duct occurs in approx 40–69% of pseudocysts (31–33) and is frequently seen in pseudocysts associated with chronic pancreatitis rather than those seen with acute pancreatitis (34). An ERCP is performed followed by a pancreatic duct sphincterotomy with subsequent insertion of a 5–7 Fr stent over a guidewire and left *in situ* for a mean of 3 mo (Fig. 1). Because of the potential risk of bacterial seeding, and abscess formation, antibiotic prophylaxis with either Ciprofloxacin or Ceftazidime (35) is administered preoperatively followed by a 7-d course of oral antibiotics. Some authors recommend the insertion of a nasocystic drain to allow irrigation and drainage of intracystic debris or pus.

In a combined series of 117 patients, successful drainage was accomplished with transpapillary cystic drainage in 84% of patients with a reported recurrence rate of 9%, and a complication rate of 12% (36). Procedure-related complications were few with no reported deaths. Bleeding occurred in one patient that did not require intervention. The most frequently encountered complication was acute pancreatitis (6 patients), which, however, was mild and self-limited. Stent occlusion was uncommon and secondary pancreatic cyst infection was seen in 3 patients, which resolved with stent change (37,38). Failures were associated with pancreatic pseudocysts localized to the pancreatic tail (34, 39). There was no added advantage of combined TCD and transmural drainage over TCD alone (34). Pancreatic ductal irregularities frequently associated with chronic pancreatitis are seen in approx 50% of patients following transpapillary stenting (40).

TRANSMURAL OR TRANSENTERIC DRAINAGE

Transmural or transenteric endoscopic drainage procedures are performed through several endoscopic approaches through the stomach (endoscopic cystogastrostomy) or duodenum (endoscopic cystoduodenostomy).

Several prerequisites need to be fulfilled pursuant to endoscopic transmural drainage (Table 1). Ideally, the pseudocyst must be situated within the pancreatic head or body, and must be firmly adherent to the gastrointestinal tract to cause a visible impression on the gastric or duodenal wall at the time of endoscopy. Additionally, the distance between the pseudocyst and the adjacent gastric or duodenal wall should not exceed 1 cm on CT scan or endoscopic ultrasound (41-44).

Cystic neoplasms should be identified and managed appropriately. Cystic neoplasms managed inappropriately as pseudocysts may result in serious complications and compromise future surgical resection (21,45).

Pseudoaneurysms occur in approx 10% of pseudocysts (12,47,48) and represent an absolute contraindication to endoscopic intervention, unless arterial embolization has been accomplished first (6). Gastric varices in the setting of portal hypertension should be identified to minimize the risk of inadvertent puncture and hemorrhage (49).



Fig. 1. Transpapillary pancreatic pseudocyst drainage. A 5–7 French stent placed after pancreatic sphincterotomy.

Table 1	
Guidelines for Endoscopic Pancreatic Pse	eudocyst Drainage

- 1. Cysts must be allowed to mature prior to drainage
- 2. Assess for the presence of pseudoaneurysm
- 3. Rule out the presence of a cystic neoplasm
- 4. Identify gastric varices in the presence of portal hypertension
- 5. Identify debris within the pseudocyst
- 6. Outline the pancreatic duct by ERCP
- 7. The pseudocyst should be in close approximation to the gastric or duodenal wall
- 8. Utilize a transpapillary approach whenever feasible
- 9. Endoscopic needle localization should be used prior to puncture

Some authors advocate endoscopic needle localization (ENL) or needle aspiration of a pseudocyst prior to cyst puncture to reduce the risk of bleeding (50). Repeated bloody aspirate may represent inadvertent puncture of a blood vessel wall or pseudoaneurysm formation and should warrant further investigation prior to any attempt at drainage.

Pancreatic necrosis as demonstrated by contrast-enhanced CT might result in inadequate cyst evacuation, and subsequently increases the risk of infection, and should serve as a deterrent to, but not preclude endoscopic transmural drainage (51,52).

Endoscopic cystogastrostomies (ECG) and cystoduodenostomies (ECD) require the puncture of the gastric or duodenal wall at the point of an identifiable impression in the visceral lumen. A side-viewing endoscope is used and access into the cyst is achieved



Fig. 2. Endoscopic cystgastrostomy. Two pigtail stents passed in the pseudocyst through posterior gastric wall.

with a diathermic needle. Once entry into the cyst is confirmed, a guidewire is inserted and the opening is enlarged to approx 3-50 mm. Balloon tract dilatation has been utilized to enlarge the opening to reduce the risk of bleeding. One or two 7-10 Fr stents are subsequently inserted into the cyst to maintain patency and are left in place for a mean period of 2-4 mo or until ultrasonographic confirmation of cyst resolution occurs (Fig. 2).

In a cumulative series of 50 patients who underwent endoscopic cystogastrostomy, successful pseudocyst drainage was achieved in 82 % of patients with a recurrence rate of 18%. Major complications included bleeding in 8%, and perforation in 8% of patients with no reported deaths. The collective incidence of bleeding requiring surgical intervention was 7%. Bleeding occurred at the time of gastrostomy enlargement with the sphincterotome. Small incisions and balloon dilatation of the gastrotomy tract have been recommended to reduce the risk of these complications (37,42-44).

Concurrently, in a series of 71 patients who underwent ECD, drainage was successful in 89% of patients with a reported recurrence rate of 6%, with a median follow-up of 9–48 mo. Complications were less frequent, with perforation in 4% and severe bleeding in 4%. All three patients that developed perforations were successfully managed with antibiotics. The overall incidence of bleeding requiring surgery was 3%. In two reported cases of bleeding, which resulted in one death, bleeding occurred as a result of a pseudoaneurysm (43). ECD confers the advantage of longer cystoduodenal fistula patency over ECG (41).

ENDOSCOPIC ULTRASOUND (EUS)

EUS offers several advantages over the standard endoscopic drainage procedures (49) (Table 2).

Endosonography allows confirmation of pseudocyst depth, and distance to the gastrointestinal lumen, and has the best sensitivity for detecting submucosal blood vessels, which may be a direct contraindication to direct puncture (44,54). However, in two large series, (37,38) there were four reported cases of major bleeding, out of which

Table 2 Value of Endosonography in Endoscopic Drainage of Pancreatic Pseudocysts

- 1. Measures the distance between the pseudocyst and gastrointestinal wall
- 2. Images gastric varices and submucosal vessels
- 3. Identifies the presence of pseudoaneurysms
- 4. Distinguishes between cystic neoplasms and pancreatic pseudocysts
- 5. Demonstrates the presence of intracystic debris
- 6. Allow visualization of the puncture site in the absence of an intraluminal bulge
- 7. Endoscopic drainage may be performed under EUS guidance alone

three required surgical intervention. It is of value in allowing optimal visualization and localization of the puncture site in circumstances where there is no visible intraluminal bulge (54,55). This can be accomplished, however, by endoscopic needle localization, which provides the same information without interfering with any anticipated therapeutic procedures (50).

With the advent of the large-channel therapeutic curvilinear array ultrasound endoscopes, pseudocyst drainage can be performed solely under sonographic guidance, thereby obviating the need for separate endoscopies and fluoroscopy (56,57). In the absence of an adequate pancreatogram performed with the standard procedures, pancreatic strictures and ductal disruptions are likely to be missed resulting in a potentially higher rate of pseudocyst recurrence (49).

ALTERNATIVE PROCEDURE AND COST

Pancreatic pseudocyst can also be drained surgical and radiologically. Merits and demerits of these procedures are discussed in detail in Chapter 20. To date, there are no studies comparing the cost of management of pseudocyst by available techniques.

SUMMARY

- 1. Pancreatic pseudocyst is an uncommon complication of pancreatitis. Most of the pseudocyst resolve spontaneously. Only large symptomatic pseudcysts need treatment.
- 2. Several radiological modalities are available to diagnose pseudocysts. CT scan seems to have advantage over ultrasound in providing better definition of the pseudocyst. Role of ERCP is controversial.
- 3. Endoscopic drainage is effective in relieving symptoms and has a low complication rate. EUS prior to endoscopic therapy is helpful in delineating the anatomy and ruling out pseudoaneurysm.

REFERENCES

- 1. Bradley EL. A clinically based classification system for acute pancreatitis. Arch Surg 1993;128: 586–590.
- 2. Elechi EN, Calender CO, Leffal LD, et al. The treatment of pancreatic pseudocysts by external drainage. Surg Gynecol Obstet 1979;148:707–710.
- 3. Siegelman SS, Copeland BE, Saba GP, et al. CT of fluid collections associated with pancreatitis. Am J Radiol 1980;134:1121–1132.
- 4. Pitchhumoni CS, Agarwal N. Pancreatic pseudocysts. When and how should drainage be performed? Gastroenterol Clinics 1999;28:615–639.

- 5. Bradley EL, Clements J, Gonzalez AC. The natural history of pancreatic pseudocysts: A unified concept of management. Am J Surg 1979;137:135–141.
- 6. Howell D, Elton E, Parsons W. Diagnosis and management of pseudocysts of the pancreas 2000 UpToDate.www.uptodate.com 1999;8:1–5.
- 7. Stephen RB, Bradley EL. Pancreatic infections. Gastroenterologist 1996;4:163-168.
- 8. Hanna WA. Rupture of pancreatic cysts: Report of a case and review of the literature. Br J Surg 1960; 47:495–498.
- 9. Santos JCM, Feres O, Rocha J, et al. Massive lower gastrointestinal hemorrhage caused by pseudocyst of the pancreas ruptured into the colon: Report of two cases. Dis Colon Rectum 1992;35:75–77.
- Adams DB, Zellner JC, Anderson MC. Arterial hemorrhage complicating pancreatic pseudocyst: Role of angiography. J Surg Res 1993;54:150–156.
- 11. Vatic I. Vascular complications of pancreatitis. Radiol Clint North Am 1989;27:81-91.
- El Haml A, Park R, Addax G, et al. Bleeding pseudocysts and pseudoaneurysms in chronic pancreatitis. Br J Surg 1991;8:1059–1063.
- 13. Adams DB, Harvey TS, Anderson MC. Percutatneous catheter drainage of infected pancreatic and peripancreatic fluid collections. Arch Surg 1990;125:1554–1557.
- Van Sonnenberg W. Wittich GR, Casola G, et al. Percutaneous drainage of infected and non-infected pancreatic pseudocysts: Experience in 101 cases. Radiology 1989;170:757–761.
- Sugawa C, Walt AJ. Endoscopic retrograde pancreatography in the surgery of pancreatic pseudocysts. Surgery 1979;86:639–647.
- 16. Nealon WH, Townsend CM, Thompson JC. Preoperative endoscopic retrograde cholangiopancraticography (ERCP) in patients with pancreatic pseudocyst associated with resolving acute and chronic pancreatitis. Ann Surg 1989;209:532–537.
- 17. Ahearne PM, Baille JM, Cotton PB, et al. An endoscopic retrograde cholangiopancraticography (ERCP) based algorithm for the management of pancreatic pseudocysts Am J Surg 1992;163:111–116.
- 18. Lillemoe K, Yeo CJ. Management of complications of pancreatitis. Curr Prob Surg 1998;35:3-98.
- 19. Walt AJ, Bowman Dl, Weaver DW, et al. The impact of technology in the management of pancreatic pseudocysts. Arch Surg 1990;125:759–763.
- 20. Yeo CJ, Sarr MG. Cystic and pseudocystic diseases of the pancreas. Curr Prob Surg 1994;31:167–243.
- 21. Warshaw AL, Rutledge PL. Cystic tumors mistaken for pancreatic pseudocysts. Ann Surg 1987;205: 393–398.
- 22. Steinenberg M, Gelfard R, Anderson K, et al. Comparison of the sensitivity and specificity of the CA 19.9 and carcinoembryonic antigen assays in detecting cancer of the pancreas. Gastroenterology 1986; 90:343–349.
- 23. Sand JA, Hyoty MK, Mattila J, et al. Clinical assessment compared with cyst fluid analysis in the differential diagnosis of cystic lesions in the pancreas. Surgery 1996;119:274–280.
- 24. Vitas GJ, Sarr MG. Selected management of pancreatic pseudocysts: Operative versus expectant management. Surgery 1992;111:123–130.
- 25. Yeo CJ, Bastides JA, Lynch-Nyhan A, et al. The natural history of pancreatic pseudocysts documented by computed tomography. Surg Gynecol Obstet 1990;170:411–417.
- 26. Warshaw AL, Rattner DW. Timing of surgical drainage for pancreatic pseudocyst. Ann Surg 1985;202: 720–724.
- 27. Kohler H, Schafmayer A, Lutdke FE, et al. Surgical treatment of pancreatic pseudocysts. Br J Surg 1987;74:813–812.
- 28. Williams KJ, Fabian TC. Pancreatic pseudocyst: recommendations for operative and non-operative management. Am Surg 1992;58:199–205.
- Froeschle G, Meyer-Panwitt U, Breuckner M, et al. A comparison between surgical endoscopic and percutaneous management of pancreatic pseudocysts long-term results. Acta Chir Belg 1993;93:102–106.
- 30. Barthet M, Buggalo M, Moriera LS, et al. Management of cysts and pseudocysts complicating chronic pancreatitis: A retrospective study of 143 patients. Gastroenterol Clin Biol 1993;17:270–276.
- 31. Barkin JS, Hyder SA. Changing concepts in the management of pancreatic pseudocysts. Gastrointest Endosc 1989;35:62–64.
- 32. Ligouroy C, Lefebvre JF, Vitale GC. Endoscopic drainage of pancreatic pseudocysts. Can J Gastroenterol 1990;4:568–571.
- Kolars, JC, O'Connor M, Ansel H, et al. Pancreatic pseudocysts: clinical and endoscopic experience. Am J Gastroenterol 1989;84:259–263.

- Barthet M, Sahel J, Bodiou-Bertei C, et al. Endoscopic transpapillary drainage of pancreatic pseudocysts. Gastrointest Endosc 1995;42:208–213.
- Mani V, Cartwright K, Dooley L, et al. Antibiotic prophylaxis in gastrointestinal endoscopy: A report by a working party for the British Society of Gastroenterology endocopy committee. Endoscopy 1997; 29:114.
- 36. Beckingham IJ, Krige EJ, Bornman PC, et al. Endoscopic management of pseudocysts. Br J Surg 1997; 84:1638–1645.
- 37. Smits ME, Rauws EA, Tygat GN, et al. The efficacy of endoscopic treatment of pancreatic pseudocysts, Gastrointest Endosc 1995;42:202–207.
- 38. Binmoeller KF, Seifert H, Walter A, et al. Transpapillary and transmural drainage of pancreatic pseudocysts. Gastrointest Endosc 1995;42:219–224.
- 39. Catalano MF, Geenan JE, Schmalz MJ, et al. Treatment of pancreatic pseudocysts with ductal communication by transpapillary pancreatic duct endoprosthesis. Gastrointest Endosc 1995;42:214–218.
- 40. Kozarek RA. Pancreatic stents induce ductal changes consistent with chronic pancreatitis. Gastrointest Endosc 1990;36:93–95.
- 41. Cremer M, Deviere J, Engelholm L. Endoscopic management of cysts and pseudocysts in chronic pancreatitis: long term follow up after 7 years of experience. Gastrointest Endosc 1989;35:1–9.
- Dohmoto M, Rupp KD. Endoscopic management of pancreatic pseudocysts. Diagnos Therap Endosc 1994;1:29–35.
- 43. Sahel J, Bastid C, Pellat P, et al. Endoscopic cystoduodenostomy of cysts of chronic calcifying pancreatitis: a report of 20 cases. Pancreas 1987;2:447–453.
- 44. Grimm H, Binmoeller KF, Soehendra N. Endosonography guided drainage of a pancreatic pseudocyst. Gastrointest Endosc 1988;34:170–171.
- 45. Warsaw AL, Rutledge PL. Cystic tumors mistaken for pancreatic pseudocysts. Am Surg 1987;205:393.
- 46. Sperti C, Cappellazzo F, Pasqual C, et al. Cystic neoplasm of the pancreas: Problems in differential diagnosis. Am Surg 1993;59:740.
- 47. Kiviluoto T, Schroder T, Kivilaakso E, et al. Acute hemorrhage associated with pancreatic pseudocyst and chronic pseudocyst and chronic pancreatitis. Ann Chir Gynaecol 1984;73:214.
- 48. Pitkaranta P, Haapianen R, Kivisari I, et al. Diagnostic evaluation and aggressive surgical approach in bleeding pancreatic pseudocysts. Arch Surg 1996;131:278.
- 49. Chak A. Recent advances in endoscopic ultrasonography. Endoscopic guided therapy of pancreatic pseudocysts. Gastrointest Endosc 2000;52:1–7.
- 50. Howell DA, Holbrook RF, Bosco JJ, et al. Endoscopic needle localization of pancreatic pseudocysts before transmural drainage. Gastrointest Endosc 1993;39:693–698.
- 51. Hariri M, Slivka A, Carlock DL, et al. Pseudocyst drainage predisposes to infection when pancreatic necrosis is unrecognized. Am J Gastroenterol 1994;89:1781.
- 52. Baron TH, Thaggard WG, Morgan DE, Stanley RJ. Endoscopic therapy for organized pancreatic necrosis. Gastroenterology 1996;111:755–764.
- 53. Fockens P, Johnson TG, van Dulleman HM, Huibregtse K, Tytgat GN. Endosonographic imaging of pancreatic pseudocysts before endoscopic transmural drainage. Gastrointest Endosc 1997;46:412–416.
- 54. Savides TJ, Gress F, Sherman S, Rahaman S, Lehman GA, Hawes RH. Ultrasound catheter probe assisted endocopic cystogastrostomy. Gastrointest Endosc 1995;41:145–148.
- 55. Chan AT, Heller SJ, Van Dam J, et al. Endoscopic cystgastrostomy: role of endoscopic ultrasonography. Gastrointest Endosc 1995;41:145–148.
- 56. Wiersema MJ. Endosonography guided cystduodenostomy with a therapeutic ultrasound endoscope. Gastrointest Endosc 1996;44:614–617.
- 57. Siefert H, Dietrich C, Schmitt T, et al. Endoscopic ultrasound guided one-step transmural drainage of cystic abdominal lesions with a large channel echo endoscope. Endoscopy 2000;32:255–259.

