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MANAGEMENT OF COMPLICATIONS AFTER LAPAROSCOPIC GASTRIC BYPASS

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INTRODUCTION

The optimal management of complications following laparoscopic Roux-en-Y gastric bypass (LRYGBP) in obese patients requires a low threshold of suspicion and early diagnosis of complications. Clinical manifestations, especially those of intra-abdominal septic complications, differ from standard descriptions in the nonobese patient. It is thus essential for the clinician to be specifically trained and experienced in the management of bariatric surgical patients.

Every effort must be made to prevent complications, and this objective requires a process that starts with the proper constitution of the bariatric program itself. Adequately trained surgeons; operating room staff who are specifically trained for minimally invasive bariatric surgery; a multidisciplinary team that also includes dietetic support, medical specialty staff, and a radiology suite that can cater to morbidly obese patients are all necessary components of a comprehensive program dedicated to bariatric surgery.

ANTICIPATING COMPLICATIONS

Anticipating complications starts with preoperative screening and the preoperative workup. Careful preoperative workup will reveal significant occult comorbidities in up to 30% of patients. Patient education during this period through workshops, videos, and printed educational material also helps by priming the patient to report important symptoms that may indicate a complication at an early stage.

The timeline in Figure 26.1 provides a framework for some notable complications that may arise after an LRYGBP and includes a checklist for clinical, radiological, and laboratory data. Abnormalities that should alert the clinician of an impending complication or the potential to develop one are also included. A strategy of anticipating complications and identifying them before they are overtly manifest can be achieved by following a routine pathway in the postoperative period. The timeline is not meant to provide exclusive time periods for risk, but rather to serve as a guide to what type of complications occur in which postoperative time frame. Exceptions to this framework do occur.

MANAGEMENT OF EARLY POSTOPERATIVE COMPLICATIONS

Apneic Arrest

Apneic arrest is a feared complication in patients whose sleep apnea has remained undiagnosed or mismanaged. Sleep apnea is defined as a respiratory disturbance index (number of apnea-hypopnea episodes per hour of sleep) of 5 or more in the presence of excessive daytime somnolence.[1] Patients with a body mass index (BMI) over 50, with hypersomnolence, hypertension, or a history of loud snoring should be assumed to have sleep apnea.[1] Preoperative evaluation of patients should include administering the Epworth Sleepiness scale questionnaire[2] or a Multivariable Apnea Prediction (MAP)[3] questionnaire, with scores greater than 9 or 0.7, respectively, predicting a high probability of sleep apnea. These instruments serve to identify those patients who need a formal sleep study. An inpatient polysomnography[4] is the "gold standard" of sleep studies. High-risk patients should preoperatively undergo this test, with estimation of positive airway pressure needed to keep the upper airway patent.[5]

Two prospective studies have proven the value of continuous positive airway pressure (CPAP) during the perioperative period in preventing apneic arrest without an attendant increase in the risk of anastomotic leaks.[6,7] It is important for those caring for the patient to understand

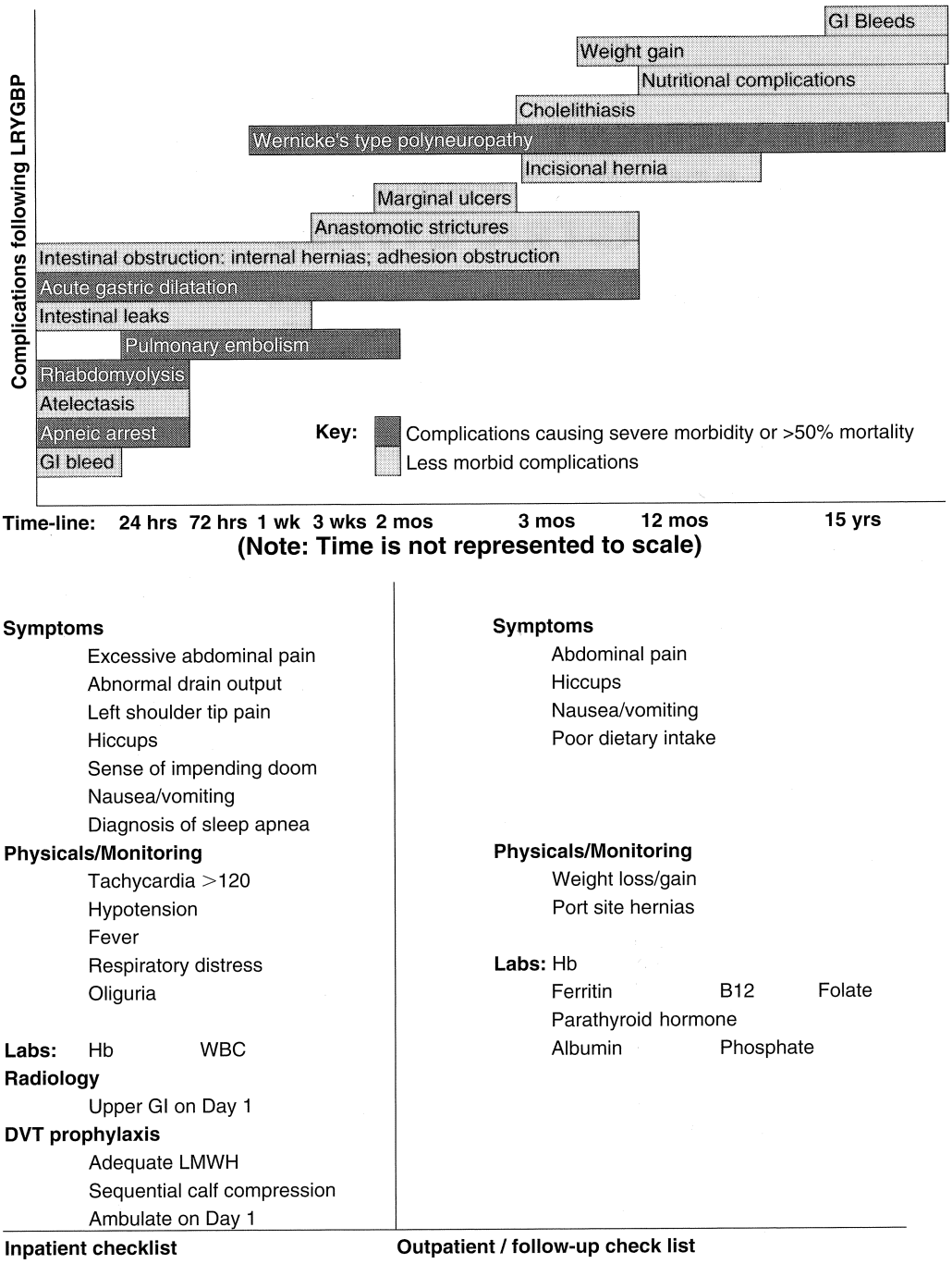

Figure 26.1. A mental timeline and checklist help with proactive search for complications.

the increased sensitivity of sleep apnea patients to narcotics which, in the absence of CPAP support, can precipitate upper airway collapse, hypoventilation, and respiratory acidosis leading to respiratory or cardiac arrest.[5,8,9] These patients should be in monitored beds, on CPAP, while they are on opiod analgesics. When CPAP has been prescribed, the patient should undergo a period of acclimatization to the face mask for at least 2 weeks prior to the surgery.

The clinical deterioration leading to apneic arrest can be very subtle and easily overlooked. A high-risk patient may be deceptively comfortable, having episodes of desaturation that are readily corrected by oxygen. However, unless one regularly checks for progressive hypercapnia, there is a risk of CO_2 narcosis and respiratory acidosis leading to cardiac arrest. In such a situation, emergent intubation may be technically difficult, but the use of a nasopharygeal airway and hand-ventilation with a good-

fitting rebreather face mask will allow adequate gas exchange and may be lifesaving.

