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should undergo a second-look laparotomy in a 24- to 48-h period. After revascularization, peristalsis and return of a pink color of the bowel wall should be observed. Palpation of major arterial mesenteric vessels can be performed, as well as applying a Doppler flowmeter to the antimesenteric border of the bowel wall, but neither is a definitive indicator of viability. Acute-on-chronic mesenteric ischemia typically involves the orifice of the SMA. Therefore, the entire small bowel is compromised. Revas cularization with an endovascular, open, and/or a hybrid approach should be individualized based the patient's critical status, comorbidi ties, and anatomy. Endovascular stenting, suction thrombectomy, and/ or a thrombolysis catheter should be considered for intervention. The bowel should be evaluated for viability typically via a laparoscopic or exploratory laparotomy. Nonocclusive or vasospastic mesenteric ischemia presents with gen eralized abdominal pain, anorexia, bloody stools, and abdominal dis tention. Often these patients are obtunded, and physical findings may not assist in the diagnosis or be obscured by the underlying etiology. The presence of leukocytosis, metabolic acidosis, and/or lactic acidosis is useful in support of the diagnosis of advanced intestinal ischemia; however, these markers may not be indicative of either reversible isch emia or frank necrosis. Emergent admission to a monitored bed or intensive care unit is recommended for resuscitation and further evaluation, and the patient should be started on broad-spectrum antibiotics. Anticoagulation is not recommended as the goal is resuscitation to maintain hemody namics. For select patients, intramesenteric infusion of vasodilators such as papaverine, prostaglandins, or nitroglycerin can be used for the reversal of spasm and mesenteric ischemia, but the priority should be resuscitation and treatment of the underlying pathology. If ischemic colitis is a concern, colonoscopy should be considered to assess the integrity of the colonic mucosa. Ischemia of the colonic mucosa is graded as mild with minimal mucosal erythema or as moderate with pale mucosal ulcerations and evidence of extension to the muscular layer of the bowel wall. Severe ischemic colitis presents with severe ulcerations resulting in black or green discoloration of the mucosa, consistent with full-thickness bowel-wall necrosis. Ischemic colitis is optimally treated with resection of the ischemic bowel and formation of a proximal stoma. Onset of mesenteric venous thrombosis can be acute or subacute based on the location of thrombosis in the splanchnic circulation. Patients often present with vague abdominal pain associated with nau sea and vomiting. Physical examination findings include abdominal distention with mild to moderate tenderness and signs of dehydra tion. Findings on CT delayed venous phase include diffuse bowel-wall thickening and thrombus within the splanchnic system. IV therapeutic anticoagulation, broad-spectrum antibiotics, and correction of electro lyte

abnormalities should be performed. Surgical intervention is not performed unless there is evidence of peritonitis and/or bowel perfora tion. If there is evidence of bowel compromise, an exploratory laparot omy should be performed, with resection of the compromised bowel. Second-look laparotomy after 24–48 h should be attempted because anticoagulation can help prevent resection of viable bowel. Hyperco agulability testing should be performed, and if underlying inherited disorders are diagnosed, life-long anticoagulation is recommended. Acknowledgments Rizwan Ahmed contributed to the 19th edition and Maryam Khan and Jaideep Das Gupta contributed to the 21st edition. ■ ■FURTHER READING Bala M et al: Acute mesenteric ischemia: Updated guidelines of the World Society of Emergency Surgery. World J Emerg Surg 17:54, 2022. Cirillo-Penn NC et al: Midterm clinical outcomes of retrograde open mesenteric stenting for mesenteric ischemia. Ann Vasc Surg 89:20, 2023. Deng QW et al: Risk factors for postoperative acute mesenteric isch emia among adult patients undergoing cardiac surgery: A systematic review and meta-analysis. J Crit Care 42:294, 2017.

