Postoperative ileus

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INTRODUCTION

Postoperative ileus, sometimes referred to as adynamic or paralytic ileus, refers to obstipation and intolerance of oral intake due to nonmechanical factors that disrupt the normal coordinated propulsive motor activity of the gastrointestinal tract following abdominal or nonabdominal surgery [1-3].

There is general consensus that some degree of postoperative ileus is a normal obligatory and physiologic response to abdominal surgery that resolves without serious sequelae [4,5]. However, when ileus is pathologic and "prolonged," it increases patient discomfort, dissatisfaction, and morbidity, and could result in prolonged hospitalization and increased healthcare expenditures. Furthermore, it must be differentiated from mechanical bowel obstruction or other postoperative complications.

The epidemiology, clinical features, and diagnosis of postoperative ileus are reviewed here. Measures to prevent prolonged postoperative ileus are reviewed separately. (See "Measures to prevent prolonged postoperative ileus".)

NORMAL GASTROINTESTINAL MOTILITY

Control of gastrointestinal function and motor activity is complex and based upon the interactions of various neural networks and neurohumoral peptides [6]. (See "Overview of

gastrointestinal peptides in health and disease".)

Neurons located in the gut wall comprise the intrinsic neural network, which is called the enteric nervous system (figure 1). The enteric nervous system also has the basic circuitry necessary to run independently through local circuits that rely on signals from intrinsic sensory neurons and neurohumoral peptides, such as substance P, vasoactive intestinal peptide, and nitric oxide, which can act either in a paracrine or endocrine fashion [7-9].

The extrinsic network consists of visceral sensory afferents in the vagus, splanchnic, and pelvic nerves and visceral motor efferents of the autonomic nervous system. These extrinsic neurons synapse with the enteric nervous system and connect it to the central nervous system. In general, vagal afferents are thought to be important in the coordinated integration of motility, secretion, and absorption, while spinal afferents are thought to be responsible for the transmission of noxious stimuli and inflammation. Visceral motor efferent outflow consists of the sympathetic thoracolumbar and parasympathetic craniosacral arms of the autonomic nervous system. Sympathetic activity is generally inhibitory to smooth muscle activity, while parasympathetic input can be excitatory or inhibitory.

ETIOLOGY OF POSTOPERATIVE ILEUS

Postoperative ileus refers to obstipation and intolerance of oral intake due to factors that disrupt the normal coordinated propulsive motor activity of the gastrointestinal tract [1,2,4]. An international consensus panel defined postoperative ileus as the period of time from surgery until the passage of flatus or stool and tolerance of an oral diet [10].

Postoperative gastrointestinal dysmotility is generally related to one or more of the mechanisms (inflammation, neural reflexes, neurohumoral peptides, pharmacologic agents) described below. The inflammatory and the neurohumoral pathways appear to be interrelated as activated inflammatory cells release a variety of substances (eg, cytokines, cyclooxygenase-2 [COX-2], and leukocyte-derived inducible nitric oxide synthase) that further increase inflammatory cell recruitment and inhibit gastrointestinal motility.

Inflammation — Postoperative ileus following abdominal surgery appears to result from an inflammatory response to intestinal manipulation and trauma.

Animal studies shows that the degree of intestinal manipulation of both the small and large intestine is directly related to both the degree of leukocyte infiltration into the intestinal muscularis and the amount of intestinal dysmotility [11,12]. The prospective SANICS-II trial

shows that inflammatory parameters in blood samples are increased in postoperative ileus patients undergoing colorectal surgery [13].

Moreover, this effect is not necessarily limited to the manipulated segment, as there appears to be an inflammatory field effect affecting the entire gastrointestinal tract [14-16]. Nonabdominal procedures have also been shown to lead to intestinal inflammation. The mechanism for this remains poorly understood, but it is hypothesized to originate by reduced intestinal blood perfusion (ischemia) and translocation of endogenous cellular danger molecules and cytokines from the distant site of surgical trauma.

Intestinal manipulation activates a network of local macrophages triggering an inflammatory response that results in muscle dysfunction. Depletion and inactivation of these resident macrophages [17], as well as treatment with semapimod, a powerful anti-cytokine molecule, has prevented postoperative ileus in rodents [18,19]. Intestinal manipulation also appears to increase COX-2 expression and to elevate prostaglandin levels, which, in turn, decreases jejunal contractility. This effect is blocked in COX-2-deficient mice and by the administration of selective COX-2 inhibitors [20].

As COX-2 inhibitors are also in widespread clinical use, they have been tested in clinical trials wherein Celecoxib showed a marked reduction in the incidence of postoperative ileus [21,22]. A meta-analysis of randomized controlled trials concluded that COX-2 inhibitors improve postoperative motility after elective colorectal surgery and showed these drugs to be safe in terms of anastomotic healing [23]. However, COX-2 inhibitors are still not recommended for treatment of postoperative ileus, as patients treated with nonsteroidal anti-inflammatory drugs, known to nonselectively inhibit COX-1 and COX-2 function, are at risk to induce postoperative healing of colorectal anastomosis [24]. (See "Measures to prevent prolonged postoperative ileus", section on 'COX-2 inhibition'.)