Atelectasis

Morbidly obese patients are predisposed to basal atelectasis because of a reduction in forced vital capacity and forced expiratory volume. It typically manifests during the 24 to 48 hours after surgery with mild tachypnea, fever, leukocytosis, and loss of lung volume on one or both sides on a chest x-ray. Untreated, it may progress to a basal bronchopneumonia. Prevention is geared toward optimal pain management, ambulation on the evening of surgery, and incentive spirometry to reduce the tendency to atelectactic collapse by increasing forced vital capacity. An often misunderstood aspect of care is that the end point of pain management should be a painless and effective cough and not merely the achievement of favorable numbers on a visual analogue pain scale. Awake flexible bronchoscopy is a valuable tool in helping to clear out large mucus plugs and expand collapsed atelectatic segments and should be used early and without hesitation, especially in smokers.[10,11] The routine or prophylactic use of CPAP cannot be recommended because this has not been shown to reduce pulmonary complications or duration of hospital stay. In fact, there is concern regarding the risk of exerting pressure causing distension of the foregut anastomosis.[6,7]

Venous Thromboembolism

Gastric bypass carries a mortality rate of between 0.5 and 1%, with pulmonary embolism (PE) being the commonest cause of death. Currently employed prophylactic methods use low–molecular-weight heparins (LMWH) in combination with mechanical calf compression.[12] However, despite implementation of these standard measures, the reported incidence of fatal PE has ranged from 0.2 to 0.64%,[13–15] accounting for between 30 and 50% of all deaths after this operation.[16,17] One possible explanation for this high incidence of PE may be that the dose for LMWH was derived, without modification, from proven strategies developed for the average-sized adult undergoing general surgical operations.

The particularly high-risk group for venous thromboembolism among morbidly obese patients include those with lower limb venous stasis,[18] BMI greater than 55,[19] history of previous venous thromboembolism, and obesity hypoventilation syndrome (OHS),[20] the latter acting as a marker of occult pulmonary hypertension. Indeed it has been proposed that in the presence of any of the above clinical features,[18,19,21] or in the presence of OHS and a mean pulmonary artery pressure of 40 mm Hg or more,[18,22,23] consideration should be given to the prophylactic placement of a vena caval filter. Such measures have been associated with a reduction in postoperative PE-related mortality.

Pulmonary embolism must be considered in any postoperative patient with diaphoresis, tachycardia, or hypoxia, especially in the absence of a fever or raised white count. These may be indistinguishable from pulmonary atelectasis or intestinal leaks. Occasionally, the appearance is more subtle, with mild shortness of breath or pleuritic pain on deep inspiration. Confirmation of diagnosis with CT pulmonary angiogram[24] should ideally precede institution of therapeutic anticoagulation in the early postoperative period because of the risk of hemorrhage. However, most CT suites are unable to accommodate patients over 300 lb, and it may not be possible to obtain this investigation or a ventilation-perfusion scan. In this situation, it is safer to empirically start full anticoagulation, accepting an 11% risk of major hemorrhage (of which 3% will be fatal) for 5 days of intravenous heparin compared to an overall 6% risk of death and 2% risk of serious permanent disability associated with pulmonary embolism.[25] Activated partial thromboplastin and prothrombin time (APTT and PT) monitored therapy with unfractionated heparin followed by warfarin has been the mainstay of the management of venous thromboembolism. Unmonitored therapy with low-molecular-weight heparin provides an equivalent practical alternative.[26]

There is an emerging body of evidence suggesting the value of extended, out-of-hospital, prophylaxis against venous thromboembolism in high-risk patients.[26a,26b] While these studies have been on patients undergoing lower limb joint replacement surgery, there would be a good case for extending the results to the postoperative morbidly obese patient. Our current practice is to provide extended prophylaxis for a further 12 days in patients with a BMI 60 or in a lower BMI patient who is at added risk of VTE.

Intestinal Leaks

Intestinal leaks are probably the most common preventable cause of death after pulmonary embolism, complicating between 2 and 7% of Roux-en-Y gastric bypass (RYGBP) procedures.[13,27,28] Strategies to prevent leaks are important and can be summarized to the following: (1) avoid devascularizing the gastric pouch, (2) avoid excess tension on gastrojejunal anastomosis, (3) oversew the staple line, and (4) identify and repair leaks intraoperatively: check for leaks endoscopically (air insufflation) or with methylene blue dye lavage.

Despite intraoperative precautions, leaks that become apparent in the postoperative period and are inadequately managed can result in considerable morbidity and mortality. While some leaks result in overt peritonitis and sepsis, a proportion will have less obvious manifestations. It is necessary to have a high index of suspicion because a delay in diagnosis can lead to a general deterioration in the clinical condition of the patient. Excessive abdominal pain, shoulder tip pain, hiccups, and a sense of impending doom are ominous symptoms and, along with persistent tachycardia or tachypnea or arterial desaturation, should prompt a search for a leak.

There are good arguments for performing a routine early upper gastrointestinal contrast study (UGI). A UGI on the first postoperative day detects 33% of all leaks with a specificity of 100%,[29] with this early detection translating to lesser morbidity[30] and significantly shorter hospital stay.[29] The routine use of this test alerts the clinician to early leaks and forms an important component of decision-making. Absence of an early contrast study results in an average time to diagnosis of 7 days in gastrojejunal leaks and a mortality of approximately 10%.[31]

While a negative UGI on the first postoperative day is reassuring, it should be kept in mind that the peak incidence of leaks (by clinical or radiologic criteria) is on day 5,[13] and as such continued vigilance is needed. The advent of earlier hospital discharge owing to the benefits of laparoscopic gastric bypass has led to more patients returning to the hospital with leaks than was seen in the days of open gastric bypass. Furthermore, contrast studies lack a degree of sensitivity and therefore a negative result must be overridden by clinical findings of tachycardia or respiratory distress.[32]

A clinical pathway for the management of suspected leaks is outlined in Figure 26.2. Where the clinical condition of a patient suggests a possible leak, and in the absence of radiologic evidence, it is safer to inspect the peritoneal cavity, preferably laparoscopically. The principal value of a positive radiologic leak is in guiding the clinician in deciding whether to treat the leak operatively or nonoperatively. With a weight limit of 300 lb on most imaging tables, heavier patients will require operative management on the sole basis of clinical suspicion.

The clinical status of the patient and the radiologic features of the leak guide the management of this complication. In the presence of a contained or well-drained leak (Fig. 26.3) and in the absence of hemodynamic instability, there is ample evidence in supporting the nonoperative management of the majority of leaks. This approach is safe[13,33] and consists of placing the patient on nothing by mouth, on intravenous antibiotics, and feeding parenterally or via a gastrostomy tube placed under radiologic or laparoscopic guidance. Where there is a localized collection, these could be drained percutaneously under radiologic guidance.

In our experience of 40 leaks complicating 2,675 consecutive LRYGBP, only 10% of leaks required a laparotomy; 30% required laparoscopic feeding access placement (gastrostomy tube) while 60% were managed nonoperatively with no deaths or long-term morbidity in this latter group (unpublished data). This approach differs from the more aggressive stance advocated by others[34] proposing early operative intervention for all patients with leaks.

An uncontained leak (Fig. 26.4), or one associated with hemodynamic instability, requires urgent operative intervention. This should consist of repair of the leak, placement of drains, and the creation of enteral feeding access, in addition to intravenous antibiotics. In a hemodynamically stable patient, this could be undertaken laparoscopically, but

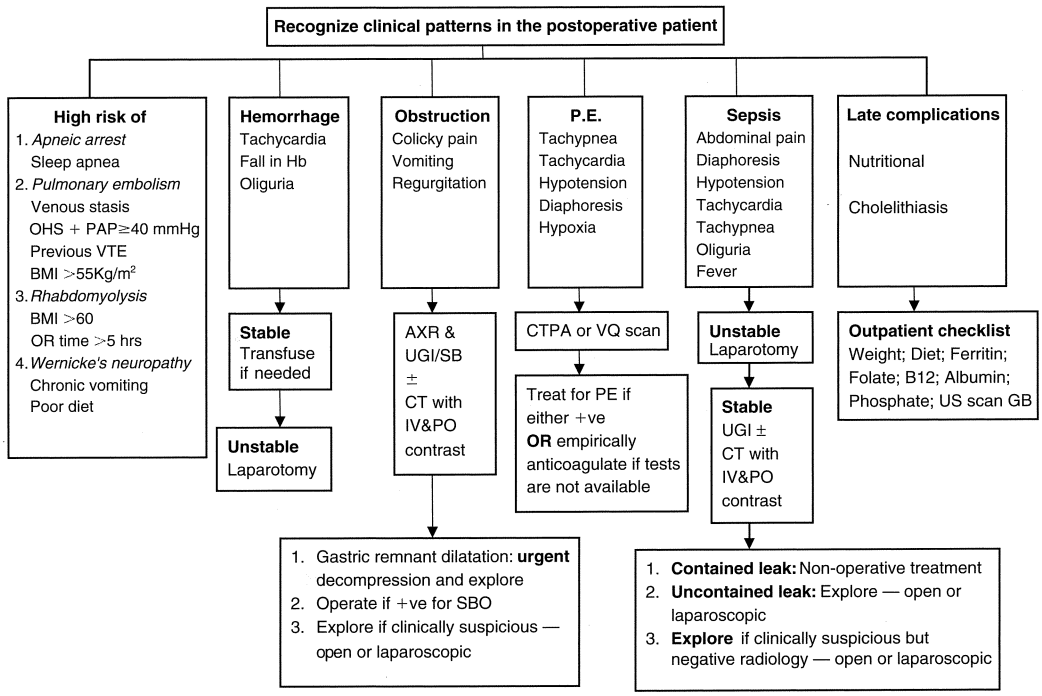

Figure 26.2. Algorithm for recognition and management of complications. See key for abbreviations.
Key: PE, pulmonary embolism; OHS, obesity hypoventilation syndrome; PAP, pulmonary artery pressure; VTE, venous thromboembolism; DVT, deep venous thrombosis; CTPA, computerized tomography pulmonary angiogram; OR, operating room; SBO, small bowel obstruction; UGI, upper gastrointestinal contrast study AXR, abdominal x-ray