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Acute Intestinal

Obstruction ■ ■EPIDEMIOLOGY Globally, although the incidence and prevalence of acute intestinal obstruction have increased over the past two decades, morbidity and mortality appear to be decreasing. Diagnosis remains challenging. The extent of mechanical obstruction is typically described as partial, high grade, or complete—generally correlating with the risk of complica tions and the urgency with which the underlying disease process must be addressed. Obstruction is also commonly described as being either “simple” or, alternatively, “strangulated” if vascular insufficiency and intestinal ischemia are evident. CHAPTER 341 Acute intestinal obstruction occurs either mechanically from block age or from intestinal dysmotility when there is no blockage. In the latter instance, the abnormality is functional. Mechanical bowel obstruction may be caused by extrinsic processes, intrinsic abnor malities of the bowel wall, or intraluminal abnormalities (Table 341-1). Within each of these broad categories are many diseases that can impede intestinal propulsion. Intrinsic diseases that can cause intes tinal obstruction are usually congenital, inflammatory, neoplastic, or traumatic in origin, although intussusception and radiation injury can also be etiologic. Acute Intestinal Obstruction Acute intestinal obstruction accounts for ~1–3% of all hospitaliza tions and a quarter of all urgent or emergent general surgery admis sions. Approximately 80% of cases involve the small bowel, and about one-third of these patients show evidence of significant ischemia. The mortality rate for patients with strangulation who are operated on within 24–30 h of the onset of symptoms is ~8% but triples shortly thereafter. Extrinsic diseases most commonly cause mechanical obstruction of the small intestine. In the United States and Europe, almost all cases are caused by postoperative adhesions, carcinomatosis, or herniation of the anterior abdominal wall. Carcinomatosis most often originates from the ovary, but can originate from the pancreas, stomach, or colon. Rarely, metastasis from distant organs like

the breast and skin can also occur. Adhesions are responsible for the majority of cases of early post operative obstruction that require intervention. Approximately 20% of patients who were treated conservatively and between 5 and 30% of patients who were managed operatively will require readmission within 10 years for recurrence. Open operations of the lower abdomen, including appendectomy and colorectal and gynecologic procedures, are especially likely to create adhesions that can cause bowel obstruction (Table 341-2). Although laparoscopic procedures may generate fewer postopera tive adhesions compared with open surgery, the risk of obstructive adhesion formation is not eliminated. The risk of internal herniation is increased by abdominal procedures such as laparoscopic or open Roux-en-Y gastric bypass. Volvulus, which occurs when bowel twists on its mesenteric axis, can cause partial or complete obstruction and vascular insufficiency

TABLE 341-1 Most Common Causes of Acute Intestinal Obstruction Extrinsic Disease Adhesions (especially due to previous abdominal surgery), internal or external hernias, neoplasms (including carcinomatosis and extraintestinal malignancies, mostly commonly ovarian), endometriosis or intraperitoneal abscesses, and idiopathic sclerosis Intrinsic Disease Congenital (e.g., malrotation, atresia, stenosis, intestinal duplication, cyst formation, and congenital bands—the latter rarely in adults) Inflammation (e.g., inflammatory bowel disease, especially Crohn’s disease, but also diverticulitis, radiation, tuberculosis, lymphogranuloma venereum, and schistosomiasis) Neoplasia (note: primary small-bowel cancer is rare; obstructive colon cancer may mimic small-bowel obstruction if the ileocecal valve is incompetent) Traumatic (e.g., hematoma formation, anastomotic strictures) Other, including intussusception (where the lead point is typically a polyp or tumor in adults), volvulus, obstruction of duodenum by superior mesenteric artery, radiation or ischemic injury, and aganglionosis, which is Hirschsprung’s disease Intraluminal Abnormalities Bezoars, feces, foreign bodies including inspissated barium, gallstones (entering the lumen via a cholecystoenteric fistula), enteroliths affecting the sigmoid colon most commonly comprising approximately two-thirds of all cases of volvulus and 4% of all cases of large-bowel obstruction. The cecum and terminal ileum can also volvulize, or the cecum alone may be involved as a cecal bascule. Risk factors include institutionalization, the presence of neuropsychiatric condi tions requiring psychotropic medication, chronic constipation, and aging; patients typically present in their seventies or eighties. Colonic volvulus is more common in Eastern Europe, Russia, and Africa than it is the United States. It is rare for adhesions or hernias to obstruct the colon. Cancer of the descending colon and rectum is responsible for approximately two-thirds of all cases, followed by diverticulitis. Functional obstruction, also known as ileus and pseudo-obstruction, is present when dysmotility prevents intestinal contents from being propelled distally and no mechanical blockage exists. Ileus that occurs after intraabdominal surgery is the most commonly known form of functional bowel obstruction, but there are numerous other causes (Table 341-3). Although postoperative ileus is most often transient, it is a common reason why hospital discharge is delayed. Pseudoobstruction of the colon, also known as Ogilvie’s syndrome, is a rela tively rare disease. Some patients with Ogilvie’s syndrome have colonic dysmotility due to abnormalities of their autonomic nervous system that may be inherited. PART 10 Disorders of the Gastrointestinal System ■ ■PATHOPHYSIOLOGY The manifestations of acute intestinal obstruction depend on the nature of the underlying disease process, its location, and changes in blood flow (Fig. 341-1). Increased intestinal contractility, which occurs proximally and distal to the obstruction, is a characteristic response. Subsequently, intestinal peristalsis slows as the intestine or stomach proximal to the point of obstruction dilates and fills with gastrointesti nal secretions and swallowed

air. Although swallowed air is a primary TABLE 341-2 Acute Small-Intestinal and Colonic Obstruction Incidences CAUSE INCIDENCE Postoperative adhesions

