TABLE 355-2 ACUTE SMALL-INTESTINAL AND COLONIC OBSTRUCTION INCIDENCES

Cause	Incidence
Postoperative adhesions	>50%
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%

adhesions that can cause bowel obstruction (Table 355-2). Overall, small-bowel obstruction is slightly more common in women. The risk of internal herniation is increased by abdominal procedures such as laparoscopic or open Roux-en-Y gastric bypass. Although laparoscopic procedures may generate fewer postoperative adhesions compared with open surgery, the risk of obstructive adhesion formation is not eliminated.

In many patients who are successfully treated for adhesive small-bowel obstruction, obstruction will recur. The rate varies according to how patients were initially managed. 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.

Volvulus, which occurs when bowel twists on its mesenteric axis, can cause partial or complete obstruction and vascular insufficiency. The sigmoid colon is most commonly affected, accounting for 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 conditions 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 in 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 and volvulus.

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 identified form of functional bowel obstruction, although there are many other causes (Table 355-3). Although postoperative ileus is most often transient, it is the most common reason why hospital discharge is delayed. Pseudo-obstruction of the colon, also known as Ogilvie's syndrome, is a relatively rare disease. Some patients with Ogilvie's syndrome have colonic dysmotility due to abnormalities of their autonomic nervous system that may be inherited.

TABLE 355-3 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 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

PATHOPHYSIOLOGY

The manifestations of acute intestinal obstruction depend on the nature of the underlying disease process, its location, and changes in blood flow (Fig. 355-1). 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 gastrointestinal secretions and swallowed air. Although swallowed air is the primary contributor to intestinal distension, intraluminal air may also accumulate from fermentation, local carbon dioxide production, and altered gaseous 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 the bacteria counts within the jejunum and ileum. The most commonly cultured intraluminal organisms are *Escherichia coli*, *Streptococcus faecalis*, and *Klebsiella*, which may also be recovered from mesenteric lymph nodes and other more distant sites.

Other manifestations depend on the degree of hypovolemia, the patient's metabolic response, and the presence or absence of associated intestinal ischemia. Inflammatory edema eventually increases the production of reactive oxygen species and activates neutrophils and macrophages, which accumulate within the bowel wall. Their accumulation, 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 gastric or intestinal wall and intraperitoneally.

Anorexia and emesis tend to exacerbate intravascular volume depletion. In the worst case scenario that is most commonly identified after distal obstruction, emesis leads to losses of gastric potassium, hydrogen, and chloride, while dehydration stimulates proximal renal tubule bicarbonate reabsorption. Intraperitoneal fluid accumulation, especially in patients with severe distal bowel obstruction, may increase intraabdominal pressure enough to elevate the diaphragm and inhibit respiration and to impede systemic venous return and promote vascular instability. Severe hemodynamic compromise may elicit a systemic inflammatory response and generalized microvascular leakage.

Closed-loop obstruction results when the proximal and distal openings of a given bowel segment are both occluded, e.g., due to volvulus or a hernia. It is the most common precursor for strangulation, 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 more rapidly, and emergency intervention is indicated. Irreversible bowel ischemia progresses to transmural necrosis even if the obstruction is relieved. It is also important to remember that patients with high-grade distal colonic obstruction who have competent ileocecal valves may present with closed-loop obstruction. In the latter instance, the cecum may progressively dilate such that ischemic necrosis results in cecal perforation. This risk is generally greatest when the cecal diameter exceeds 12 cm, as informed by Laplace's law. Patients with distal colonic obstruction whose ileocecal valves are incompetent tend 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. Early