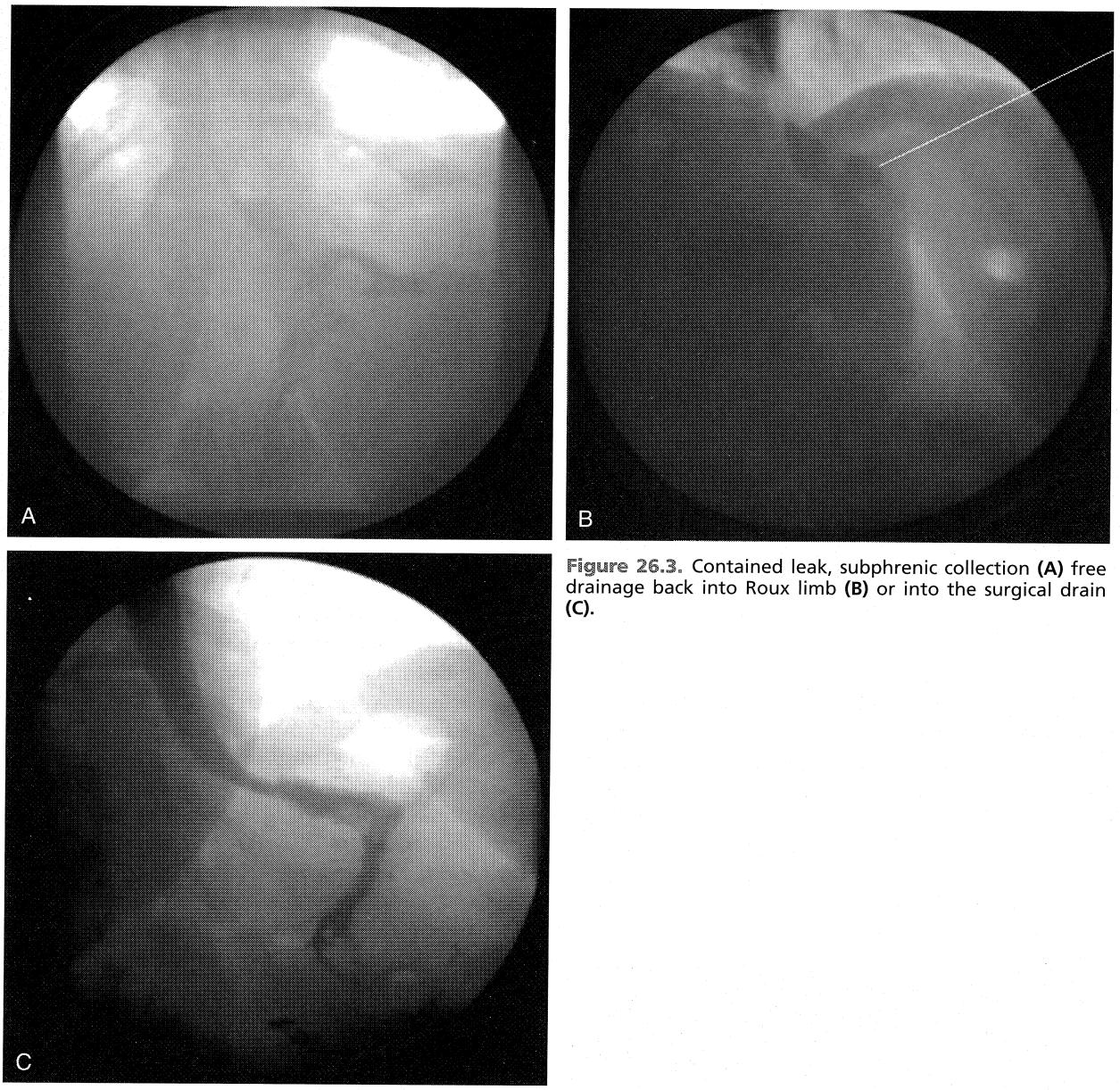

Figure 26.3. Contained leak, subphrenic collection **(A)** free drainage back into Roux limb **(B)** or into the surgical drain **(C)**.

where there is hypotension or cardiac dysrrhythmias, an open laparatomy is preferred.

Early Gastrointestinal Hemorrhage

Early upper gastrointestinal bleeding, within 24 hours following LRYGBP, is almost invariably reactionary hemorrhage from the gastric remnant staple line or the gastrojejunostomy[35] and represents inadequate hemostasis. Occasionally, the source is a splenic tear, the mesentery, or another intra-abdominal source. Use of low–molecular-weight heparins after induction of anesthesia can predispose to a general ooze or more significant hemorrhage.

Operative intervention may be necessary in about 40% of cases, depending on the rate of blood loss and hemodynamic stability.[35] Angiography with embolization is not a useful option in the highly vascular stomach with its multiple arterial supply.

Intestinal Obstruction and Internal Hernias

The rate of postoperative bowel obstruction following open bariatric surgery has been reported to range from 1 to 3%.[36] Similar rates have been reported with the laparoscopic approach (0.6–3.5%).[13,28] The mean time to presentation is variable, with some series reporting early obstruction (within 15 weeks) after a retrocolic approach,[37] but others reporting later obstruction with the antecolic approach (1 to 3 years).[38]

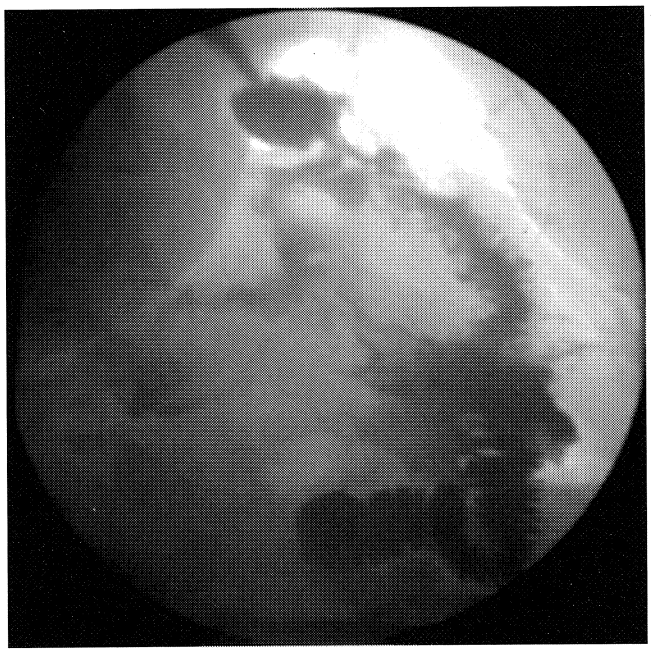

Figure 26.4. Uncontained leak from the gastrojejunostomy with diffuse spillage into the peritoneal cavity.

Unlike open bariatric procedures where adhesive obstruction is most common, intestinal obstruction after laparoscopic gastric bypass is caused primarily by nonadhesive disease. Causes of post-LRYGBP intestinal obstruction include internal hernias, formation of mesocolic constrictions, anastomotic strictures, intussusception, and volvulus or kinking of the bowel distal to the Roux limb at the site of the jejuno-jejunostomy.

Internal hernias constitute the most common cause of intestinal obstruction after laparoscopic bariatric surgery and may be explained by the relative lack of adhesions (which in the open situation facilitate fixation of the small intestine thus preventing its displacement) and technical issues related to closing mesenteric defects.[39] The commonest site of obstruction with a retrocolic Roux loop is the mesocolic window,[40] especially through the dorsal and lateral aspects of the defect,[37] which complicates 5% of these operations. The high proportion of adhesive obstruction (38%) in the series reported by Champion[38] may be a reflection of the fact that 55% of their obstructed patients had previously undergone an open abdominal procedure.