> 50% overall Neoplasms ~20% Hernias (especially ventral or internal types, where the risk of strangulation is increased) ~10% Inflammatory bowel disease, other inflammation (obstruction may resolve if acute inflammation and edema subside) ~5% Intussusception, volvulus, other miscellaneous diseases <15%

TABLE 341-3 Most Common Causes of Ileus (Functional or Pseudo-Obstruction of the Intestine) Intraabdominal procedures, lumbar spinal injuries, or surgical procedures on the lumbar spine and pelvis Metabolic or electrolyte abnormalities, especially hypokalemia and hypomagnesemia, but also hyponatremia, uremia, and severe hyperglycemia Drugs such as opiates, antihistamines, and some psychotropic (e.g., haloperidol, tricyclic antidepressants) and anticholinergic agents Intestinal ischemia Intraabdominal or retroperitoneal inflammation or hemorrhage Lower lobe pneumonias Intraoperative radiation (likely due to smooth muscle damage) Systemic sepsis Hyperparathyroidism Pseudo-obstruction (Ogilvie's syndrome) Ileus secondary to hereditary or acquired visceral myopathies and neuropathies that disrupt myocellular neural coordination Some collagen vascular diseases such as lupus erythematosus or scleroderma source of intestinal distension, intraluminal air may also accumulate from fermentation, local carbon dioxide production, and altered gas eous diffusion. Intraluminal dilation also increases intraluminal pressure. When luminal pressure exceeds venous pressure, venous and lymphatic drainage is impeded. Edema ensues, and the bowel wall proximal to the site of blockage may become hypoxemic. Epithelial necrosis can be identified within 12 h of obstruction. Ultimately, arterial blood supply may become so compromised that full-thickness ischemia, necrosis, and perforation result. Stasis increases bacteria counts within the jeju num and ileum. Bacteria, such as Escherichia coli, Streptococcus faecalis, and Klebsiella, and other pathogens may be recovered from intestinal cultures, mesenteric lymph nodes, the bloodstream, and other sites. Other manifestations depend on the degree of hypovolemia, the patient's metabolic response, and the presence or absence of associ ated intestinal ischemia. Inflammatory edema eventually increases the production of reactive oxygen species and activates neutrophils and macrophages, which accumulate within the bowel wall. Their accumu lation, along with changes in innate immunity, disrupts secretory and neuromotor processes. Dehydration is caused by loss of the normal intestinal absorptive capacity as well as fluid accumulation in the gas tric or intestinal wall and intraperitoneally. Anorexia and emesis tend to exacerbate intravascular volume deple tion. In the worst-case scenario after high-grade distal obstruction, emesis leads to losses of gastric potassium, hydrogen, and chloride, while dehydration stimulates proximal renal tubule bicarbonate reab sorption. Intraperitoneal fluid accumulation, especially in patients with severe distal bowel obstruction, may increase intraabdominal pressure enough to elevate the diaphragm, inhibit respiration, impede systemic venous return, and promote vascular instability. Severe hemodynamic compromise may elicit a systemic inflammatory response and general ized microvascular leakage. Closed-loop obstruction results when the proximal and distal open ings of a given bowel segment are both occluded, for example, due to volvulus or a hernia. It is the most common precursor for stran gulation, but not every closed loop strangulates. The risk of vascular insufficiency, systemic inflammation, hemodynamic compromise, and irreversible intestinal ischemia is much greater in patients with

closed-loop obstruction. Pathologic changes may occur rapidly, such that emergent intervention is indicated. Irreversible bowel ischemia may progress to transmural necrosis even if obstruction is relieved. The provider should remember that patients with high-grade distal colonic obstruction who have competent ileocecal valves may pres ent with closed-loop obstruction. In this instance, the cecum may progressively dilate such that ischemic necrosis results in perforation, especially when the cecal diameter exceeds 10–12 cm. Patients with distal colonic obstruction whose ileocecal valves are incompetent tend