VII SURGERY ON AORTA AND ITS BRANCHES

22

Surgery of the Abdominal Aorta and Branches

Stephanie Saltzberg, MD, Justin A. Maykel, MD, and Cameron M. Akbari, MD

CONTENTS

INTRODUCTION SURGICAL CONSIDERATIONS OPERATIVE TECHNIQUE ALTERNATIVE PROCEDURES COMPLICATIONS OF AORTIC SURGERY COST SUMMARY REFERENCES

INTRODUCTION

Diseases of the aorta and its branches may be commonly thought of as either occlusive or aneurysmal. Within the aorta itself, the most commonly affected area is the abdominal aorta, and, more specifically, the disease is mostly confined to the level of aorta below the renal arteries, or the so-called infrarenal segment. Whereas arteriosclerosis obliterans clearly is the etiologic agent in nearly all cases of occlusive disease, the pathogenesis of atherosclerotic aortic aneurysms is less clear, despite their name. Indeed, emerging evidence has promoted the participation of other factors in addition to atherosclerosis, such as excessive collagenase and elastase activity, genetic susceptibility, and hemodynamic factors (1).

Arteriosclerotic occlusive disease of the aorta and iliac vessels known as, "aortoiliac disease" is one of the most common conditions encountered by vascular surgeons. Because atherosclerosis is a systemic process, the occlusive disease is seldom confined to the aortoiliac vessels alone, and most patients will have occlusive disease involving the infrainguinal or even visceral (mesenteric and renal) arteries as well. In fact, aortic "spillover" atherosclerotic disease, in which atherosclerotic plaque extends from the aorta into the orifice of the visceral vessels, is the most common cause of visceral arterial

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Fig. 1. CT scan of an abdominal aortic aneurysm with intramural thrombus.

occlusive disease. It is, therefore, axiomatic that treatment of renal or mesenteric occlusive disease should consider this process (2).

The single most important consideration in the approach to abdominal aortic aneurysms (AAA) is their propensity for rupture and death (Fig. 1). In the United States, it is the 13th leading cause of death, and implicated in more than 15,000 deaths annually. Because of Laplace's Law, in which wall tension is directly related to radius, the risk of rupture increases with increasing size of the aneurysm, and, in fact, the greatest predictor of rupture remains the absolute size of the aneurysm itself (1).

The normal diameter of the infrarenal aorta (by far, the most common site of abdominal aortic aneurysms) is about 2 cm, and because an aneurysm is defined as a localized increase in diameter greater than 50% normal, almost all AAAs are 3.5 cm or larger. Several natural history studies have shown that the risk of rupture dramatically increases once the aneurysm approaches 5 cm, and the risk rises exponentially above this number. For example, the rupture rate for a 5-cm AAA is about 5% annually, which rises to 7% annually for a 6-cm AAA, and then to 20% annually for aneurysms greater than 7 cm in diameter. Aneurysms smaller than 5 cm have about a 1% annual rupture rate, and most of these small asymptomatic aneurysms are followed carefully to ensure that they do not enlarge suddenly (1).

SURGICAL CONSIDERATIONS

Emerging technology has been responsible for multiple new treatments in patients with aortic disease, including percutaneous transluminal angioplasty (PTA), either with or without stenting, stent grafts in patients with aortic aneurysms, and improved tech-

niques with the "gold standard" of open surgical repair. Whatever modality is used, the underlying question should address whether *any* treatment is needed. For example, based on the natural history and rupture risk, most surgeons will consider treatment for an aneurysm of the abdominal aorta greater than 5 cm, but this decision may be modified in a high-risk patient. Similarly, an active middle-aged person with lifestyle-limiting buttock, and thigh claudication secondary to aortoiliac occlusive disease should be approached differently from an elderly patient who is bedridden.

Insofar as operative approaches are treating a *localized* manifestation of a systematic process (i.e., atherosclerosis), consideration should be directed toward some assessment of coronary reserve and cardiac risk. Multiple studies and scoring systems have focused on this, and their discussion is beyond the scope of this chapter; however, suffice it to say that more than 50% of patients undergoing vascular reconstruction will have some element of cardiac disease. Specifically, this becomes more important in the patient undergoing aortic surgery, in that clamping of the aorta presents a tremendous increase in cardiac afterload and strain. Ultimately, preoperative evaluation (including bedside assessment, physiologic functional testing, coronary angiography, or some combination of these) should be tailored to the individual patient, and postoperative care should include a heightened awareness that the most common cause of death in these patients remains cardiac-related.

OPERATIVE TECHNIQUE

The abdominal aorta may be approached by either a transperitoneal (using either a midline or transverse abdominal incision) or a retroperitoneal (flank) approach. The traditional, and most common, technique is via a midline, transperitoneal approach. Although there are some data to suggest a decreased incidence of postoperative pulmonary complications and ileus with the retroperitoneal approach, the final decision is ultimately based on the patient's anatomy, body habitus, and on the individual surgeon's personal preference and experience with the two approaches.

The transperitoneal technique is usually through a midline incision from the xiphoid to the pubis (Fig. 2). Once the peritoneal cavity is entered, the abdominal organs are manually and visually inspected for any untoward and unexpected pathology. Because the abdominal aorta is located in the retroperitoneum, below the root of the small bowel mesentery, it is usually approached by first lifting, and carefully retracting the small bowel and fourth portion of the duodenum to the right of midline. The retroperitoneum is then incised, allowing for exposure of the aorta. The left renal vein is an important landmark, usually crossing anterior to the aorta, and signifies the cephalad extent of the dissection for most infrarenal pathology.

The dissection proceeds more distally on top of the aorta to the level of the iliac arteries. These are carefully dissected free for subsequent control and clamping. For more distal exposure of the iliac arteries, attention should be directed to finding the ureters as they cross anterior to the vessels.

If the suprarenal aorta need be exposed, as for renal artery reconstruction or mesenteric artery bypass, the dissection is directed more cephalad above the left renal vein. The aorta can be clamped at this location either suprarenally (i.e., above the renal arteries, but below the superior mesenteric artery), or it may be clamped in a supraceliac position. The latter implies cross-clamping of the aorta at the level of the diaphragm and assumes a



Fig. 2. Abdominal aortic aneurysm via transperitoneal approach. Dotted line shows the extent of the aneurysm.

greater physiologic insult owing to the warm ischemia time experienced by the visceral organs (3).

Once the aorta is dissected free, the patient is anticoagulated with a bolus of heparin and the aorta is clamped. The iliac vessels are usually clamped as well, to prevent troublesome back-bleeding from the open aorta. For infrarenal aortic aneurysms, the aneurysm sac is opened longitudinally, the aorta is completely divided proximal to the aneurysm, and similarly divided at the level of the iliac vessels (Fig. 3). Lumbar arteries typically arise from the infrarenal aorta, and their orifices are oversewn from within the aneurysm sac. The graft is then sewn in place proximally and distally, and subsequently the opened aneurysm sac is closed over the graft (Figs. 4–6). If the aneurysm extends to above the renal vessels, the renal arteries are "reimplanted" and sewn into the side of the graft.

If the operation is being performed for occlusive disease, the proximal infrarenal aortic anastomosis may be performed either "end to end," that is sewn to the divided aorta. Alternatively, it may be performed "end to side," in which the aorta is not divided, but instead a small elliptical piece of aorta is removed, and the end of the graft sewn to



Fig. 3. Opening of aneurysm sac with proximal control of the aorta and distal control of the iliac arteries. Dotted line shows the excised wall of the aneurysm.

the side of the aorta. As the occlusive process typically involves the iliac arteries, and because of concern over progression of disease in the iliac segment, the usual outflow site for these grafts is at the femoral level.

ALTERNATIVE PROCEDURES

Endovascular stent grafts are an alternative to the conventional open surgical repair of abdominal aortic aneurysms in carefully selected patients. Improvements in technology have increased the ability to perform these procedures. With current technology, 50% of patients with AAA are candidates for an endovascular repair rather than conventional surgery. These compact systems are comprised of prosthetic grafts coupled to stents that are deployed via a transfemoral approach. The reported theoretical advantages of the endovascular approach to AAA include decreased cardiac and pulmonary complications, fewer blood transfusions, minimal surgical dissection, and increased availability to those with comorbidities that would exclude them from conventional repair (4).



Fig. 4. Proximal anastomosis of a graft and aortic remnant.

Patients initially selected for the procedure were considered too high risk for the traditional "open" approach. This group included patients of advanced age, low ejection fraction, poor respiratory function, and prior abdominal operations. Because of its initial success, ndovascular repair has since been expanded to include lower risk patients. The most important criteria for suitability are anatomic considerations. These criteria include: suitable proximal neck (>15 mm length, <28 mm diameter) without thrombus or calcification, suitable distal cuff (>10 mm length, <28 mm diameter), suitable iliac vessels, no evidence of mesenteric occlusive disease, and no evidence of severe tortuosity (4). Technical aspects, complications and other details are described in Chapter 23.

COMPLICATIONS OF AORTIC SURGERY

Improvements in perioperative management and surgical technique have led to a significant decrease in operative mortality and morbidity for operations performed on the abdominal aorta. Most large series published within the past decade have reported perioperative mortality rates of less than 3-5%, as compared to a greater than 20% operative mortality three decades earlier (1).



Fig. 5. Proximal anastomosis of aortic graft, and visualization of the site for distal anastomosis.

Despite this, however, it must be recognized that clamping of the aorta poses a considerable physiologic strain, and in view of the systemic nature of the atherosclerotic process, the first consideration should be of prevention and management of cardiac complications. These may include myocardial infarction (MI), congestive heart failure, and arrhythmias, and indeed in patients with both occlusive and aneurysmal aortic disease, the most common cause of early and late mortality remains these cardiac complications. Intraoperative hypotension, which also compromises coronary flow, may also occur. This is usually caused by either intraoperative hemorrhage or following release of the aortic clamp (declamping hypotension) (5).

Pulmonary complications may also arise in these patients, again owing to the high incidence of cigarette smoking and preexisting pulmonary disease. Not uncommonly, postoperative atelectasis and airway collapse may also occur, often accentuating the already compromised pulmonary status. The reasons for this include the presence of a nasogastric tube (described in more detail below), which impairs the normal clearance of secretions, and postoperative pain, which prevents the voluntary mechanisms of cough and deep breathing. In the patient who has sustained prolonged intraoperative



Fig. 6. Completed aortic aneurysm graft repair.

hypotension (from either hemorrhage or declamping), and thus, has required significant transfusions and fluid, both adult respiratory distress syndrome (ARDS) and congestive failure may occur. Differentiation of these entities is difficult on clinical judgment alone and usually requires the added information obtained from a pulmonary artery (Swan-Ganz) catheter (6).

Acute renal failure complicating aortic aneurysm repair is associated with a greater than 50% mortality. Two principal causes are nephrotoxic agents (such as radiographic contrast agents or perioperative antibiotics) and ischemic injury. The latter is more common, and is typically characterized by oliguria, a rapid rise in the creatinine level, and electrolyte imbalance. Etiologies of ischemic injury include hypovolemia, prolonged renal artery clamp time, and atheroembolization (typically from injudicious use of the aortic clamp on a diseased aorta, with subsequent "extrusion" of atheromatous debris into the renal orifices). In rare instances, and particularly in surgery for large iliac artery aneurysms and "redo" aortic surgery, ureteral injury may occur, because of its location anterior to the iliac vessels (6).

A variety of gastrointestinal (GI) complications may be seen after aortic surgery. A prolonged return of GI function may be secondary to a duodenal ileus, as there is significant retraction of the duodenum in order to expose the proximal aorta. As previously

noted, the incidence of ileus, and need for prolonged nasogastric suction may be less with the retroperitoneal approach. Further cephalad dissection of the aorta also exposes the inferior border of the pancreas, which may be inadvertently injured, leading to a postoperative pancreatitis. With any type of supravisceral aortic surgery and mesenteric revascularization, either hepatic or small intestinal ischemic injury may occur, with resultant acidosis, leukocytosis, enzyme elevation, and even death (6).

Colon ischemia is a well-recognized complication of aortic surgery. The classic postoperative presentation is bloody diarrhea, fever, tachycardia, and leukocytosis. It is seen more commonly following aneurysm repair, and its incidence is highest in patients undergoing repair of a ruptured aneurysm. The pathophysiology is related to the inferior mesenteric artery (IMA) and collaterals (namely the meandering artery, a marginal artery of Drummond, and hemorrhoidal vessels from the internal iliac artery) which supply the left colon. In repair of an abdominal aortic aneurysm, the aneurysm always involves the IMA orifice, which is therefore ligated when the aneurysm sac is opened. The main blood supply to the left colon then becomes the meandering artery from the superior mesenteric artery (SMA) and the internal iliac artery. Colonic ischemia may thereby occur if there is a stenosis or occlusion of the SMA or internal iliac artery. If the colon appears dusky at the time of surgery, the IMA may be re-implanted into the aortic graft (6).

Aortoenteric fistula is a late complication following aortic graft replacement. The majority of these occur at the duodenal level, again due to the location of the duodenum just anterior to the perirenal aortic segment. Infection is usually an antecedent cause, with erosion of the graft into the fourth portion of the duodenum. A strong suspicion is essential for the diagnosis, especially in patients who present with unexplained upper gastrointestinal bleeding following aortic surgery (6).

Technical complications include peri-operative hemorrhage, either from suture lines or from inadvertent injury to surrounding veins, mesentery, or spleen. Graft thrombosis may also occur, again usually due to a technical error.

COST

Several recent studies have evaluated the cost associated with open and endovascular repair techniques. The average cost for an open AAA repair is \$12,000, whereas the average cost for an endovascular repair is \$21,000. Although the endovascular approach decreases those expenses related to ICU admissions and length of hospital stay, the overall cost is greater because of the price of the endograft itself, approx \$10,000. Furthermore, because there is a new complication of endoleak in some cases of endovascular repair, periodic CT scanning and additional secondary procedures drive up the long-term care costs (7).

SUMMARY

- 1. With an increase in the aging population, there is an expected increase in the number of patients diagnosed with and treated for aortic pathology. Their care is dependent on an adequate knowledge of the pathophysiology and anatomy of the disease, along with an awareness of the surgical technique for repair and potential for complications.
- The diseases of aorta and its branches can be categorized into aneurysmal or occlusive diseases. Both categories represent a more generalized disorder affecting cardiovascular system.

- 3. Aortic aneurysm and aortoiliac surgery are formidable operations and despite advances in the peri- and postoperative management, have significant mortality and morbidity.
- 4. The risk of AAA rupture increases as the size of AAA increase above 5 cm. Surgical repair is indicated in these patients. Depending upon the anatomy of the AAA, endovascular repair is an alternative to surgical repair. It has a lower complication rate but is more expensive than the surgical repair.

REFERENCES

- 1. Ernst C. Abdominal aortic aneurysms. N Eng J Med 1993;328:1167-1172.
- 2. Brewster DC. Current controversies in the management of aortoiliac occlusive disease. J Vasc Surg 1997;25:365–379.
- 3. Hines GL, Chorost M. Supraceliac aortic occlusion: A safe approach to pararenal aortic aneurysms. Ann Vas Surg 1998;12:335–340.
- D'Ayala M, Hollier LH, Marin ML. Cardiothoracic and Vascular Surgery: Endovascular grafting for abdominal aortic aneurysms. Surg Clin North Am 1998;78:845–860.
- 5. Jean-Claude JM, Reilly LM, Stoney RJ, et al. Pararenal aortic aneurysms: The future of open aortic repair. J Vasc Surg 1999;29:902–912.
- 6. Hermreck AS. Prevention and management of surgical complications during repair of abdominal aortic aneurysms. Surg Clin North Am 1989;69:869–894.
- 7. Sternbergh WC, Money SR. Hospital cost of endovascular versus open repair of abdominal aortic aneurysms: a multicenter study. J Vasc Surg 2000;31:237–244.

Edovascular Repair of Abdominal Aortic Aneurysm

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INTRODUCTION

Endovascular repair of abdominal aortic aneurysm (AAA) has exploded onto the public scene over the past 2 yr. It is important for the general internist to have a balanced working knowledge of this subject because: 1) AAA is very common; 2) many patients have heard about endovascular repairs, and will turn to their internists for advice; and 3) some of the centers offering endovascular grafting promote themselves in the media in ways that may not be scientifically balanced.

The pathophysiology and surgical repair of AAA is nicely covered in the previous chapter (*see* Chapter 22). Whereas most elective cases can be safely done by conven-

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tional "open" technique with excellent and durable outcome, it is fair to say that most of the morbidity and mortality of conventional open surgery relates to the need to open the abdominal cavity and to cross-clamp the aorta in order to accomplish the repair. It is important to note that many if not most patients with a surgical-sized AAA have concomitant vascular disease in coronary, cerebral vascular, renovascular, and/or peripheral vascular distributions, all of which may increase the risk of general anesthesia and open surgery.

Because of all of the above considerations, there has been a large research and commercial effort to develop techniques to repair AAA from a "less-invasive" endovascular approach. Essentially, the goal has been to deliver a vascular graft from inside the artery to cover and exclude the aneurysm, without cross-clamping the aorta or opening the abdomen, and ideally without general anesthesia. Early attempts at grafting were made with relatively crude homemade devices constructed from metallic vascular stents sewn to standard bypass graft material, but progress has been rapid. The earliest successful endovascular AAA repairs were accomplished around 1990 (1), commercial devices were widely available in Europe by 1995, and the FDA issued United States approval of two of the commercial endovascular AAA grafting systems in late 1999. Thus far, all announced commercial and investigational endovascular systems are constructed from a mix of metallic vascular stents and biocompatible cloth or polymer materials (PTFE, Dacron, and so on) The systems currently approved for use in the United States are:

- 1. The Ancure device, produced by the Guidant company. This is an evolution of the EVT device, and was the earliest commercial device to start trials. It is a one-piece design consisting of a conventional cloth Y-shaped bifurcated graft with stented attachments at each of its ends. The entire assembly is folded into a large-bore introducer, and advanced and positioned from a bi-femoral approach (2).
- 2. The AneurRx device produced by Medtronic. This is a modular device, consisting of a series of variously shaped metallic stents covered with cloth. These are placed sequentially from a bi-femoral approach in order to build up a Y-shaped graft inside the patient (3).