Internal hernias can occur at any of three defects created during gastric bypass. These defects include the transverse mesocolic window (Fig. 26.5), the jejuno-jejunostomy mesenteric defect (Fig. 26.6), and the space between the transverse mesocolon and the mesentery of the Roux limb (Petersen's defect), accounting for 67, 21, and 7.5% respectively. An additional 4.5% of internal hernias occur at more than one site.[39] Furthermore, rapid weight reduction following gastric bypass may result in decreased intraperitoneal fat that may enlarge the mesenteric defect, facilitating hernia formation.[33]

Intestinal obstruction may be incomplete or intermittent. Patients with internal hernias often have intermittent postprandial abdominal pain with contrast radiology that may be completely normal in up to 20%.[39] The initial investigation should be a UGI contrast study. Hold-up of contrast at the gastrojejunostomy should call for endoscopic evaluation, while Roux limb obstruction usually indicates obstruction at the mesocolic defect where a retrocolic route

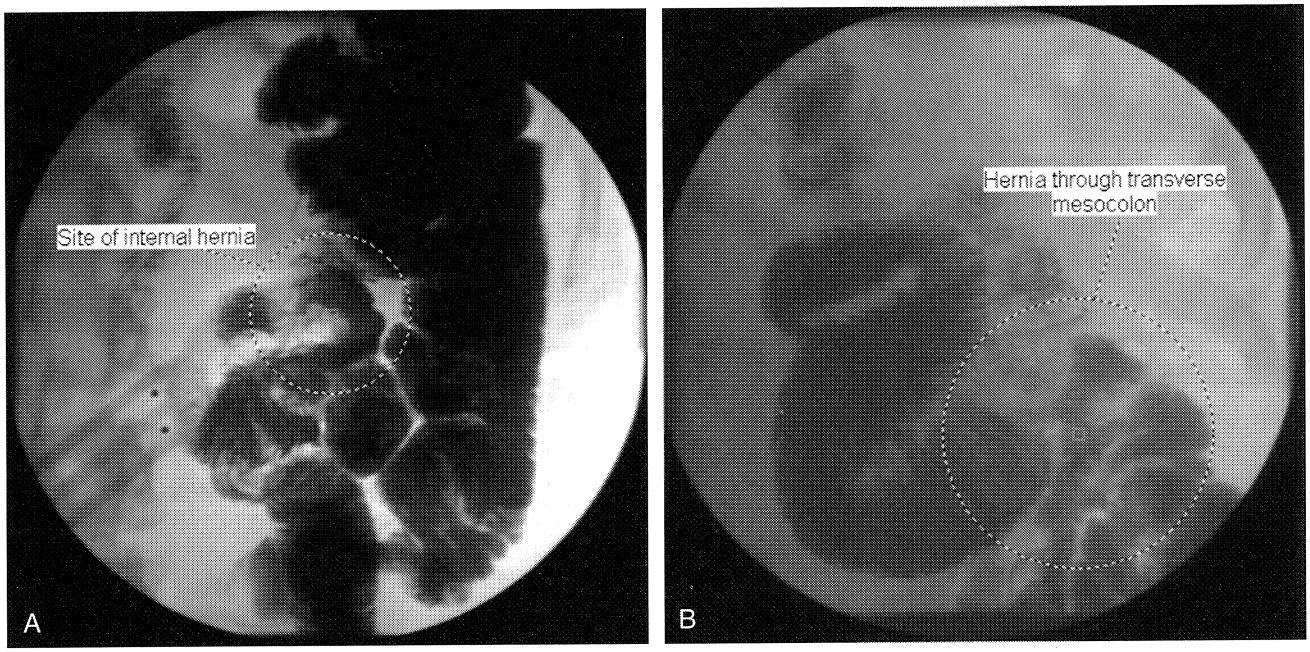

Figure 26.5. Small bowel obstruction caused by an internal hernia through a transverse mesocolic defect in a retrocolic LRYGBP.

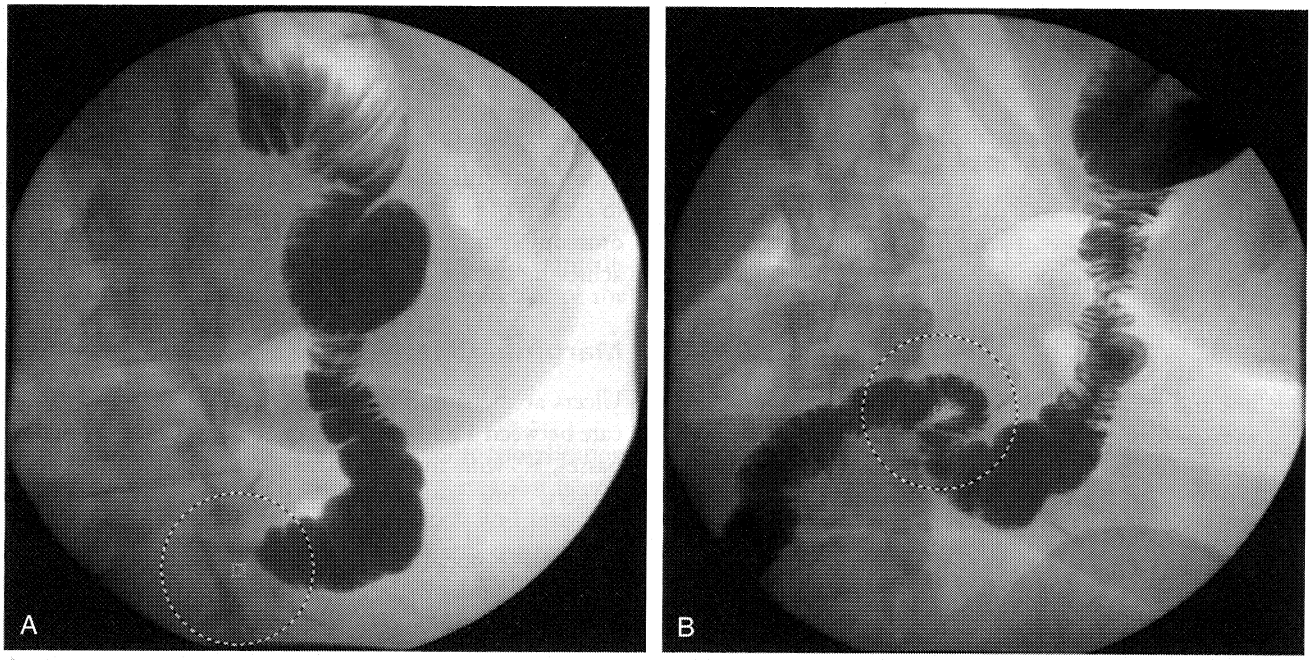

Figure 26.6. Internal hernia through the jejunostomy mesenteric defect causing incomplete small bowel obstruction.

has been used. A CT scan may be needed to supplement a UGI if there is any doubt about the diagnosis of obstruction, especially that distal to the Roux limb.

The onus on the clinician is to diagnose the presence or absence of intestinal obstruction. The precise cause of the obstruction cannot be made with any degree of safety preoperatively[41] and as such should not delay the decision to operate. All patients who present with intestinal obstruction must undergo an exploration of their peritoneal cavities, preferably laparoscopically. It is important for the bariatric surgeon to appreciate that this paradigm is different from the approach to the postoperative general surgical patient, where the dominance of adhesions justify an initial nonoperative approach.