Abnormal bacteria colonization Patients with distal obstruction may still discharge intraluminal contents Note: intraluminal obstruction is displayed FIGURE 341-1 Pathophysiologic changes of small-bowel obstruction. to present later in the course of disease and mimic patients with distal small-bowel obstruction. ■ ■HISTORY AND PHYSICAL FINDINGS Even though the presenting signs and symptoms can be misleading, many patients with acute obstruction can be accurately diagnosed after a thorough history and physical examination is performed before imaging. Even though small-bowel obstruction with strangu lation can be especially difficult to diagnosis promptly, early recogni tion allows earlier treatment, which decreases the risk of morbidity and mortality. The cardinal signs are colicky abdominal pain, abdominal disten tion, emesis, and obstipation. More intraluminal fluid accumulates in patients with distal obstruction, which typically leads to greater distention, more discomfort, and delayed emesis. This emesis is fecu lent when there is bacterial overgrowth. Patients with more proximal obstruction commonly present with less abdominal distention but more pronounced vomiting. Elements of the history that might be helpful include any prior history of surgery, including herniorrhaphy, as well as any history of cancer or inflammatory bowel disease. Most patients, even those with simple obstruction, appear to be crit ically ill. Many may be oliguric, hypotensive, and tachycardic because of severe intravascular volume depletion. Fever is worrisome for stran gulation or systemic inflammation. Bowel sounds and bowel functional activity are notoriously difficult to interpret. Classically, many patients with early small-bowel obstruction will have high-pitched, “musical” tinkling bowel sounds and peristaltic “rushes” known as borborygmi. Later in the course of disease, the bowel sounds may be absent or hypo active as peristaltic activity decreases. This contrasts with the common

Inflammatory mediators released Fluid and air accumulate; bacteria overgrowth may occur Air Epithelial necrosis Fluid Proximal bowel dilatation Inflammatory wall edema Point of obstruction from extrinsic, intrinsic, or intraluminal disease CHAPTER 341 Collapsed bowel distal to obstruction Acute Intestinal Obstruction findings in patients with ileus or pseudo-obstruction where bowel sounds are typically absent or hypoactive from the beginning. Lastly, patients with partial blockage may continue to pass flatus and stool, and those with complete blockage may even evacuate bowel contents present downstream beyond their obstruction. All surgical incisions should be examined, and the presence of a tender abdominal or groin mass strongly suggests that an incarcerated hernia may be the cause of obstruction. The presence of tenderness should increase the concern about the presence of complications such as ischemia, necrosis, or localized peritonitis. Severe pain with local ization or signs of peritoneal irritation is suspicious for strangulated or closed-loop obstruction. It is important to remember that the dis comfort may be out of proportion to physical findings mimicking the complaints of patients with acute mesenteric ischemia. Patients with colonic volvulus present with the classic manifestations of closed-loop obstruction: severe abdominal pain, vomiting, and obstipation. Asym metrical abdominal distension and a tympanic mass may be evident. Patients with ileus or pseudo-obstruction may have signs and symptoms like those with

bowel obstruction. Although abdominal distention is present, colicky abdominal pain is typically absent, and patients may not have nausea or emesis. Ongoing, regular discharge of stool or flatus can sometimes help distinguish patients with ileus from those with complete mechanical bowel obstruction. The overall risk of ileus appears to be less in patients who undergo laparoscopic procedures. ■ ■LABORATORY AND IMAGING STUDIES Laboratory testing should include a complete blood count and serum electrolyte and creatinine measurements. Serial assessments are often

useful. Mild hemoconcentration and slight elevation of the white blood cell count commonly occur after simple bowel obstruction. Emesis and dehydration may cause hypokalemia, hypochloremia, elevated blood urea nitrogen–to–creatinine ratios, and metabolic alkalosis. Patients may be hyponatremic on admission because many have attempted to rehydrate themselves with hypotonic fluids. The presence of guaiacpositive stools and iron-deficiency anemia are strongly suggestive of malignancy.