As of this writing, there are at least a half-dozen other systems in various states of development, many of them already commercially available outside the United States. All of the announced investigational devices are of the modular variety.

It should be noted that part of the FDA approval of the devices was a requirement that the manufacturers provide 2-d "hands-on" training courses to all operating physicians before selling them the grafts. This requirement set off a scramble among interested physicians to gain entry into the courses, and initially engendered a *de facto* segregation of the procedure to larger, higher volume centers, as the companies initially focused their attention on those physicians who were likely to purchase the largest number of grafts. By the time of this writing (2002), many community hospitals have achieved credentialing, and the number of institutions offering endografting is growing rapidly.

As we shall see in the following sections, endovascular repair of AAA is a promising technique, but is still early in its evolution, and entails considerable compromises relative to conventional repair. Despite the commercial availability of endograft systems, tremendous enthusiasm among vascular physicians for the technique, and the real benefit accrued by some high-risk patients, many AAAs cannot yet be repaired by endovascular techniques, and many others *probably should not be*. One supporting piece of evidence for this assertion is the sobering fact that each of these devices has had an

interval of nonavailability owing to regulatory issues arising *after* FDA approval. The regulatory climate is changing rapidly, and the reader is directed to the web address listed in ref. *11* for up-to-the-minute information. If what follows seems more an editorial than a scientific article, we ask the reader to understand this as an indication of the current state of the data.

Required Resources for Endovascular Grafting

Little in the way of specialized equipment is needed to place stent grafts. The procedure can be done in any institution with an operating room (OR), a modern c-arm fluoroscope with a basic angiographic package, and one or more qualified physicians. Most endovascular grafts are placed in the OR using a portable c-arm for imaging guidance, although some centers have constructed specialized angiography suites with OR-standard sterile environments, and a few have installed full-range angiography equipment in their operating rooms. Much of this is driven by "turf" issues within a given institution. Operating physicians come from the realms of Vascular Surgery, Interventional Radiology, and Interventional Cardiology, often working in multispecialty teams. In this author's opinion, the current ideal is a team consisting of a vascular surgeon and an interventional radiologist. This is the combination most likely to have the accumulated skills necessary to successfully address any unanticipated problems that occur during deployment of a graft. Whereas a routine procedure requires lowend surgical skills and medium catheter manipulation skills, the need for high-end surgical and/or catheter skills can develop very, very quickly during a graft deployment.

INDICATIONS AND ALTERNATIVES

It would be very easy to say that the procedure is indicated for anyone having an AAA larger than 5 cm who can be fitted for an endograft. However, there is much that is not known about the long-term durability of the devices, and the endoleak/endotension issue (see later) is far from settled. It is also important to understand that there has been a rush to the market with these devices, and the available controlled trials are not up to the task of defining indications for many patients. It is fair to say that at the current state of the art, many patients' suitability for endovascular rather than open repair is as much a matter of opinion and preference as it is a matter of science.

It is important to understand that the currently available devices simply will not fit a significant percentage of patients. The reasons for exclusion are numerous and generally involve an infrarenal neck that is too short or wide, an aneurysm that is too angulated or iliac arteries that will not permit passage of the introducer. Many investigators claim that as many as 40-60% of patients are anatomically suitable for endografting with the commercially available systems. In this author's experience, that number seems high, possibly relating to selection bias at the larger referral centers. The real number is probably closer to 30%.

HIGH-RISK PATIENTS

It is fair to say that endovascular grafting is definitely indicated for patients that have "surgical" sized AAA can be fitted with a device, and are at high risk for conventional repair because of concomitant coronary artery disease, COPD, or other co-morbidity.
HOSTILE ABDOMEN

Patients with surgically hostile abdomens caused by radiation, inflammatory bowel disease, adhesions, multiple surgeries, or other conditions that would increase the difficulty of an open repair, are good candidates for endografting.

FEMALE PATIENTS

Females generally are less likely to pass anatomic criteria for endografting, usually because of smaller iliac arteries. However, women who are successfully grafted probably do about as well as men (4).

INFLAMMATORY ANEURYSMS

These rare, but difficult cases were until recently a *terra incognita* for endografts. A recently published series of two patients suggest that endografting might not only exclude the aneurysm, but stop the inflammatory process (5). Given that the complication rate of conventional repair is considerable in inflammatory aneurysm, it is reasonable to consign these patients to endografting when anatomically possible.

YOUNG PATIENTS

As of this writing, the oldest implanted Ancure device of current design has been in place about 6 yr, and the oldest implanted AneuRx device for less than that. In my opinion, the current endografts should be considered to be of unknown and suspect durability for patients with long horizons, and those patients should be guided toward conventional repair.

GOOD OPERATIVE CANDIDATES

These patients constitute by far the largest group of AAA patients. It is currently difficult to say which of them should be treated by which method; decisions are currently driven by patient and physician choice.

CONTRAINDICATIONS

Inability to fit the AAA with a graft is by far the most common exclusionary factor for endografting.

Mycotic AAA

This should be treated with open excision and extraanatomic bypass in most cases because of the high likelihood of infection of an endograft. There have been a few reported cases of successful endografting of mycotic aneurysms in the thoracic aorta.

Acutely Ruptured AAA

Although there have been a few reported cases of successful endovascular repair of acute ruptures, this is not generally indicated. Sizing of grafts without preoperative work up is problematic, and the leaking aneurysm is not effectively sealed until the procedure is complete. The possibility also exists of worsening the leak by manipulating the large/ stiff equipment inside of the disrupted AAA. A further practical issue is that many

centers buy these expensive devices per patient, and do not have a depth of inventory to provide a device in an emergency.

TECHNIQUE

The discussion of technique is complicated by the fact that there are two different types of grafts (unibody vs modular), and three basic shapes (tube, aortouniiliac, and bifurcated). Readers are referred to the illustrations for further description.

Tube grafts were the majority of early devices used. They can be used only when there is a long usable segment of normal aorta distal to the aneurysm in which to secure the distal end of the graft. This is not a common circumstance, and only a few patients are candidates for tubes. An important consideration is that the part of the aorta comprising the infrarenal neck is physiologically stronger than the rest of the infrarenal aorta in that it has more elastin fibers in its wall. The distal landing zone for tube grafts is physiologically the same as the part of the aorta that became aneurysmal in the first place, and is, therefore, subject to expansion and weakening over time. There are reported cases of this leading to delayed leaks. Tube grafts are rapidly falling out of favor; most centers use them in specialized circumstances or not at all.

Aortouniiliac grafts are one-piece grafts that bridge the infrarenal neck through the AAA and into one common iliac artery. The opposite common iliac artery must be occluded (naturally or by intent) in order to cut it off from the AAA, and a femoral to femoral crossover graft is also required to supply the opposite leg. Aortouniiliac grafts were initially developed as a compromise solution for high-risk patients who needed endovascular treatment, and were not anatomically suitable for tube grafts. That application has been supplanted by the development of bifurcated systems. However, the aortouniiliac systems are still occasionally useful for patients who have only one iliac artery that would accommodate placement of a graft.

Bifurcated systems currently comprise the vast majority of cases being done. They bridge from the infrarenal neck into both common iliac arteries (Fig. 1). The following technical discussion is for a bifurcated system; keep in mind that the following is an average technical description and that variations on the theme are both legion and beyond the scope of this volume.

- 1. Access to both common femoral arteries is obtained via surgical cut-down.
- 2. Guidewires are manipulated from both sides to a point well above the neck of the AAA.
- 3. An angiogram is performed. The positions of all key structures (renal arteries, aortic bifurcation, iliac bifurcation, and so on) are marked.
- 4. The delivery device containing the graft (one-piece system) or the aortoiliac body of the graft (modular system) is advanced over one of the guidewires and positioned appropriately. In general, the side with the widest/straightest iliac system is chosen to be the "ipsilateral" one for this because the systems are big (18–28 fr), very rigid, and must be advanced with care.
- 5. Via the contralateral side, a snare catheter is advanced and used to capture the contralateral limb of the device (one-piece graft), or a catheter is left in place in the iliac system to use in deploying the contralateral component once the main body is deployed (modular).
- 6. The introducer sheath is retracted, deploying the aortic and ipsilateral iliac limb (both types). These are smoothed down and tacked into place using a balloon catheter.
- 7. If the graft is a unibody system, the contralateral limb is pulled into place, deployed, and ballooned. At this point, grafting is complete, and the AAA excluded. Proceed to step 9.



Fig. 1. Diagram of bifurcated endovascular aortic stent graft. The stent bridges from infrarenal aortic neck into both common iliac arteries.

- 8. If a modular system is being used, a steerable catheter is maneuvered through the contralateral iliac system and on into the aortoiliac body of the graft. (The graft has a cuff designed to accept the eventual contralateral limb.) The contralateral limb is then placed, bridging the main body of the graft with the contralateral common iliac artery.
- 9. Angiography is performed, and any necessary ancillary procedures are done. One-piece grafts are unsupported by stents as delivered, and often need to have stents placed within them to straighten out kinks. The modular systems come with a variety of extender cuffs that can be used to cover any immediate leaks.
- 10. The arteriotomies are closed, and the patient sent to recovery.

PREOPERATIVE IMAGING

Most of the preoperative work-up of a potential endograft patient revolves around imaging the AAA so that an appropriately sized graft is available at the time of operation, and that any potentially complicating anatomy (such as duplex renal arteries) is known beforehand. Keep in mind that the Ancure graft is custom ordered based on sizing derived from the preoperative imaging, and the AneuRx graft is built up out of a series of parts that come in a variety of lengths and diameters. In all but the highest volume centers, both types are typically ordered for a specific patient.

Most of the anatomic reasons to exclude patients are readily apparent on routine thin section contrast enhanced CT. CT angiography is becoming more of a standard as well, particularly as multidetector-row scanners and 3D-reconstruction capability become widely disseminated. Some centers will plan and perform a grafting procedure in the basis of CT/CTA alone. Catheter angiography is still performed at most centers preoperatively, and is the "gold standard" for imaging of aortic branch vessels. However, it is an invasive procedure, and some centers are going away from its routine use. Ultrasound is an excellent screening tool for AAA, but has little usefulness in planning of endovascular procedures. Intravascular ultrasound has attracted some recent press, but

has not garnered wide use. MRA is improving constantly, but has not caught on generally for this application. However, it can be extremely useful for patients with baseline renal insufficiency, as iodinated contrast is not needed for MRA.

ANCILLARY INTERVENTIONS

Occasionally, preoperative coil embolization is necessary to exclude a branch that the graft will later cross. Usually this involves a hypogastric artery, although inferior mesenteric arteries and very large lumbar arteries are also sometimes coiled.

POSTOPERATIVE IMAGING

The mainstay of postoperative imaging is CT scanning, as it is by far the most sensitive imaging test for endoleaks. Patients should be scanned within 48 h of receiving the graft, twice or more during the subsequent year, and at least yearly thereafter. Patients with endoleaks require more frequent scanning.

COMPLICATIONS AND THEIR MANAGEMENT

Morbidity and mortality can result from endovascular grafting as they can from open repair. It is fairly well established that in high-risk patients, endovascular grafting holds a safety advantage over open repair, but it has been much harder to establish an advantage in low-risk patients.

Open conversion refers to the abandonment of the endovascular approach in favor of a conventional open procedure. On intent to treat basis, this currently occurs acutely in 1-5% of cases. However, rates of late conversion do rise as the follow-up period increases, and ultimate rates are probably still unknown. In most cases, open conversion of a failed endograft procedure is technically more difficult than primary open repair.

Endoleaks are by far the most common and vexing complication of the procedure, occurring in some form in up to 25% of successfully grafted patients. Endoleaks occur when there is flow of blood into the aneurysm despite presence of an endograft (Fig. 2). There are four types.

- Type 1. Failure of the graft at an attachment site allowing blood flow around the graft into the aneurysm. These can be proximal or distal, and always require correction.
- Type 2. Blood flow into the aneurysm via one or more collateral vessels that connect to it. Most common culprits are the lumbar arteries and the inferior mesenteric artery. Overall, type 2 leaks are probably the most common. Although many of these close spontaneously, some do not, and their treatment is one of the major controversies in the field.
- Type 3. Failure of the graft itself. Blood flow into the aneurysm via a tear in its fabric or a disruption of the attachment sites between modular components. This latter mode of type 3 leak has been a particular problem with the AneuRx system, as it is fairly rigid, and does not conform well to changes in aortic shape brought about by decompression of the AAA. (Treated AAA shrinks longitudinally as well as in diameter.) There has been a modification in the system to make it less rigid.
- Type 4. Porosity leak. Leaking through the mesh of the graft fabric itself. These are generally self-limited, and are not an issue with the current commercial systems.



Fig. 2. Diagrammatic illustration of types of possible leaks with endografts. Type 1: Failure of the graft at an attachment site allowing blood flow around the graft into the aneurysm. Type 2: Blood flow into the aneurysm via one or more collateral vessels that connect to it. Type 3: Failure of the graft itself. Blood flow into the aneurysm via a tear in its fabric or a disruption of the attachment sites between modular components. Type 4: Porosity leaks through the mesh of the graft fabric itself.

There are essentially three possible responses to an endoleak:

- 1. Follow it. Many small- to medium-type 2s will close spontaneously.
- 2. Treat it by endovascular means such as adding an extension cuff for a type 1 leak, or embolizing the offending branch in a type 2 leak.
- 3. Perform open repair of the aneurysm. Sometimes the only safe or effective option.

Endotension is a term describing aneurysms that continue to grow in diameter without any radiographic evidence of an endoleak. There have been cases where AAAs showing this sign have been proven to have internal pressures approaching the systemic blood pressure. (i.e., the graft was providing no protection against rupture of the aneurysm)! Ruptures have been reported. It is considered likely that there are also "treated" aneurysms under endotension that do not grow right away, and are thus silently at risk of rupture (6).

Rupture of aneurysms have been reported in the presence of all types of grafts currently on the market in the US and Europe. This is very distressing, as the only reason to treat most AAAs in the first place is to prevent eventual rupture. Ruptures have been reported with and without identified endoleaks, with growing and nongrowing aneurysms, and in one case with a shrinking aneurysm. A recent report of seven delayed ruptures after AneuRx placement showed that five had no evidence of endoleak or aneurysm enlargement prior to the rupture, and probably experienced acute failure of graft fixation with sudden pressurization and rupture of the AAA (7). A recent midterm report on the UK RETA cohort gives a cumulative risk of rupture of 1.05% at 1 yr and 2.65% at 2 yr (8). The current FDA advisory refers to at least 25 known ruptures after AneuRx placement (9).

Mechanical problems with placement can occasionally lead to dissection or rupture of the iliac arteries. These can often be corrected by endovascular means, but occasionally require open surgery. Late mechanical problems occur as the aneurysm decompresses, shrinking both in diameter and length, either of which can put stress on graft components, which twist or kink as a result. This can lead to leaks or to limb occlusions. Often these problems can be corrected by endovascular means. Also, the grafts themselves can degenerate over time, leading to leaks or rupture.

Postimplantation syndrome is a poorly understood entity causing fever and pain for days to weeks after graft placement. It responds well to inflammatory drugs, and is not associated with increase in the white blood cell count. The postimplantation syndrome is always self-limited.

Infection is quite rare, occurring in less than 1% of cases thus far. Can require explanation of the graft. Mortality is low in most series, ranging from around 0-3%. Of note is a trend toward more mortality in earlier cases, and less in later cases in a given series. Endovascular grafting has a significant learning curve.

ALTERNATIVE PROCEDURE

All AAAs of diameter >5 cm need repair, and the alternatives are open vs endovascular grafting. Advantages and disadvantages of endovascular grafting are as follows:

ADVANTAGES

- 1. Much shorter hospital stays; as little as one night in uncomplicated cases
- 2. Reduced or nonexistent ICU stay.
- 3. Quicker recovery. Most of the recovery time from open surgery relates to the incision and dissection. The endovascular procedure is done through simple femoral cut-downs; patients are fully ambulatory the next day.
- 4. Safer for high risk patients.

DISADVANTAGES

- 1. Requires close follow-up, particularly in patients with endoleaks.
- 2. Unknown durability of devices. A special issue for young patients.
- 3. More expensive.
- 4. Late ruptures may be more of a risk than with conventional repair, and seem to become more of an issue over time.

COST

Much of the cost of the procedure is concentrated in the cost of the devices themselves, currently between \$10,000 and 15,000 per patient. Overall hospital cost of the procedure is approx \$21,000, as opposed to \$12,000 for conventional repair (10). With only two companies providing the grafts, there is unlikely to be any downward pressure on prices in the short run.

SUMMARY

- 1. Endovascular stent-grafting of AAA is technically feasible for one-third to one-half of all patients needing repair.
- 2. The scientific data supporting the technique is incomplete and inconsistent, with studies spanning a variety of continents, graft types, and data reporting standards.

3. Endovascular approach is the best alternative for *bona fide* high-risk patients. However, the scientific data does not allow for recommendations for the medium to low-risk AAA population to be made on an entirely rational basis. Therefore, one is left with explaining the alternatives carefully, and allowing patients to make choices.

REFERENCES

- 1. Parodi JC. Endovascular stent graft repair of aortic aneurysms. Curr Opin Cardiol 1997;12:396-405.
- 2. Moore W, Rutherford R. Transfemoral endovascular repair of abdominal aortic aneurysm: results of the North American EVT phase 1 trial EVT Investigators J Vasc Surg 1996;23:543–553.
- 3. Zarins C, White R, Schwarten D, et.al. AneuRx stent graft vs. open surgical repair of abdominal aortic aneurysms: multicenter prospective clinical trial. J Vasc Surg 1999;29:292–305.
- 4. Pena CS, Fan CM, Geller SC, et al. Endovascular stent graft repair of abdominal aortic aneurysms in female patients: Technical challenges and outcomes. Abstract Soc Cardiovasc Intervent Radiol 2001; Mar:3–8.
- 5. Cowie AG, Ashliegh RJ, England RE, et al. (2001) Endovascular repair of inflammatory aortic aneurysms. Abstract Soc Cardiovasc Intervent Radiol 2001;Mar:3–8.
- 6. White GH, May J, Petrasek P, et al. Endotension: An explanation for continued AAA growth after successful endoluminal repair J Endovasc Surg 1999;6:308–315.
- 7. Zarins CK, White RA. Fogarty TJ Aneurysm rupture after endovascular repair using the AneuRx stent graft. J.Vasc Surg 2000;31:960–970.
- 8. Thomas SM, Gaines PA, Beard JD. Midterm followup on 1000 patients on the UK registry of endovascular treatment of aneurysms (RETA). Abstract Soc Cardiovasc Intervent Radiol 2001;Mar:3–8.
- 9. FDA Safety Notification. http://www.fda.gov/cdrh/safety.html.
- 10. Stembergh C, Money S. Hospital cost of endovascular versus open repair of abdominal aortic aneurysms; a multicenter study. J Vasc Surg 2000;31:237–244.
- 11. Katzen B. The Guidant/EVT Ancure Device. JVIR 2000;11(suppl):62-69.