In addition, studies suggest that cells of the enteric nervous system also contribute to the inflammation by releasing proinflammatory cytokines and chemokines [25,26]. (See "Measures to prevent prolonged postoperative ileus", section on 'COX-2 inhibition'.)

Mast cells were once thought to play a role in the development of postoperative ileus since mast cell stabilizers prevent surgically induced intestinal inflammation and dysmotility, and mast cell deficiencies limit intestinal leukocyte migration [27]. However, a study using a more specific mast-cell-deficient mouse model demonstrated that mast cells do not appear to be involved [28].

Inhibitory neural reflexes — A series of studies from the very late 20th century showed that inhibitory neural reflexes are thought to act locally through noxious spinal afferent signals that

increase inhibitory sympathetic activity in the gastrointestinal tract [7,29]. The clinical importance of this mechanism is that blockade of spinal afferents with epidural local anesthetics was shown to improve postoperative ileus [30,31]. (See "Measures to prevent prolonged postoperative ileus", section on 'Epidural or TAP block'.)

Neurohumoral peptides — Nitric oxide, vasoactive intestinal polypeptide, and possibly substance P are thought to act as inhibitory neurotransmitters in the gut that slow gut motility. In rats, antagonists to these peptides improve surgically induced gastrointestinal dysmotility [32,33]. The contribution of substance P is only partially understood; its inhibitory effects may be related to its stimulation of inhibitory sensory signals rather than stimulatory motor activity directly in the gut. Decreases in motilin (a motor-stimulatory hormone) and increases in the inhibitory factors calcitonin gene-related peptide and corticotropin-releasing factor (CRF) have also been implicated in the pathophysiology of postoperative ileus [29,34-37].

Pharmacologic agents — Opioids have well-known inhibitory effects on the gastrointestinal tract. They increase resting tone while decreasing gastric motility and emptying, increase small intestinal periodic spasms, and decrease propulsive colonic movements [4,38-40]. These effects seem to be receptor specific and appear to be mediated primarily at the level of the enteric nervous system. (See 'Management' below and "Measures to prevent prolonged postoperative ileus", section on 'Multimodal analgesia'.)

PROLONGED POSTOPERATIVE ILEUS

A strict diagnosis of prolonged postoperative ileus is challenged by varying definitions and a lack of clear valid endpoints (eg, time to flatus, time to first oral intake). An international consensus panel defined "prolonged" postoperative ileus as the occurrence of two or more of the following signs and symptoms on postoperative day 4 or after [10]:

- Nausea or vomiting
- Inability to tolerate an oral diet over the preceding 24 hours
- Absence of flatus over the preceding 24 hours
- Abdominal distention
- Radiologic confirmation

It is well recognized, however, that some of these signs and symptoms may not be objective (eg, assessment of distention, radiographic interpretation) or may be confounded by causes other than postoperative ileus (eg, nausea due to medications). (See 'Symptoms' below.)

A wide variety of endpoints, including the ones listed above, have been used to measure gut recovery with no consensus as to which one is most clinically meaningful [41-43]. Furthermore, what constitutes an acceptable length of gastrointestinal recovery time also depends on the type of surgery and other risk factors [44]. However, as primary outcomes in clinical trials on postoperative ileus, time to solid food tolerance and having had defecation were suggested as best parameters reflecting gastrointestinal transit [45].

EPIDEMIOLOGY AND RISK FACTORS

Estimates of the incidence of prolonged postoperative ileus vary widely in the literature, depending upon the type of surgery. A common limitation of many of these studies is that they differ in the definition of and criteria used to diagnose prolonged postoperative ileus. (See 'Prolonged postoperative ileus' above.)

- In an observational study involving over 17,000 patients undergoing colectomy, postoperative ileus occurred in 17.4 percent [46].
- In a study of 27,560 patients undergoing elective colon resection during 2012 to 2013, prolonged postoperative ileus occurred in 12.7 percent, and the rate was highest in patients with ileocolonic anastomosis (15 percent) [47]. Anastomotic leak and intra-abdominal infection were associated with an increased risk of postoperative ileus, whereas oral antibiotic bowel preparation and laparoscopic surgery were associated with reduced risk.
- In a study of the placebo arms of trials evaluating prevention, postoperative ileus occurred in 15 percent undergoing bowel resection but only 3 percent of patients undergoing abdominal hysterectomy [44].
- In a systematic review of radical cystectomy, the incidence of postoperative paralytic ileus across all studies was 9.9 percent [43].
- In a systematic review of 21 studies on open repair of the abdominal aorta, the risk of paralytic ileus was significantly lower for the retroperitoneal approach compared with the transabdominal approach (1.4 versus 10.6 percent) [48].