Higher white blood cell counts with the presence of immature forms and the presence of metabolic acidosis are worrisome for severe volume depletion or ischemic necrosis and sepsis. Presently, no labora tory tests are especially useful for identifying the presence of simple or strangulated obstruction, although increases in serum d-lactate, creatine kinase BB isoenzymes, or intestinal fatty acid binding protein levels may be suggestive of the latter. Recommendations for diagnostic imaging continue to evolve. In all cases, the key is not to delay operative intervention unnecessarily when the patient’s signs or symptoms strongly suggest that highgrade or complete obstruction or bowel compromise is present. Abdominal radiography, which must include upright or cross-table lateral views, can be completed quickly and may indicate the need for emergency surgical intervention in patients who are not in the immediate postoperative period. A “staircasing” pattern of dilated air and fluid-filled small-bowel loops >2.5 cm in diameter with little or no air seen in the colon are classical findings in patients with small-bowel obstruction. Little bowel gas appears in patients with proximal bowel obstruction or in patients whose intestinal lumens are filled with fluid. Upright plain films of the abdomen of patients with large-bowel obstruction typically show colon dilatation. Small-bowel air-fluid levels may not be obvious if the ileocecal valve is incompetent. Although it can be difficult to distinguish from ileus, small-bowel obstruction is more likely when air-fluid levels are seen without significant colonic distension. Free air suggests that perforation has occurred in patients who have not recently undergone surgical procedures. A gas-filled, “coffee bean”–shaped dilated shadow may be seen in patients with volvulus. PART 10 Disorders of the Gastrointestinal System More sophisticated imaging can be beneficial when the diagnosis is unclear. Computed tomography (CT) is the most frequently used imaging modality. Its sensitivity for detecting bowel obstruction is ~95% (78–100%) in patients with high-grade obstruction, with a specificity of 96% and an accuracy of ≥95%. Its accuracy in diagnosing closed-loop obstruction is much lower (60%). CT may also provide useful information regarding location or to identify circumstances where surgical intervention is urgently needed. Patients who have evidence of contrast appearing within the cecum within 4–24 h of oral administration of water-soluble contrast can be expected to improve with high sensitivity and specificity (~95% each). Contrast studies may demonstrate a “bird’s beak,” a “c-loop,” or “whorl” deformity on CT imaging at the site where twisting obstructs the lumen when a colonic volvulus is present. Abdominal radiography, unlike CT imaging, may not accurately distinguish obstruction from other causes of colonic dysmotility. Examples of some CT images are provided in Fig. 341-2. Ultrasonographic

evaluations are especially difficult to interpret but may be sensitive and appropriate studies to evaluate patients who are pregnant or for whom x-ray exposure is otherwise contraindicated or inappropriate. CT imaging with enteral and IV contrast can also identify ischemia. Altered bowel wall enhancement is the most specific early finding, but its sensitivity is low. Mesenteric venous gas, pneumoperitoneum, and pneumatosis intestinalis are late findings indicating the presence of bowel necrosis. CT scanning after a water-soluble contrast enema may help distinguish ileus or pseudo-obstruction from distal large-bowel obstruction in patients who present with evidence of small-bowel and colonic distention. CT enteroclysis, though now rarely performed, can accurately identify neoplasia as a cause of bowel obstruction. Contrast enemas or colonoscopies are almost always needed to identify causes of acute colonic obstruction.

A B C FIGURE 341-2 Computed tomography with oral and intravenous contrast demonstrating (A) evidence of small-bowel dilatation with air-fluid levels consistent with a small-bowel obstruction; (B) a partial small-bowel obstruction from an incarcerated ventral hernia (arrow); and (C) decompressed bowel seen distal to the hernia (arrow). (Reproduced with permission from D Longo et al: Harrison’s Principles of Internal Medicine, 18th ed. New York: McGraw-Hill; 2012.) Barium studies are generally contraindicated in patients with firm evidence of complete or high-grade bowel obstruction, especially when they present acutely. Barium should never be given orally to a patient with possible obstruction until that diagnosis has been excluded. In every other instance, such investigations should only be performed in exceptional circumstances and with great caution because patients with significant obstruction may develop barium concretions as an additional source of blockage and some who would have otherwise recovered will require operative intervention. Barium