VIII SURGERY ON PORTAL VEIN

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Portasystemic Venous Shunt Surgery for Portal Hypertension

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INTRODUCTION

Portal hypertension, defined as sustained elevation of the portal pressure above 12 mmHg, can arise from a myriad of causes. In Western countries, the most common cause is alcoholic liver cirrhosis, whereas in Asia, and developing countries, it is postnecrotic cirrhosis (from viral hepatitis) and schistosomiasis. The adverse effects of chronic portal hypertension include the formation of esophageal, and extraesophageal varices, ascites, splenomegaly with hypersplenism, hepatorenal syndrome, and hepatic encephalopathy. Hemorrhage from gastroesophageal varices is the most lethal of these complications. Thus, its prevention and treatment has assumed paramount importance in the management of these patients.

The natural history of gastroesophageal varices in patients with cirrhosis is well established. About 25%–33% will bleed from the varices, mostly within the first year of

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diagnosis (1,2). The risk of recurrent bleeding with conservative management after the index bleed is about 30% at 6 wk, and approaches 70% at 1 yr (3). The mortality rate for each episode of bleeding approaches 50%. Therapy is, thus, aimed at the control of the acute episode of hemorrhage and the prevention of recurrent variceal bleeding. Portasystemic venous shunt surgery is one effective way of achieving the aforementioned therapeutic goals.

CLASSIFICATION AND HISTORICAL DEVELOPMENT

Shunts are classified as either non-selective or selective, depending on whether the entire portal circulation or only the gastroesophageal varices are decompressed. They are also categorized with respect to the ability to preserve prograde (hepatopetal) flow in the portal vein.

Total Shunts

Total shunts are nonselective, and divert the entire portal circulation away from the liver. The classic example is the portacaval fistula, first performed by Eck in dogs in 1877 (4). The portal vein was sutured side-to-side to the inferior vena cava, and the distal hepatic limb of the portal vein was ligated to ensure that all portal blood was diverted to the systemic circulation. Vidal performed the first successful portacaval anastomosis in man in 1903 (5). Widespread interest, however, only occurred after the seminal publication of successful portacaval shunting by Whipple (6).

A side-to-side portacaval anastomosis that is greater than 12 mm in diameter also functions like a total shunt, and in addition, the hepatofugal flow in the portal vein allows decompression of the liver sinusoids, alleviating ascites. Large-diameter interposition grafts (12–22 mm) placed between the portal vein or superior mesenteric vein, and the inferior vena cava, left renal vein, or right atrium also behave like total shunts, as does the proximal splenorenal shunt devised by Linton (7). Whereas total shunts may be effective in controlling variceal hemorrhage and ascites, the high incidence of encephalopathy (30%–40%) and progressive liver failure from diversion of hepatic portal flow has resulted in the loss of enthusiasm for performing these operations. Furthermore, any procedure that involves dissection of the liver hilum, such as a portacaval shunt, will complicate or even exclude the future possibility of liver transplantation, which is the definitive treatment for patients with end-stage liver disease.

Partial Shunts

Realizing the importance of preservation of hepatic portal flow in order to reduce the incidence of encephalopathy, and liver failure, the concept of partial shunting was actively investigated. Partial shunts are nonselective, decompressing the portal hypertension just enough to reduce variceal hemorrhage but maintaining adequate prograde portal blood flow. Initial attempts at creating a small side-to-side portacaval anastomosis (less than 12 mm) were unsuccessful as these dilated with time, with loss of hepatopetal flow (8). It was not until Sarfeh *et al.* reported their results with the use of small-diameter (8–10 mm) polytetrafluoroethylene (PTFE) portacaval H-grafts that true partial shunting became established (9,10). A relationship between shunt diameter, direction of portal flow, and incidence of encephalopathy was confirmed (11). Other authors have successfully adapted the use of small-diameter PTFE grafts at the mesocaval level in

order to avoid dissection of the hepatic hilum and potentially compromising future liver transplantation (12).

Selective Shunts

The original approach to preserve hepatopetal flow, and only selectively decompress the gastroesophageal varices was proposed by Warren in 1967 (13). Selective shunts maintain portal hypertension in the splanchnic bed, and only divert blood flow from the high-pressure gastroesophageal varices into the systemic circulation. The distal splenorenal or Warren shunt accomplishes this by way of drainage via the short gastric veins, and splenic vein into the left renal vein (end-to-side).

In contemporary practice, only the portal blood flow preserving procedures i.e., smalldiameter portacaval (PCS), mesocaval (MCS), and distal splenorenal shunts (DSRS) are still being performed and, therefore, form the basis of this chapter.

INDICATIONS

The indications for surgical portasystemic venous shunting can be classified as emergent or elective.

EMERGENT INDICATIONS

Emergent operations to control acute variceal hemorrhage have diminished, largely because of the high perioperative mortality, and the success of less invasive measures (vasopressin and nitroglycerine, octreotide, endoscopic sclerotherapy/banding and the transjugular intrahepatic portacaval shunt (TIPS) procedure). Endoscopic injection sclerotherapy can stop acute bleeding in about 90% of cases (14). When this fails, TIPS should be the second line of treatment because these patients often have advanced liver disease, and operative mortality is high in this subset (30-40%) (15). When the above measures have failed or are unavailable because of a lack of local expertise, surgery may be considered to prevent death from exsanguination. The procedure of choice is a smalldiameter MCS (or PCS) if the vascular anatomy is permissive, because rapid decompression of the varices is achieved, and the operation can be performed expeditiously. The DSRS is not appropriate in the emergent setting because it does not reliably produce immediate decompression of the varices, and is also a more time-consuming procedure. In patients who do not have a patent portal venous system, a devascularization procedure (e.g., the Sugiura operation) may be considered. Highly selected good-risk patients (Child-Pugh A, see Table 1) with acute variceal bleeding (particularly from gastric varices, which are less amenable to sclerotherapy) may be considered for surgical shunting once hemodynamic stability is achieved. Temporizing measures such as sclerotherapy or balloon tamponade may allow time for hepatic and other organ system recovery, reducing the subsequent surgical morbidity. In general, however, emergent shunt surgery is avoided in all but the most unusual circumstances.

ELECTIVE INDICATIONS

Elective operations (done in stable patients with no evidence of bleeding for at least 48 h) should only be performed in patients with adequate liver reserve (Child-Pugh Class A-B+). Those with advanced liver disease may be better served with liver transplanta-

Parameter	1 Point	2 Points	3 Points
Bilirubin (mg/dL)	<2	2–3	>3
Albumin (g/dL)	>3.5	2.8 - 3.5	<2.8
Increased Prothrombin time (s)	1-3	4–6	>6
Ascites	None	Slight	Moderate
Encephalopathy	None	1-2	3–4

Table 1 Child-Pugh Classification of Severity of Liver Disease

Grades: A = 5-6 points B = 7-9 points C = 10-15 points

tion, although donor organ shortage is a continual problem. For good-risk patients who have failed repeated sclerotherapy or those with gastric varices, surgical shunting is indicated for the prevention of recurrent variceal hemorrhage. Our preference is to perform the DSRS if the patient's anatomy is favorable, and if there is no significant ascites. The small-diameter MCS is a second alternative. We try to avoid the PCS in order to preserve the option of liver transplantation in the future.

CONTRAINDICATIONS

Surgical porta-systemic shunting should not be performed in patients with poor hepatic reserve (Child-Pugh B-C), active hepatitis, significant cardiopulmonary morbidity and cancer. As aforementioned, the presence of significant ascites is also a contraindication for the DSRS, because this procedure may exacerbate the condition.

PREOPERATIVE EVALUATION AND PREPARATION

The etiology of the portal hypertension should be determined because this has a direct impact on outcome. Patients with nonalcoholic cirrhosis (e.g., postnecrotic cirrhosis, primary biliary cirrhosis) and those with extrahepatic portal vein thrombosis or primary hepatic fibrosis do better, and have an improved survival after the DSRS than alcoholic cirrhotics (17,18). Because prognosis is directly related to liver functional reserve, the Child-Pugh class status should be assessed. Ideally, the nutritional status should be good, there should be no encephalopathy, the serum total bilirubin should be less than 2 mg/dL, the serum albumin greater than 3 g/dL, there should be no ascites and the prothrombin time should be no longer than 2 sec from the control. Ultrasound assessment of liver volume (between 1000 and 2500 mL) and a functional measurement of the liver reserve by means of the galactose elimination capacity (greater than 250 mg/min) will further aid the selection of good-risk patients for surgery. Careful evaluation of the patient's cardiopulmonary reserve and fitness to tolerate general anesthesia and a major abdominal operation is crucial.

Cirrhotic patients are at risk for the development of hepatocellular carcinoma. Screening involves an abdominal ultrasound or computed tomography (CT) scan and serum α -fetoprotein determination.

Evaluation of the vascular anatomy is performed prior to consideration for shunt surgery. Duplex ultrasound can determine the patency of the extrahepatic portal vein but visualization of the other vessels is limited. Magnetic resonance angiography (MRA) with gadolinium enhancement is a relatively new, noninvasive study that can provide information on the status of the superior mesenteric, portal, splenic, and the left renal veins. However, contrast angiography is still preferred by many surgeons for a definitive assessment of the vascular anatomy. Visualization of the inferior vena cava and left renal vein to ensure adequate patency, and measurement of the hepatic wedge pressure is performed. Selective injection of contrast is then made into the splenic and superior mesenteric arteries and images are taken during the venous phase of the study.

Optimal anatomic prerequisites for the DSRS include a patent, nontortuous splenic vein with a diameter of at least 10 mm, a short distance between the splenic and left renal veins (less than one vertebral body), and adequate drainage from the left renal vein into the inferior vena cava. If the patient had a splenectomy previously, or the splenic vein is small or thrombosed but a sizable superior mesenteric or portal vein is present, a small-diameter MCS or PCS may be performed. If the whole portal system is thrombosed, then a devascularization procedure (Sugiura operation) is considered.

Preparation of patients for elective surgery involves improving their nutritional status, optimizing cardiopulmonary function, and medical control of ascites when this is present. Electrolyte abnormalities should be corrected and abstinence from alcohol is encouraged. Patients with active liver disease, e.g., alcoholic hepatitis, and chronic active hepatitis have an increased mortality (16), and should not undergo surgery until this has been stabilized. A liver biopsy may be necessary to assess for disease activity. Perioperative antibiotics are given to reduce the risk of infection and prophylactic H2 blocker therapy is recommended for 4–6 wk. Significant coagulopathy is corrected with fresh frozen plasma and vitamin K before surgery.

OPERATIVE TECHNIQUE

Distal Splenorenal Shunt (DSRS or Warren Shunt)

There are two essential components to this operation. First, the pancreas is fully mobilized from the superior mesenteric vessels to the splenic hilum. This allows rotation of the gland and adequate visualization of the splenic vein. The vein is dissected out of the pancreatic groove, carefully ligating all the small pancreatic perforating tributaries. It is then divided flush with the portal vein and anastomosed end-to-side to the left renal vein without any tension or twist.

The second part is equally important and involves ligation of the left gastric or coronary vein, right gastric vein and the right gastroepiploic vein. This critical step preserves prograde flow in the portal vein, and confers selectivity to the shunt. Despite this, there is evidence that loss of hepatopetal flow occurs over time in alcoholic cirrhotics, and survival in this group is no better than that achieved by total portasystemic shunting (17,18). Collateral veins develop in the pancreas, which siphon blood away from the high-pressure portal vein to the low-pressure splenorenal anastomosis (Fig. 1). The additional maneuver of total spleno-pancreatic disconnection improves the selectivity of the DSRS, and maintains hepatopetal flow in the longterm (19,20). This is achieved by dividing the splenocolic ligament, and ensuring total mobilization of the splenic vein from the pancreas. The procedure is depicted in Fig. 2.

Mesocaval Shunt (MCS)

The superior mesenteric vein is exposed via a transverse incision at the base of the transverse mesocolon as the vein enters the root of the mesentery of the small bowel. The



Fig. 1. The pancreatic siphon after distal splenorenal shunt.



Fig. 2. Distal splenorenal shunt with spleno-pancreatic disconnection.



Fig. 3. Small-diameter meso-caval shunt.

inferior vena cava is exposed directly through the right transverse mesocolon. An 8-mm ringed PTFE graft is sewn on the anterior surface of the vena cava, tunneled through the mesocolon, and then sewn to the antero-lateral aspect of the superior mesenteric vein. An important maneuver is to completely mobilize the third and fourth portions of the duodenum including the ligament of Treitz to allow the duodenum to ride up and avoid potential compression by the interposed graft. Collateral veins are not ligated. This is illustrated in Fig. 3.

Portacaval Shunt (PCS)

Exposure of the inferior vena cava and portal vein is initially achieved by wide mobilization of the C-loop of the duodenum, and head of the pancreas medially (an extended Kocher maneuver). Sufficient dissection of the anterior surface of the vena cava and lateral aspect of the portal vein is performed to facilitate the performance of the anastomoses. An 8-mm ringed PTFE graft is used as the conduit to join the two structures (Fig. 4). The use of supported grafts prevents kinking, and compression by adjacent viscera. Some authors feel that the ligation of portal collateral veins is important to divert more blood flow toward the liver, and the small diameter shunt, increasing the likelihood of preserving prograde portal flow (21). There is no universal agreement on this. If this is chosen, the umbilical vein is divided at the liver edge. The gastroepiploic, peri-esophageal, coronary and inferior mesenteric veins are also ligated.

POSTOPERATIVE COMPLICATIONS

Better selection of good-risk patients (Child-Pugh A, B+, nonemergent operations, good cardiopulmonary reserve) for elective surgical shunting has resulted in markedly improved operative mortality rates of less than 5% (22).



Fig. 4. Small-diameter portacaval shunt.

The overall postoperative morbidity averages about 30%. Complications include perioperative bleeding requiring multiple transfusions, postoperative ascites (including chylous ascites), pancreatitis from operative trauma to the gland, sepsis, and portal vein thrombosis. Specific complications from portasystemic shunting include postoperative hepatic encephalopathy, deterioration of liver function, and recurrent variceal hemorrhage.

Worsening of ascites following DSRS is due to the maintenance of portal hypertension, and the interruption of retroperitoneal lymphatics. Most surgeons feel that the presence of significant ascites is a contraindication for this procedure; the small-diameter MCS or PCS may be a better alternative. Medical management is successful in most cases: sodium restriction before and after surgery, using fresh frozen plasma or salt-poor albumin for maintaining plasma volume, spironolactone for diuresis, and restricting dietary fat to 30 g/d to minimize the risk of chylous ascites. Refractory cases may require paracentesis or rarely peritoneo-venous shunting.

Hepatic encephalopathy is defined as mental confusion related by the patient or family, or the detection of disorientation and asterixis by the physician. Subclinical encephalopathy, which is part of the spectrum, is characterized by elevated blood ammonia levels, electroencephalographic changes and abnormal psychometric tests. The emergence of postshunt encephalopathy occurs over time, necessitating close follow-up of patients after surgery. The incidence of this complication has been reduced with recognition of the importance of preserving hepatic portal blood flow, and avoiding shunt surgery in patients with severe liver dysfunction. Prospective, randomized studies have shown that portal blood flow preserving procedures have a reduced incidence of encephalopathy, and improved survival compared to total shunts (23,24). Treatment of hepatic encephalopathy includes control of precipitating factors (sepsis, electrolyte abnormalities, and hypovolemia), restricting protein intake to 40 g/d, use of oral neomycin and lactulose, and in refractory cases, ligation of the shunt.

Recurrent variceal bleeding after portasystemic shunt surgery occurs in less than 10% of cases, and is usually caused by shunt thrombosis. There is evidence to suggest that prosthetic grafts have a higher thrombosis rate compared to autogenous splenorenal

shunts (25). Clinical manifestations of a thrombosed DSRS include left upper quadrant pain, splenomegaly, ascites, and recurrent variceal formation. When identified early (within 2 wk of occurrence), it may be salvaged by thrombolytic therapy or reoperation. Early rebleeding after DSRS may also occur despite a patent shunt, and is caused by inadequate decompression of the varices. Development of an adequate outflow (via the short gastric, splenic, left renal veins, and the inferior vena cava) for complete gastroe-sophageal decompression may take up to 4–6 wk in some patients. One study showed that 24% of patients had inadequate immediate decompression 1–2 wk after DSRS (26). Hence, the use of this shunt in the acute setting of active variceal hemorrhage is controversial. Management of rebleeding in such patients should be nonoperative: vasopressin, balloon tamponade, and sclerotherapy. Prosthetic shunts also have a higher likelihood of causing portal vein diameter reduction or thrombosis compared to DSRS (25).

RESULTS OF PORTASYSTEMIC SHUNT SURGERY

Evaluation of the efficacy of each type of shunt should take into consideration the operative mortality and morbidity, rates of recurrent bleeding, and shunt thrombosis, risk of postoperative encephalopathy, and long-term survival. In general, by selecting good-risk patients and preferentially employing only portal blood flow preserving procedures under elective circumstances, 30-d operative mortality rates have greatly improved (about 5% or less) in reported series.