Laparoscopic exploration should be the preferred initial approach and in the vast majority of obstructive complications, the underlying cause can be effectively treated,[28] aided by the relative lack of adhesions. Exploration, whether laparoscopic or open, should always include evaluation of bowel viability. Operative treatment primarily includes hernia reduction and closure of all mesenteric defects. Early and aggressive management of intestinal obstruction after gastric bypass is essential to prevent the dangers of closed-loop obstruction (Fig. 26.7) and acute dilatation of the remnant stomach. Morbidity remains high, with an incidence of perforations (9.1%) and death (1.6%) in one published series.[39]

Several strategies have been deployed to minimize the incidence of intestinal obstruction following gastric bypass. Internal hernias can be prevented by meticulous closure of all potential defects with a continuous running technique.[39] The

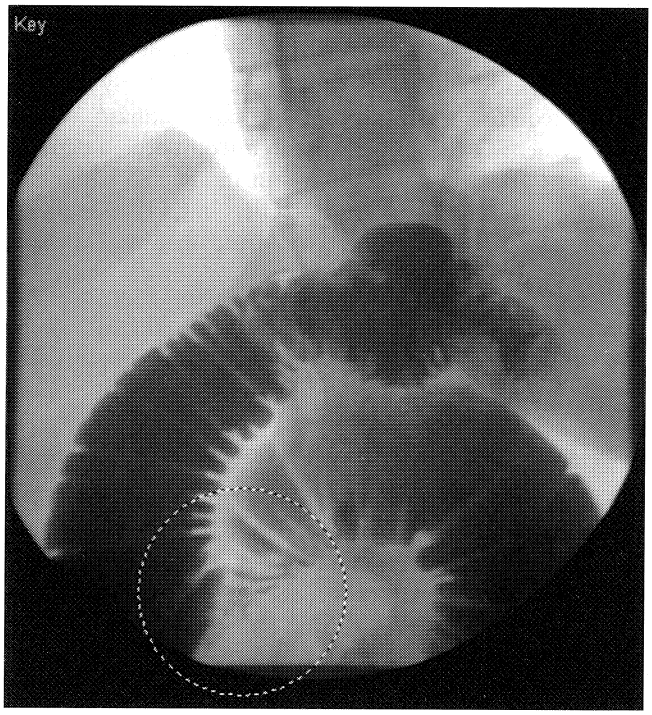

Figure 26.7. Closed-loop obstruction of the Roux limb caused by obstruction at the jejuno-jejunostomy.

adoption of nonabsorbable sutures resulted in a decrease of 50% in internal hernia formation.[28] Jejunum distal to the jejuno-jejunostomy may be prevented from kinking by placing a single nonabsorbable stitch between the jejunum immediately distal to the anastomosis and the stapled end of the

biliary limb ("Brolin stitch").[42] Antecolic placement of the Roux limb is associated with a lower risk of obstruction (0.43%) compared to a retrocolic loop (4.5%).[38]

Acute Gastric Dilatation

Acute dilatation of the gastric remnant is a rare (0.6%)[43] but potentially catastrophic event resulting from the closed-loop obstruction that follows obstruction of the biliopancreatic limb. Severe epigastric pain in conjunction with gastric dilatation on a plain abdominal x-ray or CT scan is diagnostic. It leads to rapid clinical deterioration with blowout of the staple line and hemodynamic instability that may appear *ab initio* with cardiac arrest.[44] It requires urgent percutaneous gastrostomy tube decompression[45] and subsequent management of the underlying biliary limb obstruction.

Rhabdomyolysis

Rhabdomyolysis is a rare complication typically affecting the superobese male patient who lies supine during a long operative procedure. It results from a gluteal compartment syndrome resulting in myonecrosis.[46–48] It carries a 50% mortality that rises to 100% in those progressing to renal failure.[46] Preventive measures include providing adequate buttock padding and reducing the duration of the surgical procedure, especially in the superobese male. Early identification requires serial creatinine phosphokinase (CPK) measurements. Median CPK rise in uncomplicated patients is 1,200 IU/L (SD 450–9,000), but in affected patients it ranges from 26,000 to 29,000 IU/L. If CPK rises above 5,000, aggressive hydration and forced mannitol diuresis should be started.[46]

MANAGEMENT OF LATE POSTOPERATIVE COMPLICATIONS

Gastrojejunal Anastomotic Strictures

Gastrojejunal anastomotic strictures complicate 3 to 12% of patients undergoing gastric bypasses. Patients present with gastric pouch obstruction at between 3 and 60 weeks after surgery,[49–51] peaking at between 8 and 10 weeks. These lesions are believed to result from ischemia at the site of the anastomosis or from subclinical anastomotic leaks. The effect of the type of anastomostic technique is not clear, with some reporting fewer strictures with the use of linear staplers or hand-sewn anastomosis[52] compared to circular staplers. Where circular staplers are used, a 25-mm anvil causes fewer strictures compared to a 21-mm anvil, without adversely affecting weight loss.[53] Others have reported equal stricture rates between linear and circular staplers.[54]

Painless postprandial regurgitation is the principal presenting symptom and should lead to a UGI contrast study. The diagnosis is confirmed by the inability to pass a 9-mm endoscope through the anastomosis.[51]

Treatment consists of endoscopic balloon dilatation (to 13 to 18 mm)[51,55] of the anastomosis. An average of two separate procedures relieves the obstruction in 95% of cases[49] for strictures presenting early, but restenosis occurs in 3% of these patients.[51] Strictures presenting after 3 months are still amenable to endoscopic dilatation, but up to a third of patients may require operative revision.[56] Fluoroscopy guided dilatation is an alternative treatment, achieving sustained patency in 71% after one dilatation.[57]

Marginal Ulcers

Ulcers at the site of the gastrojejunal anastomosis complicate between 1 and 16% (mean 10%) of isolated gastric bypasses,[58,59] with the highest risk in the first 2 months after surgery,[50] but may develop as late as 10 years after surgery.[50] Their etiology is multifactorial, being usually associated with use of nonabsorbable suture material[60] and a gastric pouch size larger than 50 ml.[61,62] Nonsteroidal anti-inflammatory drugs (NSAIDs) and *Helicobacter pylori* have also been associated with marginal ulcers. Patients who were preoperatively screened and treated for *H. pylori* had a significantly lower incidence of marginal ulcers at 3 years (2.4%) compared to those who were not screened for *H. pylori*.[63] Alcohol and smoking have also been causally implicated in patients with marginal ulcers.[64]

Nausea, vomiting, epigastric pain, and occult gastrointestinal bleeding are the primary features of this complication and should prompt endoscopic examination of the pouch.[60,61]

Pouch ulceration heals with proton pump inhibitors and/or sucralfate along with cessation of NSAID intake and smoking. In patients with a large pouch, ulcer recurrence with medical therapy alone is common and in this case consideration should be given to a reduction of the pouch size along with excision of the ulcer.[58,61] Where tension in the Roux limb and mucosal ischemia is thought to underlie the marginal ulcer, mobilization of the Roux limb should be undertaken by further division of a mesenteric arcade. Recurrent ulceration associated with a foreign body (including sutures) requires removal of the foreign body. Sutures may be removed endoscopically, but patients with eroding silastic or Marlex bands will require operative revision.

Incisional Hernias

Laparoscopic gastric bypass is notable for the rarity of incisional hernias (0.7%),[13] in contrast to the open procedure (15 to 20%).[65,66] However, while the 5-mm port is not associated with fascial hernias, the 10-mm ports can present with Richter's type of hernias.[67] Their early recognition is important because, unlike the broad-based incisional hernias complicating the open procedure, port site hernias following the laparoscopic approach have a small defect that renders them prone to intestinal strangulation. Patients may present with focal pain with or without colicky abdominal pain, but with-

out a palpable lump, especially if the panniculus is still large. Trocar site herniation is best demonstrated radiologically using anterior abdominal wall ultrasound or by CT scan, but it is often diagnosed laparoscopically.

Trocar site hernias are prevented by closing the fascia of 10-mm and 12-mm port sites using nonabsorbable sutures.[67] Closure of the fascia can be achieved using the laparoscopic fascia closure device.

Once recognized, hernias should be managed immediately, with laparoscopic reduction and suture repair of the hernia defect using a nonabsorbable material.

Wernicke's Polyneuropathy

Wernicke's polyneuropathy is an uncommon complication but it carries the potential to cause severe permanent neurological deficits. It manifests as limb weakness and unsteadiness of gait with diplopia and confusion, and typically follows a period of nutritional deprivation during which vitamin B-1 is not supplemented.[68] The usual clinical setting is a patient with nausea and vomiting of more than a week, a patient maintained on a prolonged clear liquid diet postoperatively without oral multivitamin supplementation, or a patient on intravenous feeding, typically in the first 6 to 12 weeks after surgery,[69] who is not provided a parenteral source of thiamine. This period corresponds to a nadir in thiamine and red cell transketolase levels that can occur after surgery.[70]

Because time is of the essence in minimizing residual disability, any patient with limb weakness should be promptly started on treatment with intravenous thiamine (500 mg every 8 hours) while investigations are conducted. It is important to note that attempts to rehydrate the patient with a solution containing glucose that does not include thiamine may in fact precipitate and aggravate the neurological damage. Plasma thiamine or red cell transketolase levels are unreliable[70] in establishing the diagnosis, which is made on clinical grounds. The routine addition of thiamine (100 mg) (in addition to 1 mg folic acid and multivitamin) to a 1 liter bag of lactated Ringers intravenous fluids once a day for patients with gastric bypass on a restricted oral intake is a safe practice designed to prevent this complication.