opacification also renders cross-sectional imaging studies or angiogra phy uninterpretable. TREATMENT Acute Intestinal Obstruction An improved understanding of the pathophysiology of bowel obstruction and the importance of fluid resuscitation, electrolyte repletion, intestinal decompression, and the selected use of antibi otics has likely contributed to a reduction in mortality from acute bowel obstruction. Patients should be stabilized as quickly as possi ble. Nasogastric tube suction decompresses the stomach, minimizes further distention from swallowed air, improves patient comfort, and reduces the risk of aspiration. Urine output should be assessed using a Foley catheter. In some cases, for example, in patients with cardiac disease, central venous pressures should be monitored. The use of antibiotics is controversial, although prophylactic adminis tration may be warranted if operation is anticipated. Complete bowel obstruction is an indication for intervention. Stenting may be possible and warranted for some patients with high-grade obstruction due to unresectable stage IV malignancies. Stenting may also allow elective mechanical bowel preparation before operation. Because treatment options are so variable, it is helpful to make as precise a diagnosis as possible preoperatively. ILEUS Patients with ileus are treated supportively with IV fluids and nasogastric decompression while any underlying pathology is treated, taking care to optimize the use of narcotics. Pharma cologic treatments continue to be evaluated with some studies showing that treatment with peripherally active μ-opioid recep tor antagonists (e.g., alvimopan and methylnaltrexone) or 5-HT4 agonists that stimulate the release of acetylcholine from enteric neurons (e.g., mosapride and prucalopride) may accelerate gas trointestinal recovery in some patients who have undergone abdominal surgery. COLONIC PSEUDO-OBSTRUCTION (OGILVIE’S DISEASE) Neostigmine is an acetylcholinesterase inhibitor that increases cho linergic (parasympathetic) activity, which can stimulate colonic motility. Some studies have

shown it to be moderately effective in alleviating acute colonic pseudo-obstruction. It is the most com mon therapeutic approach and can be used once it is certain that there is no mechanical obstruction. Cardiac monitoring is required, and atropine should be immediately available. Intravenous admin istration induces defecation and flatus within 10 min in the major ity of patients who will respond. Sympathetic blockade by epidural anesthesia can successfully ameliorate pseudo-obstruction in some patients. VOLVULUS Patients with sigmoid volvulus can often be decompressed using a flexible tube inserted through a rigid proctoscope or using a flexible sigmoidoscope. Successful decompression results in sudden release of gas and fluid with evidence of decreased abdominal distension and allows definitive correction to be scheduled electively. Cecal volvulus most often requires laparotomy or laparoscopic correction. INTRAOPERATIVE STRATEGIES Approximately 60–80% of selected patients with mechanical bowel obstruction can be successfully treated conservatively. Most cases of radiation-induced obstruction should be managed nonoperatively if possible. Early consultation with a surgeon is prudent when there is concern about strangulation obstruction or other abnormality that needs to be addressed urgently. Deterioration signifies a need for intervention. At this time, the decision as to whether the patient can continue to be treated nonoperatively can only be based on clinical judgment, although, as described earlier, imaging studies

can sometimes be helpful. The frequency of major complications after operation ranges from 12 to 47%, with greater risk being attributed to resection therapies and the patient’s overall health. Risk is increased for patients with American Society of Anesthesi ologists (ASA) physical status of class III or higher.

At operation, dilation proximal to the site of blockage with distal collapse is a defining feature of bowel obstruction. Intraopera tive strategies depend on the underlying problem and range from lysis of adhesions to resection with or without diverting ostomy to primary resection with anastomosis. Resection is warranted when there is concern about the bowel’s viability after the obstructive pro cess is relieved. Laparoscopic approaches can be useful for patients with early obstruction when extensive adhesions are not expected to be present. Some patients with high-grade obstruction secondary to malignant disease that is not amendable to resection will benefit from bypass procedures. ADULT INTUSSUSCEPTION AND GALLSTONE ILEUS Primary resection is prudent. Careful manual reduction of any involved bowel may limit the amount of intestine that needs to be removed. A proximal ostomy may be required if unprepped colon is involved. The most common site of intestinal obstruction in patients with gallstone “ileus” is the ileum (60% of patients). The gallstone enters the intestinal tract most often via a cholecysto duodenal fistula. It can usually be removed by operative enteroli thotomy. Addressing gallbladder disease during urgent or emergent surgery is not recommended. CHAPTER 341 POSTOPERATIVE BOWEL OBSTRUCTION Early postoperative mechanical bowel obstruction is that which occurs within the first 6 weeks of operation. Although it tends to respond and behave differently from classic mechanical bowel obstruction and may be very difficult to distinguish from post operative ileus, most are partial and can be expected to resolve spontaneously. A higher index of suspicion for a definitive site of obstruction is warranted for patients who undergo laparoscopic surgical procedures. Patients who first had ileus and then subse quently develop obstructive symptoms after an initial return of normal bowel function are more likely to have true postoperative small-bowel obstruction. The longer it takes for a patient’s obstruc tive symptoms to resolve after hospitalization, the more likely the patient is to require surgical intervention. Acute Intestinal Obstruction Acknowledgment The wisdom and expertise of Dr. William Silen are gratefully

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