All three types of shunts are equally effective in preventing recurrent variceal hemorrhage. The DSRS is technically more demanding because of the extensive dissection required with a higher risk of bleeding and pancreatitis. It is also attended by a higher incidence of postoperative ascites. Furthermore, not every patient has the suitable vascular anatomy for this procedure. When successfully performed, however, the long-term results are excellent. Control of variceal bleeding is achieved in 88–97% of patients and late shunt thrombosis occurred in only 2% of patients who were followed by serial angiography (27). The highest risk of variceal rebleeding is during the first month after DSRS (about 10-15%), possibly because of shunt thrombosis or delayed decompression of the varices as aforementioned (28). The rate of hepatic encephalopathy is also lower compared to the small-diameter MCS and total shunts, owing to preservation of hepatopetal flow (25). Data from the Emory randomized trial comparing selective vs total shunts showed encephalopathy rates of 5% at 2 yr, 12% at 3-6 yr, and 27% at 10-yr followup in the selective shunt group (17). At all time intervals, this incidence was significantly lower than in patients randomized to total shunts. The available data suggest that DSRS does not significantly accelerate the natural history of the underlying liver disease. Longterm maintenance of portal flow is achieved in 90% of nonalcoholic patients but in only 25 to 50% of alcoholic patients due to the development of transpancreatic collaterals that siphon blood away (29). The important maneuver of total splenopancreatic disconnection in addition to the standard DSRS improves this to 84% in alcoholic cirrhotics (30). Long-term survival and quality of life are also improved in good-risk patients undergoing the DSRS, especially in patients with preserved liver function (31). Such patients may never need liver transplantation, and the only life-threatening problem they face is variceal hemorrhage.

Of the narrow-diameter PTFE porta-systemic shunts, 8-mm grafts maintain prograde portal flow in 80% of patients and are associated with about a 10% postoperative

encephalopathy rate (compared with 50 and 20%, respectively with 10-mm grafts) (21). The largest experience with these small 8-mm portacaval shunts showed an operative mortality of 6.3%, variceal rebleeding rate of 3.3% (after mean follow-up of 43 mo) and preservation of hepatopetal flow in 90% of patients perioperatively (32). At 1 yr, 12% of patients had reversal of portal flow; and the overall incidence of encephalopathy was 13%. A major problem with prosthetic grafts in the portal system had been the higher incidence of graft thrombosis, which has been reduced with the use of supported PTFE grafts. In the studies using small-diameter ringed PTFE grafts, the perioperative graft thrombosis rate was about 5-8% (32-34). Cumulative long-term patency rates of the shunts (primary and secondary) have been good at greater than 90%. The only prospective, controlled, non-randomized trial comparing the DSRS vs the 10-mm PTFE MCS, however, showed significantly higher shunt thrombosis and encephalopathy rates in the MCS group (25). Therefore, despite the good results achieved with the small-diameter PTFE MCS, we believe that DSRS is the preferred surgical option for elective decompression of gastroesophageal varices if the patient has appropriate anatomy. There is probably no advantage with respect to shunt patency by placing the graft at the portacaval level; hence, PCS should be avoided as it does complicate liver transplantation in the future.

CURRENT ROLE OF SURGICAL SHUNTS

The management of a complex disease such as portal hypertension requires a multidisciplinary effort, and each specialist has a defined role and contribution: gastroenterologist (overall management of the liver disease, medical management of variceal hemorrhage, sclerotherapy or variceal banding), vascular / transplant surgeon (surgical shunting, liver transplantation), and interventional radiologist (TIPS). The treatment of each patient should be selected based on the severity of the underlying liver disease, the amount of functional liver reserve and his life expectancy. There is little doubt that patients with severe liver dysfunction should not undergo surgical porta-systemic shunting as a high likelihood of perioperative mortality, and encephalopathy can be expected. Such patients are best managed with sclerotherapy or TIPS as a bridge to an eventual liver transplantation. If the patient is not a transplant candidate, TIPS may still be an effective palliation for the short life expectancies of these patients. In the acute setting of active hemorrhage, sclerotherapy, and TIPS are better options with lower morbidity and mortality compared to emergency surgical shunts.

For the prevention of recurrent bleeding in good-risk patients (Child-Pugh A-B, stable liver disease, long life expectancy), the alternatives are between sclerotherapy, surgical shunting or TIPS. DSRS has been compared with sclerotherapy in four randomized, prospective trials and the overall outcome has been summarized in a meta-analysis study (35). Bleeding control was significantly better after DSRS than sclerotherapy in all the studies and the rate of hepatic encephalopathy was essentially identical. Rebleeding after sclerotherapy is high at 40–60% in most long-term series (36). Although this can often be effectively treated by repeat endoscopic sclerosis or banding, the eventual failure rate (defined as death from recurrent bleeding or need for alternative treatment) is as high as 40% (37). Sclerotherapy is acceptable as a first-line treatment only if the patient is compliant as multiple sessions, and close follow-up for life are required. Most impor-



Fig. 5. Algorithm for management of bleeding gastroesophageal varices.

tantly, an effective salvage procedure for sclerotherapy failures is needed so as not to compromise on patient survival. Therefore, the patient has to have rapid access to a tertiary care facility in the event of recurrent hemorrhage.

In most institutions, TIPS has replaced surgical shunts for sclerotherapy failures, even in good-risk patients. The effectiveness of this strategy is unproven, and is currently undergoing investigation. The long-term results of surgical shunts are well established, and are presented earlier. The disadvantages of TIPS are the high rates of shunt stenosis or thrombosis (50% failure rate at less than 1-yr follow-up) (38) with increased recurrent variceal bleeding, and the need for frequent follow-up studies and reintervention. Furthermore, TIPS functions like a non-selective shunt with encephalopathy rates similar to the portacaval shunt. There have been one prospective, randomized trial (39) and two case-control studies (40,41) comparing surgical shunts (DSRS or small-diameter PCS) with TIPS in good-risk cirrhotic patients. All showed a significantly lower incidence of recurrent hemorrhage, encephalopathy, shunt occlusion and post-procedure death in patients undergoing surgical shunts. Current evidence therefore supports the use of surgical shunts for the prevention of recurrent variceal bleeding in good-risk patients, either primarily or following failure of sclerotherapy.

COST

In a decision-analysis comparing TIPS versus DSRS, it was shown that DSRS is a more cost-effective treatment for good-risk (Child's A) cirrhotics (42). The cost for a TIPS procedure in a patient with a predicted life expectancy of 1.96 yr averaged \$41,700, compared to \$26,900 for DSRS in a patient with a predicted life expectancy of 1.86 yr. Patients undergoing TIPS had prohibitively greater expenditures of almost \$150,000 per life-year saved, mainly because of the need for frequent ultrasound surveillance, and the high incidence of shunt dysfunction.

SUMMARY

- 1. The current excellent results for surgical porta-systemic shunting have been achieved by restricting surgery only to good-risk patients and performing the operations in an elective setting.
- 2. Such patients include those with good hepatic reserve (Child-Pugh A, B+), nonalcoholic cirrhosis, extrahepatic portal vein thrombosis, and hepatic fibrosis.
- 3. DSRS confers an improved survival and quality of life, and is more cost-effective compared to repeated sclerotherapy or TIPS.
- 4. A majority of these patients will not require liver transplantation and the DSRS provides a durable, definitive treatment for their portal hypertension, eliminating the threat of death from hemorrhage. Our approach to the management of patients with bleeding varices is illustrated in Fig. 5.

REFERENCES

- 1. De Francis R, et al. The North Italian Endoscopic Club for the Study and Treatment of Esophageal Varices. Prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices: a prospective multicenter study. N Engl J Med 1988;319:983–989.
- 2. Gores GJ, Wiesner RH, Dickson ER, et al. Prospective evaluation of esophageal varices in primary biliary cirrhosis: development, natural history and influence on survival. Gastroenterology 1989;96: 1552–1559.
- 3. Graham D, Smith JL. The course of patients after variceal hemorrhage. Gastroenterology 1981;80: 800–809.
- Child CG. Eck's fistula. Surg Gynecol Obstet. 1953;96:375–376. Originally published in Russian: Eck NV. K voprosu o perevyazkie vorotnois veni: predvaritelnoye soobshtshjenye. Voen Med J. 1877; 130:1–2.
- Donovan AJ, Covey PC. Early history of the portacaval shunt in humans. Surg Gynecol Obstet. 1978;147:423–430. Originally published in French: Vidal. Traitment chirurgical des ascites dans les cirrhoses du foie. 16th Cong Fr Chir 1903;16:294–304.
- 6. Whipple AO. The problem of portal hypertension in relation to the hepatosplenopathies. Ann Surg 1945;122:449–475.
- 7. Linton RR, Jones CM, Volwiler W. Portal hypertension: the treatment by splenectomy and splenorenal anastomosis with preservation of the kidney. Surg Clin North Am 1947;27:1162–1170.

- 8. Bismuth H, Franco D, Hepp J. Portal-systemic shunt in hepatic cirrhosis: does the type of shunt decisively influence the clinical result? Ann Surg 1974;179:209–218.
- 9. Sarfeh IJ, Rypins EB, Conroy RM, et al. Portacaval H-graft: relationships of shunt diameter, portal flow patterns and encephalopathy. Ann Surg 1983;197:422–426.
- Rypins EB, Mason GR, Conroy RM, et al. Predictability and maintenance of portal flow patterns after small-diameter porta-caval H-grafts in man. Ann Surg 1984;200:706–710.
- Sarfeh IJ, Rypins EB. Partial versus total portacaval shunt in alcoholic cirrhosis. Results of a prospective, randomized clinical trial. Ann Surg 1994;219:353–361.
- 12. Paquet KJ, Lazar A, Koussouris P, et al. Mesocaval interposition shunt with small diameter polytetrafluoroethylene grafts in sclerotherapy failure. Brit J Surg 1995;82:199–203.
- 13. Warren WD, Zeppa R, Fomon JJ. Selective trans-splenic decompression of gastroesophageal varices by distal splenorenal shunt. Ann Surg 1967;166:437–455.
- 14. D'Amico G, Pagliaro L, Bosch J. The treatment of portal hypertension: a meta-analytic review. Hepatology 1995;22:332–354.
- 15. van der Vliet JA, De Visser E, Buskens FGM. Changing pattern of portasystemic shunt surgery. Eur J Surg 1995;161:877–880.
- McCormick PA, Burroughs AK. Relation between liver pathology and prognosis in patients with portal hypertension. World J. Surg 1994;18:171–175.
- 17. Millikan WJ, Warren WD, Henderson JM, et al. The Emory prospective randomized trial: selective versus nonselective shunt to control variceal bleeding: ten-year follow-up. Ann Surg 1985;201:712–722.
- 18. Warren WD, Millikan WJ, Henderson JM, et al. Ten years portal hypertensive surgery at Emory: results and new perspectives. Ann Surg 1982;195:530–542.
- 19. Inokuchi K, Beppu K, Koyanagi N, et al. Exclusion of nonisolated splenic vein in distal splenorenal shunt for prevention of portal malcirculation. Ann Surg 1984;200:711–717.
- Warren WD, Millikan WJ, Henderson JM, et al. Splenopancreatic disconnection. Improved selectivity
 of distal splenorenal shunt. Ann Surg 1986;204:346–355.
- 21. Sarfeh IJ, Rypins EB, Mason GR. A systematic appraisal of portacaval H-graft diameters. Clinical and hemodynamic perspectives. Ann Surg 1986;204:356–363.
- 22. Orozco H, Mercado MA. The evolution of portal hypertension surgery. Lessons from 1000 operations and 50 years' experience. Arch Surg 2000;135:1389–1393.
- 23. Sarfeh IJ, Rypins EB. Partial versus total portacaval shunt in alcoholic cirrhosis. Results of a prospective, randomized clinical trial. Ann Surg 1994;219:353–361.
- 24. Millikan WJ, Warren WD, Henderson JM, et al. The Emory prospective randomized trial: selective versus nonselective shunt to control variceal bleeding. Ten-year follow-up. Ann Surg 1985;201:712–722.
- 25. Mercado MA, Morales-Linares JC, Granados-Garcia J, et al. Distal splenorenal shunt versus 10-mm low-diameter mesocaval shunt for variceal hemorrhage. Am J Surg 1996;171:591–595.
- 26. Richards WO, Pearson TC, Henderson JM, et al. Evaluation and treatment of early hemorrhage of the alimentary tract after selective shunt procedures. Surg Gynecol Obstet 1987;164:530–536.
- 27. Henderson JM, Millikan WJ, Galloway JR. The Emory perspective of the distal splenorenal shunt in 1990. Am J Surg 1990;160:54–59.
- 28. Richards WO, Pearson TC, Henderson JM, et al. Evaluation and treatment of early hemorrhage of the alimentary tract after selective shunt procedures. Surg Gynecol Obstet 1987;164:530.
- 29. Henderson JM, Gong-Liang J, Galloway J, et al. Portaprival collaterals following distal splenorenal shunt: incidence, magnitude and associated portal perfusion changes. J Hepatol 1985;1:649–661.
- 30. Henderson JM, Warren WD, Millikan WJ, et al. Distal splenorenal shunt with splenopancreatic disconnection: a four year assessment. Ann Surg 1989;210:332–341.
- 31. Orozco H, Mercado MA, Takahashi T, et al. Survival and quality of life after portal blood flow preserving procedures in patients with portal hypertension and liver cirrhosis. Am J Surg 1994;168:10–14.
- 32. Collins JC, Rypins EB, Sarfeh IJ. Narrow-diameter portacaval shunts for management of variceal bleeding. World J Surg 1994;18:211–215.
- 33. Paquet KJ, Lazar A, Koussouris P, et al. Mesocaval interposition shunt with small-diameter polytetrafluoroethylene grafts in sclerotherapy failure. Brit J Surg 1995;82:199–203.
- 34. Rosemurgy AS, McAllister EW, Kearney RE. Prospective study of a prosthetic H-graft portacaval shunt. Am J Surg 1991;161:159.
- 35. Spina GP, Henderson JM, Rikkers LF, et al. Distal splenorenal shunt versus endoscopic sclerotherapy in the prevention variceal bleeding: a meta-analysis of 4 randomized clinical trials. J Hepatol 1992; 16:338.

- 36. Snady H. The role of sclerotherapy in the treatment of esophageal varices: personal experience and a review of randomized trials. Am J Gastroenterology 1987;82:813–822.
- 37. Rikkers LF, Jin G, Burnett DA, et al. Shunt surgery versus endoscopic sclerotherapy for variceal hemorrhage: late results of a randomized trial. Am J Surg 1992;165:27–33.
- 38. Sanyal AJ, Freedman AM, Luketic VA, et al. The natural history of portal hypertension after transjugular intrahepatic portasystemic shunts. Gastroenterology 1997;112:889–898.
- Rosemurgy AS, Goode SE, Zwiebel BR, et al. A prospective trial of transjugular intrahepatic portasystemic stent shunts versus small-diameter prosthetic H-graft portacaval shunts in the treatment of bleeding varices. Ann Surg 1996;224:378–386.
- 40. Khaitiyar JS, Luthra SK, Prasad N, et al. Transjugular intrahepatic portasystemic shunt versus distal splenorenal shunt a comparative study. Hepato-gastroenterology 2000;47:492–497.
- 41. Helton WS, Maves R, Wicks K. Transjugular intrahepatic portasystemic shunt versus surgical shunt in good-risk cirrhotic patients. Arch Surg 2001;136:17–20.
- 42. Zacks SL, Sandler RS, Biddle AK, et al. Decision-analysis of transjugular intrahepatic portasystemic shunt versus distal splenorenal shunt for portal hypertension. Hepatology 1999;29:1399–1405.

Transjuglar Intrahepatic Portosystemic Shunt

Grant J. Price, MD

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INTRODUCTION

Portal venous hypertension is a common end point of most chronic diseases of the liver. Portal hypertension can be caused less frequently by obstruction of the main portal vein by thrombus or tumor or by obstruction of hepatic venous outflow from the liver (Budd-Chiari syndrome). However, most cases of portal hypertension seen in internal medicine practices are caused by alcoholic cirrhosis or advanced chronic viral hepatitis. The most common deadly consequence of severe portal hypertension is bleeding from varices of the esophagus and stomach, but other sequelae such as intractable ascites are common and difficult to treat medically.

The transjugular intrahepatic portosystemic shunt (TIPS) procedure is a percutaneous method for decompression of the portal venous system (1). The TIPS consists of a stented tract placed inside the liver between the portal venous system and the systemic venous circulation, usually between the right portal vein and the right hepatic vein. This shunt allows flow of blood around the diseased liver, effectively lowering the portal pressure and alleviating many of the sequelae of portal hypertension. In the 10 yr since the

From: *Clinical Gastroenterology: An Internist's Illustrated Guide to Gastrointestinal Surgery* Edited by: George Y. Wu, Khalid Aziz, and Giles F. Whalen © Humana Press Inc., Totowa, NJ introduction of the TIPS procedure, it has virtually replaced surgical portosystemic shunts in most centers.

In the United States, TIPS is performed almost exclusively by interventional radiologists. Procedures are almost always done within a radiology department. Required equipment consists of an angiography table with digital subtraction capabilities, a selection of standard catheters, guidewires, balloon catheters and stents, a physician qualified to perform the procedure, and an intensive care unit appropriate for the preand postoperative care of these often quite ill patients. Although the procedure is offered at essentially all University and tertiary centers, it is also quite commonly done in community hospitals. (This author has been performing TIPS in a community hospital since early 1992). Case volumes have exploded nationally; and are currently in the many thousands per year.

Elective TIPS on stable patients can usually be performed on a one-night admission, although longer stays can sometimes be necessary. Outpatient procedures can be done on occasion, although in the author's experience this has usually involved a semicompliant patient who refused to stay overnight. Emergency TIPS on acutely bleeding patients will involve a longer stay due to the poor overall condition of most such individuals.

INDICATIONS

Treatment and Prevention of Variceal Bleeding

This is by far the most common indication for the TIPS procedure. Timing of TIPS for this indication is somewhat controversial, depending on overall condition of the patient, candidacy for liver transplant, and institutional availability of alternative methods for treating variceal bleeding. However, it is important to remember that while TIPS can be performed with good results on very ill and unstable patients who are actively bleeding, there is a very large difference in outcomes of emergency vs elective TIPS. Consequently, most interventional radiologists would advocate creation of the TIPS *before* the catastrophic bleed or after the patient has been stabilized following a bleed. In the author's center, for instance, the 30-d mortality of patients brought for elective TIPS done for prevention of variceal bleeds is zero, whereas the 30-d mortality of patients receiving TIPS while hypotensive, intubated, and actively bleeding approaches 50%. All of this being said, TIPS is a highly effective treatment for variceal bleeding. Rebleeds occur in 4-17%, and most of these occur in patients whose shunts have become compromised (2,3).