MANAGEMENT OF LONG-TERM COMPLICATIONS

Nutritional

Protein-Calorie Malnutrition

Protein-calorie malnutrition is an uncommon complication in the standard RYGBP. However, where a long-limbed or distal gastric bypass results in a 50-cm common channel, 28.5% of patients develop protein-calorie malnutrition.[71] A low serum albumin and phosphate level indicates depletion in total body proteins. In these situations, it is important to carefully evaluate the cause of this complication. This may arise from extreme restriction with pouch outlet obstruction or from malabsorption. In the former case there would be a prolonged history of vomiting or inability to tolerate solids and an upper gastrointestinal endoscopy and contrast examination is called for.

Mild protein-calorie malnutrition can be treated with protein supplements (skimmed milk or proprietary protein powder) and oral nonenteric-coated pancreatic enzymes to improve absorption. In the absence of a mechanical cause, excessive malabsorption may be reversed by conversion to a 150- to 200-cm common channel. In either case, it is important to pay attention to thiamine replacement and to avoid the refeeding syndrome that may accompany institution of full-strength feeding.[72–74] The latter results from the recommencement of food intake after a period of prolonged starvation and manifests as acute hypophosphatemia with cardiac failure[75,76] and typically occurs within 72 hours of refeeding.

Metabolic Bone Disease

Metabolic bone disease is an insidious long-term complication of gastric bypass and arises from calcium and/or vitamin D malabsorption.[77,78] Loss of bone mass, resulting from osteoporosis or osteomalacia, is preceded by secondary hyperparathyroidism and is asymptomatic until complicated by pathologic fractures. Elevation in serum immunoreactive parathyroid hormone associated with low or normal calcium levels is the earliest indicator of this condition and should prompt dietary supplementation with calcium and vitamin D and close monitoring of lumbar spine and femoral neck bone mineral density.[79]

Micronutrient Deficiency

Deficiency of iron (47%), folate (35%), and vitamin B-12 (37%) are common postoperatively and contribute to the development of anemia found in 54% of patients.[80] Iron deficiency anemia occurs in 22% of men and 51% of women,[80] with a higher proportion in menstruating women.[81] The routine administration of the following is recommended:

Multivitamin supplement

One adult multivitamin preparation daily effectively prevents folate and thiamine deficiency; however, the commonly available preparations do not contain sufficient iron or vitamin B-12 to prevent iron or B-12 deficiency,[80] and must therefore be obtained from alternate sources. Acute thiamine (B-1) deficiency may be precipitated by recurrent nausea and vomiting and may lead to a disabling polyneuropathy of the Wernicke's type.[71] If a patient is unable to take this supplement by mouth, it must be administered parenterally.

Calcium

The equivalent of 1,000 mg of elemental calcium, either as the carbonate or citrate salt, in two divided doses is commonly used to prevent bone loss. However, its efficacy has not been formally evaluated in this group of patients.

Iron

Oral iron has been shown to prevent and correct depleted iron stores after gastric bypass. Most studies have employed between 50 and 100 mg of elemental iron, with a 500 mg vitamin C supplement to improve iron absorption.[82,83] The dose of iron may have to be doubled in menstruating women. However, anemia associated with iron deficiency responds to oral iron therapy in only 57% of menstruating patients with microcytic indices.[83]

Vitamin B-12

Daily supplementation with 500 mcg of B-12 orally is enough to prevent B-12 deficiency in more than 80% of patients.[80] Intramuscular administration is only needed where compliance is poor. It should be noted that B-12 deficiency often remains asymptomatic and may not manifest as megaloblastic anemia (0 to 7%),[80,84,85] in part because of folate supplements.

Zinc

There is no evidence for the routine use of dietary zinc supplement. However, in cases of patients with hair loss following LRYGBP, the likelihood of zinc deficiency should be borne in mind.

Cholelithiasis (Including Common Bile Duct Stones)

Routine cholecystectomy concomitant with a LRYGBP remains controversial.[86,87] However, what is not in doubt is that weight loss following LRYGBP is accompanied by a rise in incidence of gallstones, with 38 to 52.8% of patients who did not have stones preoperatively going on to develop stones in the 12 months after surgery,[88,89] sometimes as early as 3 months postoperatively.[90] Between 15 and 27% of all patients, irrespective of gallstone status at LRYGBP surgery, will require urgent cholecystectomy within 3 years,[84,88] while 42% of those initially without gallstones will require urgent cholecystectomy during this period.[88]

While the safety and feasibility of undertaking laparoscopic cholecystectomy and LRYGBP has been established, the combined procedure nearly doubles length of hospital stay and adds about 50 minutes to the operation.[91,92] The prophylactic use of oral ursodiol at 600 mg daily for 6 months after LRYGBP significantly reduces incidence of gallstones (2% vs. 32% in placebo, $P < .01$).[93] However, efficacy is limited by poor patient compliance.[94] Thus, the decision to prophylactically remove the gallbladder will have to be made by the surgeon based on the likely compliance of the patient to postoperative ursodiol versus the risk of prolonging the procedure, especially in the superobese.

The management of post–gastric bypass choledocholithiasis becomes difficult because of loss of endoscopic access to the duodenum. If the remnant stomach is anchored to the anterior abdominal wall, preferably with a radiological marker,[95] this may provide a safe point for percutaneous access to the stomach for endoscopic retrograde cholangiopancreatography (ERCP). A laparoscopic transgastric approach for ERCP has also been described.[96]

Weight Regain

Failure of weight loss after LRYGBP occurs in 5 to 10% of patients. It is thought to arise from a progressive dilatation of the pouch outlet and the pouch itself and is probably related to poor eating habits.[97,98] Patients can also defeat the restrictive component of the operation by consuming sweets or other high-calorie foods despite feeling full. This can be a difficult problem with no satisfactory solution. The best results are achieved by frequent patient supervision and close cooperation with a nutritionist, a psychologist, and support groups. The value of reducing pouch or stoma size either operatively or endoscopically remains unproven.[99]

Late Gastrointestinal Hemorrhage

Late upper gastrointestinal hemorrhage has been reported after a mean of 15.5 years (range: 13–17 years) and may be the result of new pathology in the duodenum or remnant stomach.[100] Late hemorrhage is noteworthy for the diagnostic dilemma it can present, especially because the remnant stomach is generally inaccessible to endoscopic examination. Intraoperative endoscopy may be helpful.[100] However, where available, an enteroscope or pediatric colonoscope can be used to gain access to the duodenum and remnant stomach through a transoral route via the Roux limb and may be both diagnostic and therapeutic.

WORKUP OF PATIENTS WITH A SUSPECTED COMPLICATION

Early identification of dangerous complications is greatly helped by having a postoperative routine, a checklist, and an algorithm to act on the information gathered.

Unusual postoperative abdominal pain, left shoulder-tip pain, hiccups, vomiting, or a sense of impending doom should all be a call to action as should tachycardia (>100), tachypnoea, or oliguria.

The algorithm in Figure 26.2 outlines a plan for investigating a patient who is suspected of having developed a complication. It should be noted that not all the cited laboratory or radiological indicators are necessary, but every surgeon should develop his or her own algorithm.

REFERENCES

1. Flemons WW: Clinical practice. Obstructive sleep apnea. *N Engl J Med* 2002; 347:498–504.
2. Johns MW: A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991; 14:540–545.