Intractable Ascites

TIPS is a very effective therapy for intractable ascites as a result of portal hypertension, and leads to improvement or resolution in most patients. However, shunting is indicated only when medical management fails. One should keep in mind that patients undergoing TIPS for this indication are almost always in the very late stages of their liver disease. TIPS has an unpredictable effect on liver function in such patients, and can lead to accelerated encephalopathy or frank liver failure. Conversely, liver function can paradoxically improve.

Intractable Pleural Effusions

There is considerably less data for this indication. However, on the basis of the available experience, it would seem that effectiveness of shunt for pleural effusions is similar to that for ascites. Again, these patients are often fragile with tenuous liver function, and TIPS should be done only when medical management fails.

Hepatorenal Syndrome

The literature consists mainly of case reports with variable but generally positive outcomes. Given the generally poor outcomes experienced with conservative management of these patients, TIPS should be considered for any otherwise salvageable patient suffering from hepatorenal syndrome.

Budd-Chiari Syndrome

In the very earliest going, Budd-Chiari syndrome was considered a relative contraindication to TIPS because of the concomitant restricted access to the hepatic veins. However, as techniques have improved, TIPS has become a primary mode of therapy for portal hypertension related to Budd-Chiari (*see* "Advanced Techniques") (4).

As a Bridge to Transplantation

Because it does not disturb native anatomy, TIPS is greatly preferable to surgical shunt in patients who are candidates for liver transplants and need portal decompression for any of the above reasons. Care is taken in these patients not to extend the stents any farther into the main portal vein than absolutely necessary (5).

CONTRAINDICATIONS

Right heart failure is an absolute contraindication to TIPS, as these patients respond badly to arrival of high-volume portal venous flow directly into their compromised right hearts. Fatal cases of congestive heart failure have occurred. Severe hepatic encephalopathy or liver failure is a relative contraindication to TIPS, as the shunt decreases intrahepatic portal venous flow in most patients, potentially diminishing already compromised function. That being said, TIPS is often performed in this patient population on a "nothing to lose" basis, as it is sometimes the only available therapy for a severely encephalopathic patient who is also bleeding. Whereas untreatable encephalopathy, liver failure, and death sometimes do result, many patients do surprisingly well, and seemingly unsalvageable patients can sometimes be salvaged. Severe coagulopathy caused by liver dysfunction is unfortunately common in this patient population, but is only a relative contraindication to TIPS. The procedure can be performed safely in patients with coagulopathy or thrombocytopenia by careful attention to technique; capsular punctures are to be avoided. Patients who are in DIC at the time of shunt rarely do well. Cavernous transformation of the portal vein can preclude TIPS, but can sometimes be overcome by transhepatic access into an intrahepatic portal vein branch followed by recanalization of the main portal vein via PTA and stent. Although most patients with cavernous transformation are well enough collateralized to avoid sequelae of portal hypertension; those needing shunt should be referred to an experienced operator.



Fig. 1. Portal and hepatic venous anatomy.

TECHNIQUE

The following is a basic step-by step description of the procedure. All moves are made with fluoroscopic guidance. The reader may find the illustrations helpful in visualizing the below (Fig. 1) shows the relevant anatomy (6,7).

- 1. The right internal jugular vein is accessed percutaneously, and a large-bore vascular sheath is advanced through the right atrium into the upper inferior vena cava (IVC).
- 2. The right hepatic vein is accessed with a steerable angiographic catheter, and a hepatic venogram performed (Fig. 2).
- 3. The catheter is advanced peripherally in the hepatic vein, and a wedged venogram performed in an attempt to opacify the portal venous system. Some operators use CO_2 for this. Some experienced operators (including this author) omit this step (Fig. 3).
- 4. The sheath is advanced into the hepatic vein. A long, curved cutting needle is advanced over a guidewire inside the sheath until its tip rests in the hepatic vein.
- 5. The guidewire is removed, the needle tip is unsheathed, and the needle advanced into the hepatic parenchyma. Passes are made until the needle tip finds an intrahepatic branch of the portal vein. Care is taken not to pass the needle through the liver capsule. Great care is also taken that the hepatic venous and portal venous ends of the final needle position are both intrahepatic. Finding a usable needle position in the portal tree is generally the most difficult step in the procedure.
- 6. A guidewire is passed through the needle into the main portal vein, creating a connection between the hepatic vein and the portal vein. A wire or catheter is left in place through this tract from now until the end of the procedure (Fig. 4).
- 7. Pressures are measured in the main portal vein and in the upper inferior vena cava in order to establish the baseline portosystemic gradient. A portogram is then performed to outline the portal system for shunt, and to identify any high-flow varices that are present. Any important stenoses or occlusions in the portal system are also identified and corrected.



Fig. 2. Catheterization of the hepatic vein.

- 8. An angioplasty balloon is positioned across the intrahepatic tract and inflated to expand the tract to usable size.
- 9. A metallic vascular stent is placed across the newly created tract. Most available stent designs have been successfully used (Fig. 5) (8).
- 10. A second portogram is obtained to evaluate the morphology of the newly created shunt and any changes in the flow pattern of the portal system (Fig. 6). Additional stents are placed if needed.
- 11. Pressure measurements are obtained in the portal vein and IVC. The gradient between them should be no more than 10–12 mmHg at this point. If the gradient is significantly higher than 10–12 mmHg, the shunt is expanded with a larger balloon, or a second "parallel" TIPS is created.
- 12. Once acceptable hemodynamic result is achieved, the sheath is exchanged for a large bore catheter which is left in place at SVC or right atrial level until the patient is stable and follow-up Doppler confirms shunt patency.

ADVANCED TECHNIQUES

TIPS have been successfully created through occluded hepatic veins, thrombosed portal veins, and tumor-occluded portal veins. Techniques for accessing difficult portal anatomy have included shunt into left, middle, or caudate portal veins, portal access via transhepatic or transsplenic punctures, minilaparotomy allowing portal access via jejunal veins, and portal access through enlarged umbilical veins (9). Techniques to avoid difficult hepatic venous anatomy include use of the left and middle hepatic veins,



Fig. 3. Wedged hepatic venous portagram.

shunt directly to the intrahepatic IVC, stent of stenotic or occluded hepatic veins, and transhepatic access to intrahepatic branches of the hepatic vein. Techniques to overcome absence of the right internal jugular vein include access from the right external jugular vein, left internal jugular vein, and right common femoral vein, as well as transhepatic punctures (9).

It is fair to say that the only limitations are in the imagination and experience of the interventionalist seeing the patient, and that no patient needing a shunt should be excluded *a priori* for reasons of anatomy. Rather, patients presenting with anatomic challenges should be counseled regarding lower chance of technical success, and should be sent to an interventional radiologist who is very experienced in the procedure. Overshunted patients can be salvaged by the creation of a reducing stent, which is placed into the TIPS to reduce, but not occlude its flow. Varices showing high-grade hepatofugal flow *after* hemodynamically successful shunt creation can be catheterized via the TIPS and embolized with metallic coils. It is important to remeasure the portosystemic gradients after embolization because closing a high flow varix cuts off a high-flow exit from the portal system and can raise portal pressure (9).

Covered stents are a new and potentially revolutionary development. The available designs consist of conventional metallic stents covered with PTFE or other biocompatible material. The covering excludes the shunted blood from the surrounding hepatic envi-



Fig. 4. Catheterization of the portal vein.

ronment. Available data suggest that this has a major positive effect in reducing the incidence of stenosis and occlusion of the shunts. Original studies were done with homemade devices constructed of standard stents sewn to PTFE bypass material. Commercially manufactured covered stents have been available in Europe since the mid-1990s, and are recently available in the United States. A device developed specifically for TIPS is in the late stages of commercial development.

PREOPERATIVE TESTING AND PREPARATION

In the elective or reasonably stable patient, preoperative testing is desirable for planning the approach to the shunt and alerting to the risk of preventable complications. Keep in mind that patients requiring emergency TIPS for catastrophic bleeds may not have time for any preoperative imaging at all. Imaging is focused on the evaluation of patency of the portal vein and hepatic veins and in planning approach to the more challenging patients. Doppler ultrasound is the easiest and least invasive way to do this, although CT, MRI, and angiography have all been used. CT scanning can be useful for understanding the morphology of the liver. Keep in mind that in advanced



cirrhotics, atrophy of the right lobe, hypertrophy of the left lobe, and hypertrophy of the caudate lobe are all common, and can require alteration of the plan of attack. Endoscopy is very desirable preoperatively in cases done for upper gastrointestinal bleeding. Although common, varices are not the only possible reason for upper gastrointestinal bleeding in alcoholic patients. TIPS has no beneficial effect on common nonvariceal causes of bleeding such as Mallory-Weiss tears or gastritis. Blood tests include LFTs, BUN, and Creatinine determination, PT/PTT, hemoglobin, hematocrit, and platelet count. Hydration of the patient is desirable, particularly in those with any degree of renal insufficiency. Radiographic contrast presents a special risk to those patients. Prophylactic antibiotics are generally given to patients preoperatively. Many regimens exist. Correction of coagulapathy is sometimes desirable, but fairly controversial. So many of these patients have baseline coagulopathies that many operators treat only severe coagulopathy (PT>20, plts<50,000). Meticulous technique and avoidance of capsular puncture are probably more important than medical pretreatment in coagulopathic individuals. Renoprotective drugs are a new, but promising preoperative treatment for patients with renal insufficiency. Mucomyst and Fenaldopam are the most promising. Paracentesis is usually not necessary, but in extreme cases ascites will degrade the fluoroscopy image or distort the liver orientation enough to require preoperative drainage. Keep in mind that high volume paracentesis followed by TIPS creation adds up to significant hemodynamic alteration over a short period of time, and obligates careful monitoring of the state of hydration of the patients, particularly in those who also have GI bleeding.



Fig. 6. Portagram following TIPS.

COMPLICATIONS

Transient hepatic encephalopathy is fairly common. Typical reported rates are in the 10–15% range, although much higher rates have been reported in a few series. Whereas most cases can be successfully treated with lactulose, a very few severe instances require shunt revision or closure. Intraperitoneal bleed can result from inadvertent puncture of the liver capsule by the needle. Symptomatic cases are rare (1%) in experienced hands. Delayed shunt stenosis and occlusion is discussed later under "Results." Acute fulminant hepatic failure is a rare occurrence, and can be related to inadvertent connection of a hepatic arterial branch to the shunt. It can be diagnosed and treated by hepatic arteriography and coil embolization of the offending branch. Hemobilia is a rare complication and is seen in about 1% of cases. Transient renal failure is related to contrast nephrotoxicity and hepatorenal syndrome. It occurs in about 3% of cases. Bacteremia is seen in about 3% of cases.

RESULTS

Technical success in placing the shunt and correcting the gradient ranges from 96–100% in most series. Keep in mind that this is a technically difficult procedure with a learning curve. The 30-d mortality depends on the composition of the series. Mortality often relates more to the overall condition of the patient than to the actual TIPS procedure. APACHE score was considered to be the best predictor of mortality, and it is now becoming apparent that the Childs-Pugh classification is also a good predictor. Recurrent variceal bleeds are seen in 4–17%, and are often related to shunt stenosis or occlusion (10). Late shunt stenosis and occlusion is extremely common, and relates to fibrointimal hyperplasia within the stent or in the native vessel at one end of the stent. Redilation and/or placement of additional stents easily treat this. Shunt stenosis should always be suspected when varices and/or bleeding recur. Primary shunt patency is around 75% at 6 mo and 50% at 1 yr. Primary assisted patency runs around 85% at 1 yr (10). Transgression of a biliary duct by the shunt can result in exposure of the tract to bile and accelerate the restenosis process. Covered stents are a new and promising treatment for this problem.

POSTOPERATIVE CARE

Patients are transferred from the radiology suite to a unit that can provide frequent monitoring of vital signs, fluid input, and fluid output. ICU is not generally necessary for elective or stable patients. Bed rest is recommended for 6–24 h depending on the condition of the patient. PT/PTT, H/H, BUN/CR, and LFTs are closely watched in the first 24–48 h. Doppler ultrasound is obtained on the first postoperative day to assess shunt patency, and to establish a baseline for flow velocity through the shunt. It is then obtained at 3, 6, 9, and 12 mo after the TIPS, and Q 6 mo indefinitely thereafter. Reversal of portal venous flow, absence of portal flow in the liver, or peak intra-TIPS velocity of <50–80 cm/sec are all indicators of shunt malfunction, and should occasion a portogram, and shunt revision. An interesting new concept is the senescent TIPS. There seems to be a subpopulation of patients who stop consuming alcohol, regenerate some liver, and "outgrow" their need for a shunt. These patients will present with shunt stenosis or occlusion, but will have portosystemic gradients below 10–12 mmHg. Some investigators are choosing to follow rather than revise the shunts in this population.

ALTERNATIVES

Endoscopic management of varices is the main alternative to TIPS for variceal bleeding, and is well-described in Chapter 7.

It should be noted that TIPS and endoscopic techniques are often complementary. For instance, a bleeding patient treated acutely by variceal banding may subsequently undergo TIPS to prevent a second bleed.

Surgical portosystemic shunts have largely been replaced by TIPS. However, some centers believe that Child's class A cirrhotics with variceal bleeds should undergo splenorenal or other "distal" shunt rather than TIPS. For a further discussion of surgical shunts, readers are referred to Chapter 24. Leveen and Denver Shunts (implanted peritoneal to venous catheters) are still available for treatment of intractable ascites, but have largely been supplanted by TIPS.

COST

Overall costs of TIPS will often relate more to the patient's condition and need for intensive care than to the procedure itself. Fixed costs include one or more vascular stents at a cost of around \$1100–1500 each, \$500–1000 for other consumable supplies, as well as 1–3 h of angiographic room time, and a hospital stay of one or more nights. Compared to surgical shunt, one can count on savings related to decreased duration and intensity of the hospital stay. However, the need for lifetime surveillance and episodic revision of TIPS must also be considered in any cost analysis.

SUMMARY

- 1. The TIPS procedure has become the dominant method for decompression of portal hypertension, replacing surgical shunt in all but a few subsets of patients.
- 2. It can be performed in most patients suffering from the sequelae of portal hypertension.
- 3. Transient hepatic encephalopathy is fairly common. Typical reported rates are in the 10–15% range.
- 4. Late shunt stenosis and occlusion is extremely common, and relates to fibrointimal hyperplasia within the stent or in the native vessel at one end of the stent.
- 5. Doppler ultrasound is obtained on the first postoperative day to assess shunt patency. It should then obtained at 3, 6, 9, and 12 mo after the TIPS, and Q 6 mo indefinitely thereafter.

REFERENCES

- 1. Durham Janette Cardiovascular and Interventional Radiology Research and Education Foundation Videodisc Series Vol. 2: Portal Hypertension-Options for Diagnosis and Treatment. CIRREF 1992.
- 2. Laberge JM, Ring EJ, Lake EJ, et al. Transjugular Intrahepatic Portosystemic Shunts (TIPS); preliminary results in 25 patients. J Vasc Surg 1992;162:258–267.
- 3. Rosch J, Hanafee WN, Snow H. Transjugular portal venography and radiologic portocaval shunt: an experimental study. Radiology 1996;92:1112–1114.
- Peltzer MC, Ring EJ, Laberge JM, et al. Treatment of Budd-Chiari syndrome with a transjugular intrahepatic portosystemic shunt. J Vasc Interv Radiol 1993;4:263–267.
- Ring EJ, Lake JR, Roberts JP, et al. Percutaneous transjugular portosystemic shunt.s to control bleeding prior to liver transplantation. Ann Intern Med 1992;116:304–309.
- 6. Colapinto RF, Stronell RD, Birch SJ, et al. Creation of an Intrahepatic portosystemic shunt with a Gruntzig balloon catheter. Can Med Assoc J 1982;126:267–268.
- 7. Kandarpa K and Aruny J. (1996) *Handbook of Interventional Radiologic Procedures*, second edition. Lippincott Williams and Wilkins, Philadelphia, PA.
- 8. Laberge M, Ring EL, Gordon RL, et al. Creation of transjugular intrahepatic portosystemic shunt with the Wallstent endoprosthesis: Results in 100 patients. Radiology 1993;187:413–420.
- 9. Zemel G. Becker GJ, Bancroft JW, et al. Technical advances in transjugular intrahepatic portosystemic shunt. Radiographics 1992;12:615–622.
- Haskal ZJ, Pentecost MJ, Soulen MC, et al. Transjugular intrahepatic portosystemic shunt stenosis and revision: Early and midterm results. AJR 1994;163:439–444.

IX Abdominal Hernia Surgery
26 Hernia Surgery

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INTRODUCTION

A hernia by definition involves a defect in the fascia and (the potential for) protrusion of an organ and/or tissue through the abnormal opening. The positive pressure present within the abdominal cavity, individual anatomic variations in structure, biochemical differences in collagen and interstitial matrix composition, chronic injury, and trauma (including surgical) singly or in combination account for the pathobiology of most hernias (1).

The incidence and prevalence of groin hernias are poorly documented. Estimates of the prevalence of groin hernias suggest their presence in 2-4% percent of the overall population. Men are 5-10 times more likely to have an inguinal hernia than women. The elderly have an incidence at least twice that of younger adults, and it is increased in smokers as well. In 1996, an estimated 700,000 operations for groin hernias were performed in the United States (2,3).

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In the course of fetal development in the male, the testes migrate from the abdomen into the scrotum with the blood supply and vas deferens following the testicle, coming to lie in the line of decent of the testis. Remnants of the process vaginalis allow for herniation of the bowel through the deep or internal inguinal ring and may also be present as hydroceles. The inguinal canal is the passageway by which the spermatic cord leaves the abdomen to reach the testis in the male, and by which the round ligament of the uterus travels to the labium majoris in the female. Cremasteric muscle and fascia surround the spermatic cord, containing the vas deferens, testicular veins (pampiniform plexus), testicular lymphatic vessels, autonomic nerves, and the genital branch of the genitofemoral nerve. The walls of the inguinal canal consist of the aponeuroses of the external oblique muscle anteriorly and inferiorly (as it rolls under becoming the inguinal ligament), the fascia transversalis posteriorly (with reinforcement by the conjoint tendon medially), and superiorly by the internal oblique and transversus abdominus muscles. The medial aperture of this canal is the external or superficial ring, whereas the aperture of the deep or internal inguinal ring is an aperture in the floor or posterior aspect of the inguinal canal. With coughing or straining, muscular contraction allows the roof to compress the contents of the canal against the floor so that the canal is virtually closed.

Herniation of tissue into the inguinal canal may protrude directly through the fascia transversalis, the posterior wall of the inguinal canal, and is called the direct inguinal hernia. More commonly, the herniation is through the preexisting defect in the fascia transversalis, which is the deep/internal inguinal ring, and is referred to as an indirect inguinal hernia. Large hernias may be a combination of both. Technically, if the defect is lateral to the inferior epigastric artery and vein (branches of the femoral artery and vein) it is considered an indirect hernia, medially a direct hernia. Femoral hernias occur through the femoral canal deep or posterior to the inguinal ligament (Fig. 1).

Men account for 90% of inguinal herniorrhaphies, with indirect hernias accounting for 45–60% of these hernias, direct hernias 25–40%, and the remainder are combinations of direct and indirec, as well as femoral hernias. In the female, indirect hernias are the most common, followed by the femoral hernia. Overall, women have a greater numbers of femoral hernias than men. Of recurrent hernias, approx 60% are direct, 35% are indirect, and 7% are femoral (1-3).

INDICATIONS FOR SURGERY

The natural history of the unrepaired hernia is unpredictable. Although it is clear that hernias will not regress because of the (positive) intrabdominal pressure, their rate of enlargement and/or progression to a scrotal hernia is quite variable.

The presence of a hernia is an indication for its repair. Hernias may be repaired to correct a congenital defect. In the pediatric population, the most common cause for an inguinal hernia is the presence of a patent process vaginalis. Repair is indicated (in this and any age group) to obliterate the remaining process vaginalis. Hernia repair is undertaken to prevent complications. In fact, the smaller hernia should be considered more dangerous than the large hernia owing to its ability to strangulate the tissue herniating through the (small) defect. The hernia that goes on to cause strangulation may have been asymptomatic prior to this event. The third reason to repair hernias is to resolve accompanying symptoms. Larger hernias become painful as a result of compression of nearby



Fig. 1. (A) The inguinal canal, associated structures and locations of hernias. (B) Approaches for repairs—anteriorly and preperitoneally.

structures and become cumbersome, especially with any physical activity. Hernias that cause small bowel obstructions or constipation are obvious candidates for repair.

CONTRAINDICATIONS TO SURGERY

Because repair of a groin hernia can be performed under local anesthesia with minimal amounts of invasion and accompanying morbidity, most patients are candidates for repair. However, the inability to tolerate general anesthesia limits the choices of repair that are available. In a few individuals, even the stress and invasion of this procedure is so great that they should be observed for the development of complications rather than undergo operative repair. The patient with large amounts of ascites is not repaired because of the high rate of complications associated with patients in this condition. Trusses historically were an option as therapy, however, they are reserved for nonoperative candidates. If used, a truss should be in good condition and well fitting, and used only for reducible indirect hernias. A truss does not work well with direct hernias and can cause strangulation with any hernia that is not reduced.

OPERATIONS FOR HERNIA REPAIR

The repair of all hernias, regardless of their location or the technique used, requires first the reduction of the herniated tissue; second, the closure or reduction of the peritoneal sack that contained the herniated tissue; and finally, restoration of the anatomy of the abdominal wall to prevent a future hernia. Difficulties in this operation arise from the complexity of the anatomy (especially in the groin), individual variations there of and alterations in the regional anatomy caused by the hernia itself. The hallmark of a good repair is a low incidence of morbidity and recurrence (Fig. 2).



Fig. 2. Steps in repair of a hernia. (**A**) The hernia. (**B**) Reduction of the contents of the hernia sac. (**C**) Resection of the hernia sac. (**D**) Restoration of original anatomy. (**E**) Insertion of mesh to restore anatomy.

TISSUE REPAIR

Pediatric Hernia Repair

A pediatric hernia repair is the simplest hernia repair because it only involves the first two steps aforementioned with no repair of anatomy necessary. This reflects the pathology of a congenitally persistent process vaginalis, which needs to be obliterated. The internal ring itself is usually normal and needs no interventions to prevent future herniations.

Bassini and Shouldice Repairs

These two repairs are similar in that after reducing the hernia and resecting the hernia sack the floor of the inguinal canal is rebuilt using the patient's tissues. Technically, the floor or posterior aspect of the inguinal canal is opened with the conjoint area structures being taken to the inguinal ligament where they are sutured. The Bassini repair does this with interrupted sutures, the Shouldice repair with a series of running sutures. A femoral hernia cannot be repaired by this method because the orifice to the femoral canal lies deep to the inguinal ligament (4,5).

Cooper Ligament or McVay Repair

This tissue repair (after reduction of the hernia and resection of the hernia sack) involves division of the floor or posterior wall of the inguinal canal. The conjoint area is sutured to the pectineal ligament deep to the inguinal ligament. This obliterates the orifice from the femoral canal as well. However, a significant amount of tension is produced in this closure requiring a relaxing incision to be made in the anterior rectus sheath (1-3).

Mesh Repairs

The use of polypropylene mesh in the repair of hernias has become increasingly popular over the years. Initially used for recurrent or large hernias, it has become popular for virtually all hernia repairs outside of the pediatric hernia repairs. The polypropylene mesh not only incites a significant scar formation, but also is knit as part of the scar making it more durable than the native tissues themselves. Further, when mesh is used in the repair, the tissues, which contain the hernia, do not have to be placed under tension to accomplish the repair.

Anterior Mesh or Tension Free or Lichtenstein Repair

Popularized by Lichtenstein, this repair involves the reduction of the hernia sack contents and resection or reduction of the hernia sack. Most frequently, a piece of mesh is laid over the posterior or deep wall of the inguinal canal with tails that reapproximate themselves lateral to the spermatic cords so that the internal ring is recreated by the mesh. No attempt to reapproximate the native tissues is made in obliterating the hernia defect. A plug or cone of mesh may be used alone or in conjunction with this on lay patch to plug the defect directly. Other variations use mesh in the preperitoneal position (deep to the inguinal floor) (6).

Preperitoneal Repair

The preperitoneal repair uses an incision that is superior (above) to the inguinal canal. The incision is taken deep to the transversalis fascia but superficial to the peritoneum. This allows the inguinal canal to be approached deep to the floor or posterior wall of the inguinal canal. The peritoneum is not breached so that work in this plane and materials placed here do not come into contact with the intrabdominal contents. Through this plane, the hernia sack is reduced and a piece of mesh is placed which reinforces the inguinal wall and obliterates the defect where the hernia was. This mesh is held in place by the intrabdominal pressure, which is naturally transmitted through the peritoneum to the abdominal wall (where the mesh now interposes between the two) (7,8).

Laparoscopic Hernia Repair

Two laparoscopic hernia repairs, both of which place the mesh in the same position as the preperitoneal hernia repair aforementioned, have emerged. The totally extraperitoneal approach (TEPA) uses a laparoscope to move in the same planes as described in the preperitoneal approach. The transabdominal preperitoneal repair (TAPP) uses a laparoscope that is introduced into the abdomen proper with a peritoneal flap being made and pulled down, allowing a piece of mesh to be placed into the preperitoneal space. The peritoneal flap is returned over the mesh, excluding the mesh from the intrabdominal contents. Typically both of these repairs use three trocars and require a general anesthetic (1-3).

POSTOPERATIVE COURSE

The popularity of the mesh repair reflects not only the lower recurrence rates but also the easier post-op course experienced by most patients having this repair. The tissue repairs require 4–6 wk of light activity to allow wound healing to produce adequate tensile strength to permit the patient to return to normal activity. Return to heavy activities may be postponed up to 3 mo. Mesh repairs allow in contrast resumption of normal or heavy activity within a couple of days to 2 wk depending on the repair.

COMPLICATIONS

There are several potential complications to accompany repair of the inguinal hernia. Overall complications rates for both open and laparoscopic repairs range from 7–12%. The type of repair does affect the incidence and character of complications, but no single repair can claim fewer complications overall (1-3,6,9).

The nerves of the ilioinguinal region can be entrapped or transected in the course of hernia repair. Residual neuralgia occurs in as high as 30% of patients following open hernia repair, with chronic pain occurring in up to 5%. The complication is a frustrating one for both the patient and the physician, as there are no laboratory or radiographic tests to confirm the subjective nature of the complaints. The ilioinguinal, iliofemoral, lateral femoral cutaneous, and genitofemoral nerves may be involved. Whereas complete transection results in numbness to the affected distribution, injury or entrapment of the nerve will result in neuralgia, which can be mild or incapacitating. Entrapment can arise from a ligature, a misplaced securing staple, or adherence to the mesh. Stapling injuries occur more frequently with laparoscopic repairs, particularly to the lateral femoral cutaneous nerve. Management of these injuries often requires time and patience, but may on occasion require reoperation, removal of the offending agent and possibly division of the affected nerve. Data from Lichtenstein as well as laparoscopic repairs suggest a nerve entrapment incidence of <2% (1–3,6,9).

Testicular complications are rare but include devastating ones of ischemic orchitis and testicular atrophy. The former results primarily from manipulation of the pampiniform plexus, with subsequent venous thrombosis and disruption of the arteriovenous circulation. The syndrome manifests 2–5 d postoperatively with a hard and swollen cord, testicle, and epididymis. Aggressive analgesia is the recommended treatment for the discomfort that can expect to follow for the ensuing weeks. Swelling and induration lasts for up to several months. There is no treatment to avoid the potential progression of the orchitis to testicular atrophy. If this occurs, the testicle will shrink and become painless. Orchiectomy is indicated only in the rare circumstance of associated infection. Laparoscopic techniques, with less handling of the cord and its structures, have been shown to have a lower incidence of venous manipulation and orchitis. Anterior approaches to hernia repair may also incite manipulation or injury to the vas. In the face of an abnormal contralateral side, injury to the vas can cause infertility. This rare complication (0.04%) occurs more frequently in recurrent, open repairs, and manifests as a painful spermatic granuloma as well as dry ejaculation. The recommended treatment, whether recognized intraoperatively or postoperatively, is microsurgical repair of the vas (1-3,6,9).

Visceral injuries include injuries to the colon, bladder, and small intestine. Occurring in <0.5% of cases, they are found more frequently with sliding hernias (where a side of the hernia sac is composed of bowel or bladder wall). Incarcerated hernias also have an increased risk of visceral complications, particularly if the segment is released into the peritoneal cavity with unrecognized ischemia. Laparoscopic repairs have introduced further potential complications such as trocar site herniations, small bowel obstructions secondary to adhesions, and bowel or bladder lacerations. Some of these can be avoided with meticulous technique, and all are infrequent occurrences. Less morbid visceral complications include urinary retention, infection, hematuria, and postoperative ileus (1-3,6,9).

Infectious complications vex fewer than 2% of patients. Women and older patients (>70yr) have been shown to have statistically significantly higher rates of local wound infections. Certain hernias have a higher incidence of infection, the most frequent of which is incarcerated, followed by recurrent, umbilical, and femoral. Antibiotic prophylaxis is routinely used with placement of mesh, and infection of the mesh rarely requires excision. These wounds can be managed with drainage, antibiotics, and granulation. Osteitis pubis is a complication that can arise with either suture or stapling through the periosteum. The prevalence has decreased with elimination of sutures through the periosteum. However, staple tacking of the mesh to the pubic tubercle may contribute to a resurgence of medial recurrences at the level of the pubic tubercle (1-3, 6, 9).

Fluid collections in the postoperative wound include seromas, hydroceles, and hematomas. Their frequency reflects tissue trauma (cautery, foreign body), severance of lymphatic drainage, and hemostasis, respectively. Most fluid collections, regardless of the etiology, are managed conservatively including scrotal support (for hematomas found there). Drainage is reserved for those in severe discomfort or if there is any evidence of concomitant infection. Most seromas, hydroceles, and hematomas slowly resolve over several weeks.

Laparoscopy introduces complications inherent to both general anesthesia and laparoscopy. Insufflation of carbon dioxide holds the potential to lead to untoward complications including venous air embolism, hypercarbia, and cardiac arrhythmias. Hernias in the trocar sites used to introduce the laparoscopic instruments have been described.

Recurrence of an inguinal hernia is a potential complication for any hernia repair. The incidence using a mesh repair appears to be lower than most tissue repairs. Reported recurrence rates vary from less than 1-10% for inguinal hernias and from 5-35% for recurrent hernia repair (2,3). Mortality should be extremely rare, as there are a number of large series reported without any deaths.

FEMORAL HERNIAS

Femoral hernias occur through the femoral canal, deep to the inguinal ligament and medial to the femoral vein. Occurring more frequently in women than in men, approx 80% present with the need for emergent operation because of obstruction or strangulation of the small bowel.

Repair of this hernia may take one of three approaches: through the groin below the inguinal ligament for an elective repair; through the inguinal area; or through an intrabdominal approach (especially if there is a complication such as necrosis of the small bowel due to strangulation). The steps of this hernia repair are the same as outlined for inguinal hernia with obliteration of the defect in the femoral canal being accomplished either through approximation of native tissues or the use of mesh.

Recovery from this procedure will require hospitalization if the patient presents with a complication of the femoral hernia. Complications from this procedure parallel those of the repair of the inguinal hernia. Recurrence rates are from 1 to 7% (2).

VENTRAL HERNIA

Hernias can occur anywhere in the abdominal wall. The most common are ventral and/or incisional hernias. Commonly in the midline, they include epigastric and umbilical hernias. Incisional hernias occur in at least 2–11% of abdominal incisions (10,11). Midline incisions may be at increased risk as they run perpendicular to the lines of tension. Risk factors for incisional hernias include local stresses such as wound infection, obesity, abdominal distention, ascites, and pulmonary complications, as well as systemic factors such as advanced age, post-operative chemotherapy, steroids, malnutrition, and multisystem organ failure. Indications for repair parallel those of the inguinal hernia (10,11). By contrast, diastasis of the abdominal recti muscles, representing a separation of the muscles that is apparent from the xiphoid process to the umbilicus, is a cosmetic defect that is generally painless, and poses no risk for incarceration.

OPERATIVE REPAIR AND TECHNIQUES

Following the steps for hernia repair outlined earlier, primary (tissue) repairs are used for small first-time repairs. Because of high recurrence rates with the primary repair, mesh is employed for larger defects and recurrent hernias (12). With open repairs, mesh may be used as an on-lay patch to buttress a repair; as an inlay patch placed anteriorly, posterior to the rectus sheath as a sandwich around the fascial planes, or in the preperitoneal space; or as an intraperitoneal on lay-patch. Particularly large and difficult repairs may be repaired using an approach popularized by Stoppa placing a large sheet of mesh placed very widely in the preperitoneal space (13). Laparoscopic approaches utilize an intraperitoneal placement of the mesh (14). Polypropylene and Dacron mesh, historically the most popular, ordinarily are not be used intraperitoneally because of the risk of a fistula to the bowel (Fig. 3).

POSTOPERATIVE COURSE

Larger hernia repairs or recurrent repairs usually require hospitalization because the intrabdominal components of the procedure and their sequelae (such as an ileus, larger



Fig. 3. Mesh Placements in a ventral hernia repair. (A) Onlay patch reinforcing an anatomic repair. (B) Patch interposed anteriorly in defect. (C) Patch in the posterior rectus sheath. (D) Patch in the preperitoneal space. (E) Intraperitoneal patch with hernia sac left in place. (F) Sandwich configuration of mesh.

fluid shifts, and bowel obstruction), and for pain control. Larger open repairs frequently require the use of drains due to the amount of dissection involved. The smallest repairs are performed as outpatient surgery.

COMPLICATIONS

Complications parallel those of an inguinal hernia repair including infection, hematoma, and seroma. Visceral injury, especially to the small bowel, is more common

with the ventral hernia as the adhesions to the hernia sac are more plenteous and more complex. Additional serious complications include prolonged ileus, chronic draining sinuses, extrusion of the mesh material, and erosion of the mesh into adjacent structures including intestine (resulting in a mesh infection and/or enterocutaneous fistula). Unlike groin hernias, the reoccurrence rate for ventral hernia repair, especially primary repair (without mesh), can be as high as 50%. In the most difficult and complex of hernias, the Stoppa repair offers only a 15-20% reoccurrence rate (8,13).

COST OF PROCEDURES

Most inguinal hernias and many ventral hernias can be repaired as outpatients. The cost of the procedure then includes fees for the services of the surgeon and the anesthesiologist (if used), and the surgical environment. Medicare reimbursement for a inguinal hernia repair and ventral hernia repair (with the implantation of mesh) in the same day surgery setting is \$2100 and \$4100, respectively.

SUMMARY

- 1. The current standard of care is to repair essentially all hernias to treat pathologic sequelae, prevent complications and relieve suffering and distress.
- 2. The utilization of mesh is becoming increasingly popular as this successfully lowers the rate of recurrence and may simplify the repair.
- 3. The complication rates overall, outside of recurrence, are probably little changed with the use of mesh materials.

REFERENCES

- 1. Knol JA, Eckhauser FL. (1997) Inguinal anatomy and abdominal wall hernias. In: Greenfield LJ, ed. Surgery, Scientific Principles and Practice. 2nd ed. Lippincott-Raven, Philadelphia, PA, 1997;pp. 1207–1245.
- 2. Eubanks WS. Hernias. In: Townsend CM, ed. Sabiston Textbook of Surgery, 16th ed. W.B. Saunders, Philadelphia, PA, 2001;pp. 743–801.
- Scott DJ, Jones DB. (2001) Hernias and Abdominal Wall Defects. In: Norton JA, et al., eds., Surgery:Basic Science and Clinical Evidence. Springer-Verlag, New York;2001;pp. 787–823.
- 4. Wantz GE. The Operation of Bassini as described by Attilio Catterina.SG&O 1989;168:67-80.
- 5. Welsh DR, Alexander MA. The Shouldice Repair. Surg Clin NA 1993;73:451.
- Lichtenstein IL, Shulman AG, Amid PK, Montilor MM. The tension-free hernioplasty. Am J Surg 1989;157:188–193.
- 7. Nyhus LM, Pollak R, Bombeck CT, Donahue PE. The preperitoneal approach and prosthetic buttress repair for recurrent hernia. Ann Surg 1988;733–737.
- 8. Stoppa RE. The treatment of complicated groin and incisional Hhernias. W J Surg 1989;13:545-554.
- 9. Ponka JL, ed. Hernias of the Abdominal Wall. Philadelphia, PA, Sanders, 1980.
- Carlson MA, Ludwig KA, Condon RE. Ventral hernia and other complications of 1,000 midline incisions. S Med J 1995;88:450–453.
- 11. Santora TA, Roslyn JJ. Incisional hernia. Surg Clin NA 1993;73:557–570.
- 12. Luijendijk RW. A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med 2000;343:392–398.
- 13. Veillette G, MacGillivray D, Whalen G. Practical experience with the Stoppa repair of ventral/ incisional hernias. Conn Med 2001;67–70.
- Heinford BT,Park A, Ranshow BJ, Voeller G. Laparoscopic ventral and incisional hernia repair in 407 patients. J Am Col Surg 2000;190:645–650.