3. Maislin G, et al: A survey screen for prediction of apnea. *Sleep* 1995; 18:158–66.
4. Polysomnography Task Force, ASDASoPC: Practice parameters for the indications for polysomnography and related procedures. Polysomnography Task Force, American Sleep Disorders Association Standards of Practice Committee. *Sleep* 1997; 20:406–22.
5. Rennotte MT, Baele P, Aubert G, Rodenstein DO: Nasal continuous positive airway pressure in the perioperative management of patients with obstructive sleep apnea submitted to surgery. *Chest* 1995; 107:367–74.
6. Ebeo CT, Benotti PN, Byrd RP Jr, et al: The effect of bi-level positive airway pressure on postoperative pulmonary function following gastric surgery for obesity. *Respir Med* 2002; 96:672–6.
7. Huerta S, et al: Safety and efficacy of postoperative continuous positive airway pressure to prevent pulmonary complications after Roux-en-Y gastric bypass. *J Gastrointest Surg* 2002; 6:354–8.
8. Benumof JL: Obstructive sleep apnea in the adult obese patient: implications for airway management. *Anesthesiol Clin North America* 2002; 20:789–811.
9. Cullen DJ: Obstructive sleep apnea and postoperative analgesia—a potentially dangerous combination. *J Clin Anesth* 2001; 13:83–5.
10. Tabboush ZS, Ayash RH, Badran HM: When fiberoptic bronchoscopy is indicated in the management of postoperative atelectasis. *Acta Anaesthesiol Scand* 1998; 42:384.
11. Massard G, Wihlm JM: Postoperative atelectasis. *Chest Surg Clin N Am* 1998; 8(8):503–28.
12. Wu EC, Barba CA: Current practices in the prophylaxis of venous thromboembolism in bariatric surgery. *Obes Surg* 2000; 10:7–13; discussion 14.
13. Schauer PR, Ikramuddin S, Gourash W, et al: Outcomes after laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Ann Surg* 2000; 232:515–29.
14. Fobi MA, Lee H, Holness R, Cabinda D: Gastric bypass operation for obesity. *World J Surg* 1998; 22:925–35.
15. Papavramidis ST, Milias C: Complications after vertical gastroplasty with artificial pseudopylorus in the treatment of morbid obesity: a 7-year experience. *Obes Surg* 1999; 9:535–538.
16. Melinek J, Livingston E, Cortina G, Fishbein MC: Autopsy findings following gastric bypass surgery for morbid obesity. *Arch Pathol Lab Med* 2002; 126:1091–1095 .
17. Bajardi G, et al: Postoperative venous thromboembolism in bariatric surgery. *Minerva Chir* 1993; 48:539–542.
18. Sugerman,HJ, et al: Risks and benefits of gastric bypass in morbidly obese patients with severe venous stasis disease. *Ann Surg* 2001; 234:41–46.
19. Sapala JA, Wood MH, Schuhknecht MP, Sapala MA: in *IFSO Obesity Surgery Congress* (Salamanca, Spain, 2003).
20. Sugerman HJ, et al: Long-term effects of gastric surgery for treating respiratory insufficiency of obesity. *Am J Clin Nutr* 1992; 55:597S–601S.
21. Gargiulo NJ, et al: in *Vascular and Endovascular Surgery Issues, Technology and Horizons (VEITH)* (New York, 2003).
22. Greenfield LJ, Scher LA, Elkins RC: KMA-Greenfield filter placement for chronic pulmonary hypertension. *Ann Surg* 1979; 189:560–5.
23. MacGregor MI, Block AJ, Ball WC Jr: Topics in clinical medicine: serious complications and sudden death in the Pickwickian syndrome. *Johns Hopkins Med J* 1970; 126:279–95.
24. Stone E, et al: Use of computed tomography pulmonary angiography in the diagnosis of pulmonary embolism in patients with an intermediate probability ventilation/perfusion scan. *Intern Med J* 2003; 33:74–8.
25. Kearon C, Hirsh J: Management of anticoagulation before and after elective surgery. *N Engl J Med* 1997; 336:1506–11.
26. Low-molecular-weight heparin in the treatment of patients with venous thromboembolism. The Columbus Investigators. *N Engl J Med* 1997; 337:657–62.

26a Kolb G, et al: Reduction of venous thromboembolism following prolonged prophylaxis with the low molecular weight heparin Certoparin after endoprosthetic joint replacement or osteosynthesis of the lower limb in elderly patients. *Thromb Haemust* 2003; 90:1100–5

26b Krotenberg R. Current recommendations for extended out-of-hospital thromboprophylaxis following total hip arthroplasty. *Am J Orthop* (2004); 33:180–4.

27. Fernandez AZ Jr, et al: Experience with over 3,000 open and laparoscopic bariatric procedures: Multivariate analysis of factors related to leak and resultant mortality. *Surg Endosc* 2003.
28. Higa KD, Boone KB, Ho T: Complications of the laparoscopic Roux-en-Y gastric bypass: 1,040 patients—what have we learned? *Obes Surg* 2000; 10:509–513.
29. Sims TL, Mullican MA, Hamilton EC, et al: Routine upper gastrointestinal Gastrografin swallow after laparoscopic Roux-en-Y gastric bypass. *Obes Surg* 2003; 13:66–72.
30. Ovnat A, Peiser J, Solomon H, Charuzi I: Early detection and treatment of a leaking gastrojejunostomy following gastric bypass. *Isr J Med Sci* 1986; 22:556–8.
31. Marshall JS, Srivastava A, Gupta SK, et al: Roux-en-Y gastric bypass leak complications. *Arch Surg* 2003; 138, 520–524.
32. Hamilton EC, et al: Clinical predictors of leak after laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Surg Endosc* 2003; 17:679–84.
33. Blachar A, Federle MP, Pealer KM, et al: Gastrointestinal complications of laparoscopic Roux-en-Y gastric bypass surgery: clinical and imaging findings. *Radiology* 2002; 223:625–32.
34. Arteaga JR, Huerta S, Livingston EH: Management of gastrojejunal anastomotic leaks after Roux-en-Y gastric bypass. *Am Surg* 2002; 68:1061–5.
35. Nguyen NT, Rivers R, Wolfe BM: Early gastrointestinal hemorrhage after laparoscopic gastric bypass. *Obes Surg* 2003; 13:62–5.
36. Brolin RE, Kenler HA, Gorman JH, Cody RP: Long-limb gastric bypass in the superobese. A prospective randomized study. *Ann Surg* 1992; 215:387–95.
37. Filip JE, Mattar SG, Bowers SP, Smith CD: Internal hernia formation after laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Am Surg* 2002; 68:640–3.
38. Champion JK, Williams M: Small bowel obstruction and internal hernias after laparoscopic Roux-en-Y gastric bypass. *Obes Surg* 2003; 13, 596–600.
39. Higa KD, Ho T, Boone KB: Internal hernias after laparoscopic Roux-en-Y gastric bypass: incidence, treatment and prevention. *Obes Surg* 2003; 13:350–4.
40. Felsher J, Brodsky J, Brody F: Small bowel obstruction after laparoscopic Roux-en-Y gastric bypass. *Surgery* 2003;134: 501–5.
41. Blachar A. Federle MP: Internal hernia: an increasingly common cause of small bowel obstruction. *Semin Ultrasound CT MR* 2002; 23:174–83.
42. Brolin RE: The antiobstruction stitch in stapled Roux-en-Y enteroenterostomy. *Am J Surg* 1995; 169:355–7.
43. Jones KB: Biliopancreatic limb obstruction in gastric bypass at or proximal to the jejunojejunostomy: a potentially deadly, catastrophic event. *Obes Surg* 1996; 6:485–493.
44. Olbers T, Lonroth H, Fagevik-Olsen M, Lundell L: Laparoscopic gastric bypass: development of technique, respiratory function, and long-term outcome. *Obes Surg* 2003; 13: 364–70.
45. Maingot R, Zinner M: *Maingot's abdominal operations.* Appleton and Lange, Stamford, Conn., 1997.