27 Peritoneal Shunts

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INTRODUCTION

The peritoneal cavity has a surface area estimated at one square meter. The peritoneum is an important factor in the development of ascites and contributes to the difficulty in controlling this condition. Because it can serve as a two-way dialysis membrane, physicians have utilized the absorptive power of the peritoneum to treat conditions such as renal failure and hydrocephalus.

PERITONEOVENOUS SHUNTING

Ascites represents the buildup of fluid within the peritoneal cavity such that the rate of conversion of plasma to peritoneal fluid exceeds the rate of reabsorption from the peritoneal cavity (1). In this sense, it represents a failure of the peritoneum. Ascites is commonly found in patients with chronic liver disease and those with advanced malignancies. Ascites is associated with other complications of advanced liver disease such as spontaneous bacterial peritonitis, hepatorenal syndrome, and bleeding esophageal varices (2). In addition, patients with ascites suffer from severe protein calorie malnutrition with wasting, and likely nutritionally related immunoincompetence (2).

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MEDICAL THERAPIES

Treatment of patients with ascites is intended to reduce the risk of the potentially lethal complications. Successful treatment can also relieve discomfort secondary to the abdominal distention and improve a patient's nutritional status and overall state of health. About 10% of cirrhotics have intractable or refractory ascites, which is defined as a prolonged history of ascites unresponsive to 400 mg of spironolactone or 30 mg of amiloride plus up to 120 mg of furosemide daily for 2 wk (3). The prognosis of these patients is poor, with a 2-yr mortality of 50 to 70% (1,3). For such patients and those with complications related to ascites, other means of therapy must be sought.

Failing medical therapy, the first option is large volume paracentesis. This has been shown to decrease hospitalization time and complications, but not mortality rates (3). A single large volume paracentesis combined with diuretic therapy and dietary modification is indicated for tense ascites, and can improve a patient's cardiac function (4). For refractory ascites, paracentesis can be performed repeatedly as needed with the option to give intravenous albumin if more than 5 L of fluid is removed.

SURGICAL SHUNTS

A second option is the placement of LeVeen or Denver peritoneovenous shunts, devices with one-way valves that allow the return of ascitic fluid from the peritoneal cavity back to the systemic circulation. This results in an immediate natriuresis and diuresis in most patients (3), increases renal blood flow with reduced sodium retention, improves nutritional status with the preservation of protein, increases mobility, and avoids the repeated accumulation of large amounts of ascites with the requisite paracentesis. Peritoneovenous shunts are contraindicated for the management of malignant ascites and in patients with peritonitis. Relative contraindications to placement of a peritoneovenous shunt include alcoholic hepatitis, coagulopathy, encephalopathy without an elevated BUN, and jaundice (1).

The LeVeen shunt is a non-collapsible tube with a one-way pressure sensitive valve (1). It can be placed with the patient under general or local anesthesia (accompanied with invasive hemodynamic monitoring). The proximal end of the shunt is placed into the peritoneal cavity in a procedure similar to the Tenckhoff catheter insertion. The shunt is tunneled subcutaneously and the distal end is placed into the superior vena cava, usually through the right internal jugular vein (1). When the patient inspires, the intrathoracic pressure drops (to minus 5 cm of water below atmospheric pressure) and the intraperitoneal pressure rises slightly because of the descent of the diaphragm (1). This allows the pressure sensitive valve to open and the ascites to drain into the venous system, without venous backflow (1). The use of pre- and postoperative prophylactic antibiotics is important to reduce the risk of infection. Ascites should be drained from the peritoneal cavity at the time of shunt placement to prevent the development of disseminated intravascular coagulation (1,3). In some patients alternative venous access points must be used such as the left axillary vein or even the inferior vena cava via the femoral vein if the internal jugular vein is not accessible (1).

The Denver shunt is inserted in a manner similar to the LeVeen shunt. In addition to the one-way valve, the Denver shunt has a subcutaneous pump mechanism that the patient must squeeze to move fluid from the peritoneum to the systemic circulation (to its advantage and disadvantage compared to the LeVeen shunt) (Fig. 1). In a randomized



Fig. 1. A diagram showing placement of a Denver shunt. The proximal end of the shunt is placed into the peritoneal cavity and the distal end is placed into the superior vena cava, usually through the right internal jugular vein. A subcutaneous pump mechanism can permit a patient to manually pump fluid from the peritoneum to the systemic circulation.

prospective trial by Fulenwider et al., the LeVeen shunt had better patency rate than the Denver shunt (5).

COMPLICATIONS

Immediately upon insertion profound coagulopathy can result from the release of fibrin split products and tissue plasminogen activator from the peritoneal fluid into the systemic circulation (1). Draining the ascites externally and replacing the fluid with saline at the time of shunt placement can avoid coagulopathy (1,3). If disseminated intravascular coagulopathy does occur, the patient should be treated with fresh frozen plasma, platelets, epsilon-amino caproic acid, and blood transfusions (1). Pulmonary edema and variceal bleeding caused by increased portal pressure can result secondary to the increased intravascular volume (3).

Shunt obstruction due to fibrin deposition in the shunt is found in 40% of patients within the first year and is the most common late complication (3). Obstruction can also be caused by a technical error during placement, such as kinking of the shunt tubing, improper positioning of the venous end of the shunt, or thrombosis at the venous tip. A rapid recurrence of ascites usually indicates catheter obstruction. Injecting technetium sulfur colloid into the peritoneal cavity and observing the isotope within the shunt tubing or lung can confirm the patency of the shunt (1).

Intraperitoneal infection after peritoneovenous shunt insertion is also common. Affected patients may have alteration of their mental status and worsening of their liver function without signs of peritonitis. Positive preoperative ascites cultures are predictive of post-operative peritonitis (6). Treatment consists of obtaining peritoneal fluid and blood cultures, administration of intravenous antibiotics, and in most cases, shunt removal.

The 30-d operative mortality for peritoneovenous shunting ranges from as low as 10%, but up to 25% in patients with liver failure (3). The 1-yr survival rate is 77.7% in patients with good liver function, and 61.3% and 24.7% in moderate or severe liver failure, respectively (7). Patients with a serum bilirubin less than three have a lower postoperative mortality and longer overall survival than those patients with a bilirubin greater than three (6). Although peritoneovenous shunting patients have shorter hospitalizations, survival rates are not significantly improved when compared with medical therapy plus paracentesis (2,4).

ALTERNATIVE PROCEDURE

Because of the lack of survival benefit and relatively high rate of associated complications, peritoneovenous shunting is not frequently used (4). A third option for the treatment of refractory ascites is the diminution of portal pressures via transjugular intrahepatic portosystemic shunting (TIPS) (3). Placed percutaneously by interventional radiologists as a shunt from the portal vein to the inferior vena cava (IVC), TIPS is replacing paracentesis and peritoneovenous shunting as the treatment of choice for refractory ascites in part because TIPS is the bridge to transplantation for most of these patients who have Child-Pugh Class C cirrhosis. TIPS has worked well because surgical portosystemic shunts are contraindicated in patients with ascites complications associated with the TIPS procedure (in addition to the local and technical ones). Associated complications include significant encephalopathy in 23–30% of patients and shunt occlusion (opposite complications related by virtue of the shunt diameter (and length) (3,4).

VENTRICULOPERITONEAL SHUNTS

Hydrocephalus is a condition in which the rate of cerebrospinal fluid formation is greater than the absorption rate, resulting in dilatation of the ventricles. Ventriculoperitoneal (VP) shunting is the surgical treatment of choice for the management of hydrocephalus.

Technique

The VP shunt is made of silastic materials and contains a one-way valve in order to shunt cerebrospinal fluid (CSF) from the ventricular space to the peritoneal cavity where it is reabsorbed. The proximal end of the shunt is placed into the ventricle by a neuro-surgeon, the shunt tunneled subcutaneously and the distal end inserted into the abdominal cavity in a manner similar to the LeVeen shunt (Fig. 2). Prophylactic antibiotics are administered (preoperatively) to reduce the risk of infection.

Complications

The most frequent complications related to VP shunts are infection and malfunction, usually necessitating the need for surgical revision. Malfunction may result from obstruction secondary to protein deposits within the shunt tubing or from disconnection. Shunt revision is required in at least 28% of adult patients, many of whom require multiple revisions (8). When the shunt cannot be revised to the peritoneal cavity, alternatives such as ventriculoatrial or ventriculopleural shunts can be placed.

Formation of a loculated intraperitoneal CSF collection, or pseudocyst, has been reported (9). Likely, the result of an inflammatory response to CSF infection or to the catheter itself with fibrous encapsulation of the area, these patients may present with



Fig. 2. A diagram showing ventriculo-peritoneal shunt. The proximal end is placed in the ventricle after craniotomy. The shunt is tunneled in the subcutaneous tissue into the peritoneum. A one way valve allows CSF to drain in the peritoneal cavity.

neurologic changes owing to shunt obstruction or with abdominal complaints (9). An intraabdominal mass may be palpable on physical exam and can be imaged by ultrasound or computerized tomography. Treatment in cases of shunt infection includes externalization of the shunt and antibiotics. If no infection is present, then the pseudocyst can be fenestrated by laparotomy or laparoscopy and the shunt repositioned (9).

Delayed bowel perforation is a rare abdominal complication of VP shunts with an incidence less than 0.1% (10). This problem may be caused by fibrosis around the shunt causing pressure on and then perforation of the bowel. Less than 25% of patients with this complication have signs of peritonitis making the diagnosis difficult at times (10). Patients may have ventriculitis, peritonitis, abdominal pain, or shunt malfunction. The most common presentation is the passage of the catheter through the anus (10). Treatment consists of shunt externalization and culture. Laparotomy is required for patients with peritonitis, otherwise the shunt can be removed from the peritoneal cavity percutaneously. The mortality rate in these cases is approx 15% (10).

CONTINUOUS AMBULATORY PERITONEAL DIALYSIS CATHETERS

Peritoneal dialysis catheters are placed into the abdomen most often for continuous ambulatory peritoneal dialysis (CAPD), as well as for acute dialysis and for drainage of malignant ascites. Continuous ambulatory peritoneal dialysis offers several advantages over hemodialysis including the ability to perform dialysis without the need for vascular access, systemic heparinization, or the hemodynamic changes associated with the volume shifts of hemodialysis. In addition, the quality of life of those on CAPD may be preferred as this process can be safely completed at a patient's home, work or play, saving multiple weekly trips to a dialysis unit.

SHUNT PLACEMENT

Open vs Percutaneous

Peritoneal dialysis catheters can be placed via an open or percutaneous method. General contraindications to catheter placement include abdominal wall hernias or infections, active inflammatory bowel disease, diffuse intraabdominal adhesions, respiratory insufficiency, and gastrointestinal stomas (11). The Tenkhoff catheter, which is made of Silastic and equipped with two Dacron cuffs, is the most commonly used PD catheter. Placement by the open method is done in the operating room under general or local anesthesia. A small infraumbilical midline incision is used to better allow the catheter to reach into the dependent pelvis. (A supraumbilical incision can be utilized in patients with previous lower abdominal surgery to avoid adhesions.) The abdominal wall fascia is opened, a purse-string suture placed into the peritoneum, and a catheter guide used to direct the catheter toward the pelvis. The purse-string suture is tied down, securing the catheter in position with a watertight seal with the distal Dacron cuff just outside the peritoneum. The proximal end of the catheter is then brought out through a separate small incision site in the abdomen with the proximal Dacron cuff remaining in the subcutaneous tissue helping to secure the catheter in place by fibrosis over time. In the majority of cases, peritoneal dialysis can be instituted immediately.

A laparoscopic approach to catheter placement has also been utilized. This method has the advantage of allowing the guidance of the catheter into proper position in the pelvis under direct vision.

The percutaneous placement of a PD catheter does not require an operating room and can be performed at the bedside or in a treatment room with local anesthesia plus sedation. The peritoneum is instilled with 2-4 L of dialysate via an angiocatheter and a dilator and introducer sheath are inserted into the peritoneal space over a guide wire (12). A Tenckhoff catheter is then directed through the sheath toward the pelvis, and the sheath is pealed away. The proximal end of the catheter is then tunneled subcutaneously, as in the open method, through a separate site.

COMPLICATIONS

Infection with peritonitis, which occurs in 21%-34% of patients, is the most common complication associated with PD catheters (11,13). In such cases, clinical evidence for peritonitis including fever, abdominal pain and tenderness, and an effluent dialysate leukocyte count greater than 300–500 per mL is diagnostic (11,13). Cultures of the dialysate should be taken, and treatment, consisting of administration of intravenous antibiotics and addition of antibiotics to the dialysate, initiated. The catheter does not need to be removed except in cases when the peritonitis does not improve with antibiotic therapy (11,13).

The second most frequent complication of PD catheters is occlusion, with an incidence of 19 to 22% (11,13). Obstruction may result from intra-abdominal adhesions or

formation of fibrin thrombi within the catheter. Catheter obstruction is seen more frequently in patients with prior surgery and in the obese, likely caused by entrapment of the catheter by the omentum or adhesions (11). Fogarty balloon catheters and guide wires, which can be passed safely thru occluded catheters under fluoroscopic guidance, have been used to dislodge fibrin plugs and prevent catheter failure (11).

Leakage of dialysate around the catheter or thru the wound is seen in approx 7–8% of cases (11,13). This problem can be resolved by reducing the volume of dialysate or by temporarily withholding the CAPD (13). Placement of local skin suture at leakage sites can also be effective at controlling the problem (11). Wound infections occur in 3.5–7% of patients and may necessitate catheter removal (11,13). Hernia at the incision site occurs in less than 2% of cases and may result in incarceration (11,13).

Serious complications may result during the placement of the catheter. Perforation of the bladder while guiding the catheter into the pelvis occurs in less than 1% of cases (11,13). This incidence of this complication can be decreased by placement of a Foley catheter preoperatively to decompress the bladder. Direct puncture of the small or large bowel can also occur, with an incidence of about 1% (11,13). Conversion to a formal laparotomy would be required to repair these injuries and the initiation of CAPD might be delayed. The use of laparoscopy may help to prevent or more readily identify these complications. Erosion of the catheter into the bowel has also been described as a late complication (13).

In the series of 154 patients with percutaneously placed CAPD catheters by Allon et al. the catheter failure rates due to obstruction and leak were 9.1% and 2.6%, respectively (12). There was one bladder perforation and two open operations were required to control postprocedure intraabdominal bleeding (12). Catheter failure secondary to infection was seen in about 8% of patients with additional cases in which the infection was cleared with antibiotics (12). In approx 2% of patients the catheter could not be placed percutaneously.

COST OF PROCEDURES

Assuming placement in the same day surgery setting, the approximate cost of the placement of a peritoneal venous shunt is \$1950 and of a peritoneal dialysis catheter is \$1300.

SUMMARY

- 1. The peritoneum plays an active role allowing it to be manipulated for therapeutic benefit in hydrocephalus and renal failure.
- Ascites represents an inability of the peritoneum to absorb adequate amounts of peritoneal fluid, which when exaggerated requires therapy to decrease its production, increase it reabsorption, or remove it. Peritoneo-venous shunt is effective in controlling ascites in patients who are refractory to diuretics and require frequent therapeutic paracentesis.

REFERENCES

- 1. LeVeen HH. The LeVeen shunt. Ann Rev Med 1985;36:453-469.
- 2. Moskovitz M. The peritonvenous shunt: expectations and reality. Am J Gastroenterol 1990;85:917-929.
- 3. Wong F, Blendis L. Peritoneovenous shunting in cirrhosis: its role in the management of refractory ascites in the 90s. Am J Gastroenterol 1995;90:2087–2089.
- 4. Yu AS, Hu KQ. (2001) Management of ascites. Clin Liver Dis 2001;5:541-568.
- 5. Fulenwider JT, Galambos JD, Smith RB, et al. LeVeen vs Denver peritoneovenous shunts for intractable ascites of cirrhosis. Arch Surg 1986;121:351.

- 6. Fulenwider JT, Smith RB, Redd SC, et al. Peritoneovenous Shunts: Lessons learned from an eight year experience with 70 patients. Arch Surg 1984;119:1133–1137.
- Smadja C, Franco D. The LeVeen shunt in the elective treatment of intractable ascites in cirrhosis: A prospective study on 140 patients. Ann Surg 1985;201:488–493.
- 8. Puca A, Anile C, Maira G, et al. Cerebrospinal fluid shunting for hydrocephalus in the adult: Factors related to shunt revision. Neurosurgery 1991;29:822–826.
- 9. Bryant MS, Bremer AM, Tepas JJ, et al. Abdominal complications of venticuloperitoneal shunts: Case reports and review of the literature. Am Surg 1988;54:50–55.
- 10. Sathyanarayana S, Wylen EL, Baskaya MK, et al. Spontaneous bowel perforation after ventriculoperitoneal shunt surgery: Case report and a review of 45 Cases. Surg Neurol 2000;54:388–396.
- 11. Bullmaster JR, Miller SF, Finley RK, et al. Surgical aspects of the Tenckhoff peritoneal dialysis catheter, a seven year experience. Am J Surg 1985;149:339–342.
- 12. Allon M, Soucie JM, Macon EJ. Complications with permanent peritoneal dialysis catheters: Experience with 154 percutaneously placed catheters. Nephron 1988;48:8–11.
- Cronen PW, Moss JP, Simpson T, et al. Tenckhoff catheter placement: surgical aspects. Am Surg 1985;51:627–629.

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