46. Bostanjian D, Anthone GJ, Hamoui N, Crookes PF: Rhabdomyolysis of gluteal muscles leading to renal failure: a potentially fatal complication of surgery in the morbidly obese. *Obes Surg* 2003; 13:302–5.
47. Wiltshire JP, Custer T: Lumbar muscle rhabdomyolysis as a cause of acute renal failure after Roux-en-Y gastric bypass. *Obes Surg* 2003; 13:306–13.
48. Torres-Villalobos G, et al: Pressure-induced rhabdomyolysis after bariatric surgery. *Obes Surg* 2003; 13:297–301.
49. Go MR, Muscarella P 2nd, Needleman BJ, et al: Endoscopic management of stomal stenosis after Roux-en-Y gastric bypass. *Surg Endosc* 2003; .
50. Sanyal AJ, Sugerman HJ, Kellum JM, et al: Stomal complications of gastric bypass: incidence and outcome of therapy. *Am J Gastroenterol* 1992; 87:1165–9.
51. Ahmad J, Martin J, Ikramuddin S, et al: Endoscopic balloon dilation of gastroenteric anastomotic stricture after laparoscopic gastric bypass. *Endoscopy* 2003; 35:725–8.
52. Gonzalez R, Lin E, Venkatesh KR, et al: Gastrojejunostomy during laparoscopic gastric bypass: analysis of 3 techniques. *Arch Surg* 2003; 138:181–4.
53. Nguyen NT, Stevens CM, Wolfe BM: Incidence and outcome of anastomotic stricture after laparoscopic gastric bypass. *J Gastrointest Surg* 2003; 7:997–1003.
54. Shope TR, Cooney RN, McLeod J, et al: Early results after laparoscopic gastric bypass: EEA vs GIA stapled gastrojejunal anastomosis. *Obes Surg* 2003; 13:355–9.
55. Barba CA, Butensky MS, Lorenzo M, Newman R: Endoscopic dilation of gastroesophageal anastomosis stricture after gastric bypass. *Surg Endosc* 2003; 17:416–20.
56. Sataloff DM, Lieber CP, Seinige UL: Strictures following gastric stapling for morbid obesity: results of endoscopic dilatation. *Am Surg* 1990; 56:167–74.
57. Holt PD, de Lange EE, Shaffer HA, Jr: Strictures after gastric surgery: treatment with fluoroscopically guided balloon dilatation. *AJR Am J Roentgenol* 1995; 164:895–9.
58. Sapala JA, Wood MH, Sapala MA, Flake TM, Jr: Marginal ulcer after gastric bypass: a prospective 3-year study of 173 patients. *Obes Surg* 1998; 8:505–16.
59. MacLean LD, Rhode BM, Nohr C, et al: Stomal ulcer after gastric bypass. *J Am Coll Surg* 1997;185:1–7.
60. Capella JF, Capella RF: Gastro-gastric fistulas and marginal ulcers in gastric bypass procedures for weight reduction. *Obes Surg* 1999; 9:22–28.
61. Printen KJ, Scott D, Mason EE: Stomal ulcers after gastric bypass. *Arch Surg* 1980; 115:525–7.
62. Jordan JH, Hocking MP, Rout WR, Woodward ER: Marginal ulcer following gastric bypass for morbid obesity. *Am Surg* 1991; 57:286–8.
63. Schirmer B, Erenoglu C, Miller A: Flexible endoscopy in the management of patients undergoing Roux-en-Y gastric bypass. *Obes Surg* 2002; 12:634–8.
64. Scopinaro N, et al: Biliopancreatic diversion for obesity at eighteen years. *Surgery* 1996; 119:261–8.
65. Sugerman HJ, et al: Greater risk of incisional hernia with morbidly obese than steroid-dependent patients and low recurrence with prefascial polypropylene mesh. *Am J Surg* 1996; 171:80–4.
66. Wittgrove AC, Clark GW: Laparoscopic gastric bypass, Roux-en-Y 500 patients: technique and results, with 3–60 month follow-up. *Obes Surg* 2000; 10:233–9.
67. Matthews BD, Heniford BT, Sing RF: Preperitoneal Richter hernia after a laparoscopic gastric bypass. *Surg Laparosc Endosc Percutan Tech* 2001; 11:47–9.
68. Maryniak O: Severe peripheral neuropathy following gastric bypass surgery for morbid obesity. *Can Med Assoc J* 1984; 131, 119–20.
69. Chaves LC, Faintuch J, Kahwage S, Alencar Fde A: A cluster of polyneuropathy and Wernicke-Korsakoff syndrome in a bariatric unit. *Obes Surg* 2002; 12:328–34.
70. Villar HV, Ranne RD: Neurologic deficit following gastric partitioning: possible role of thiamine. *JPEN J Parenter Enteral Nutr* 1984; 8, 575–8.
71. Sugerman HJ, Kellum JM, DeMaria EJ: Conversion of proximal to distal gastric bypass for failed gastric bypass for superobesity. *J Gastrointest Surg* 1997; 1:517–525.
72. Jacob HS, et al: Hyperalimentation hypophosphatemia: hematologic-neurologic dysfunction due to ATP depletion. *Trans Assoc Am Physicians* 1973; 86:143–53.
73. Mason EE: Starvation injury after gastric reduction for obesity. *World J Surg* 1998; 22:1002–7.
74. Terlevich A, et al: Refeeding syndrome: effective and safe treatment with phosphates polyfusor. *Aliment Pharmacol Ther* 2003; 17:1325–9.
75. Schnitker MA, Mattman PE, Bliss TL: A clinical study of malnutrition in Japanese prisoners of war. *Ann Intern Med* 1951; 35:69–96.
76. Crook MA, Hally V, Panteli JV: The importance of the refeeding syndrome. *Nutrition* 2001; 17:632–7.
77. Goldner WS, O'Dorisio TM, Dillon JS, Mason EE: Severe metabolic bone disease as a long-term complication of obesity surgery. *Obes Surg* 2002; 12:685–92.
78. Ott MT, et al: Biochemical evidence of metabolic bone disease in women following Roux-Y gastric bypass for morbid obesity. *Obes Surg* 1992; 2:341–348.
79. Shaker JL, Norton AJ, Woods MF, et al: Secondary hyperparathyroidism and osteopenia in women following gastric exclusion surgery for obesity. *Osteoporos Int* 1991; 1:177–81.
80. Brolin RE, et al: Are vitamin B-12 and folate deficiency clinically important after Roux-en-Y gastric bypass? *J Gastrointest Surg* 1998; 2:436–42.
81. Sugerman HJ: Bariatric surgery for severe obesity. *J Assoc Acad Minor Phys* 2001; 12:129–36.
82. Rhode BM, Shustik C, Christou NV, MacLean LD: Iron absorption and therapy after gastric bypass. *Obes Surg* 1999; 9:17–21.
83. Brolin RE, et al: Prophylactic iron supplementation after Roux-en-Y gastric bypass: a prospective, double-blind, randomized study. *Arch Surg* 1998; 133:740–4.
84. Amaral JF, Thompson WR: Gallbladder disease in the morbidly obese. *Am J Surg* 1985; 149:551–7.
85. Halverson JD: Micronutrient deficiencies after gastric bypass for morbid obesity. *Am Surg* 1986; 52:594–8.
86. Mason EE, Renquist KE: Gallbladder management in obesity surgery. *Obes Surg* 2002; 12:222–9.
87. Fobi M, et al: Prophylactic cholecystectomy with gastric bypass operation: incidence of gallbladder disease. *Obes Surg* 2002; 12:350–3.
88. Shiffman ML, Sugerman HJ, Kellum JM, Moore EW: Changes in gallbladder bile composition following gallstone formation and weight reduction. *Gastroenterology* 1992; 103:214–21.
89. Iglezias Brandao de Oliveira C, Adami Chaim E, Borges da Silva B: Impact of rapid weight reduction on risk of cholelithiasis after bariatric surgery. *Obes Surg* 2003; 13:625–8.
90. Worobetz LJ, Inglis FG, Shaffer EA: The effect of ursodeoxycholic acid therapy on gallstone formation in the morbidly obese during rapid weight loss. *Am J Gastroenterol* 1993; 88:1705–10.
91. Hamad GG, Ikramuddin S, Gourash WF, Schauer PR: Elective cholecystectomy during laparoscopic Roux-en-Y gastric bypass: is it worth the wait? *Obes Surg* 2003; 13:76–81.
92. Papavramidis S, et al: Laparoscopic cholecystectomy after bariatric surgery. *Surg Endosc* 2003; .

93. Sugerman H. J, et al: A multicenter, placebo-controlled, randomized, double-blind, prospective trial of prophylactic ursodiol for the prevention of gallstone formation following gastric-bypass-induced rapid weight loss. *Am J Surg* 1995; 169: 91–97.
94. Wudel LJ, Jr, et al: Prevention of gallstone formation in morbidly obese patients undergoing rapid weight loss: results of a randomized controlled pilot study. *J Surg Res* 2002; 102: 50–6.
95. Fobi MA, Chicola K, Lee H: Access to the bypassed stomach after gastric bypass. *Obes Surg* 1995; 8:289–95.
96. Peters M, Papasavas PK, Caushaj PF, et al: Laparoscopic transgastric endoscopic retrograde cholangiopancreatography for benign common bile duct stricture after Roux-en-Y gastric bypass. *Surg Endosc* 2002; 16:1106.
97. MacLean LD, Rhode BM, Nohr CW: Late outcome of isolated gastric bypass. *Ann Surg* 2000; 231:524–8.
98. Holzwarth R, Huber D, Majkrzak A, Tareen B: Outcome of gastric bypass patients. *Obes Surg* 2002; 12:261–4.
99. Spaulding L: Treatment of dilated gastrojejunostomy with sclerotherapy. *Obes Surg* 2003; 13:254–7.
100. Braley SC, Nguyen NT, Wolfe BM: Late gastrointestinal hemorrhage after gastric bypass. *Obes Surg* 2002; 12:404–7.