Risk factors — The risk for prolonged postoperative ileus may be increased by any factor or disease state that adversely affects gastrointestinal motility. These include both surgical and nonsurgical etiologies (table 1).

The most important risk factors include [49-75]:

- Prolonged abdominal or pelvic surgery.
- Lower gastrointestinal surgery.
- Open surgery – Open surgery is associated with greater intestinal manipulation and an increased risk for postoperative ileus.
- Delayed enteral nutrition/nasogastric tube placement – Routine placement of nasogastric tubes is associated with a significantly slower return of bowel function and trends toward increased complications and longer length of stay. Similarly, delayed enteral nutrition is associated with slower return of bowel function compared with early enteral nutrition in systematic reviews. (See "Inpatient placement and management of nasogastric and nasoenteric tubes in adults" and "Overview of perioperative nutrition support".)
- Intra-abdominal inflammation (eg, peritonitis, sepsis).
- Perioperative complications (eg, postoperative pneumonia, intra-abdominal abscess).
- Intraoperative and postoperative bleeding, need for transfusion – It is unknown whether there are any significant differences in the risk for postoperative ileus for intra-abdominal compared with retroperitoneal bleeding.
- Factors that increase bowel wall edema, such as hypoalbuminemia and aggressive administration of intravenous fluids, also appear to increase the risk of postoperative ileus [76].
- Perioperative opioid use can contribute to postoperative ileus; other pharmacologic agents or metabolic derangement are implicated in the development of gastrointestinal dysmotility and are not limited to the postoperative period.

Unproven or debated risk factors include:

- Obesity - Some studies provide evidence that obesity correlates to an increased risk of postoperative ileus [53,77-79], but others do not [80].
- Exposure to general anesthesia is commonly believed to play a role in depressing postoperative intestinal motility [38,81,82], but this is not well proven, and its role, if any, is likely minor and transient [50].

Additional risk factors that are specific to a type of surgery may include:

- For colorectal surgery – Male sex, peripheral vascular disease, resection at urgent operation, construction of a stoma, disseminated cancer, chronic obstructive pulmonary disease [47,54,80].

- For urologic surgery – Urine in the operating field may contribute to the development of ileus [83]. Studies are conflicting regarding whether the risk of postoperative ileus increases with age [53,84].

- For gynecologic surgery – For benign disease procedures that included bowel surgery, cystotomy and lysis of adhesions were risk factors for postoperative ileus, along with blood transfusion [85]. For ovarian cancer resection, risk of postoperative ileus was significantly higher with bowel resection compared with operations without bowel resection [86].

- For laparoscopic surgery – The development of ileus after laparoscopic surgery should increase suspicion for occult injury (eg, bowel, bladder, ureteral). (See "Complications of laparoscopic surgery", section on 'Bowel injuries' and "Complications of laparoscopic surgery", section on 'Dissection-related bowel injuries'.)

The patient's medical history should be reviewed for factors that increase the risk for prolonged postoperative ileus, including comorbid medical illnesses (eg, long-standing diabetes can cause gastroparesis) or medications that can contribute to ileus (table 1), and for conditions that increase the risk for bowel obstruction. (See "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Etiologies'.)

Identifying risk factors for postoperative ileus is important for counseling the patient and targeting prevention strategies. Available scoring systems for predicting who will or will not develop postoperative ileus have been studied only for narrow patient populations [80,87]. The development of a useful system would require validation across a broad range of surgeries. (See "Measures to prevent prolonged postoperative ileus".)

Enhanced recovery after surgery (ERAS) protocols have changed the culture of postoperative care. Implementing one of the ERAS protocols available in gynecologic, colorectal, hepatobiliary, and upper gastrointestinal surgery may reduce the incidence of postoperative ileus, though this is not always the case [88]. (See "Enhanced recovery after gynecologic surgery: Components and implementation" and "Enhanced recovery after colorectal surgery".)

CLINICAL FEATURES

Symptoms — Symptoms of postoperative ileus may include:

- Abdominal distention, bloating, and "gassiness"
- Diffuse, persistent abdominal pain
- Nausea and/or vomiting
- Delayed passage of or inability to pass flatus
- Inability to tolerate an oral diet

Physical findings — Physical examination typically reveals abdominal distention and tympany, a variable reduction of bowel sounds, and often some degree of mild diffuse tenderness. Features distinguishing postoperative ileus from bowel obstruction are given in the table (table 2) and discussed below. (See 'Mechanical bowel obstruction' below.)

DIAGNOSIS

Although there are no strict criteria, prolonged postoperative ileus is a clinical diagnosis that is often made when the symptoms or signs discussed in the previous section persist for more than three to five days (depending on the nature of the surgery and what is considered "typical"), while mechanical bowel obstruction or other entities that can lead to postoperative abdominal distention and decreased bowel activity have been excluded by imaging. (See 'Imaging' below.)

Diagnostic evaluation — Although postoperative ileus is primarily a clinical diagnosis, imaging and laboratory evaluation are important to exclude mechanical bowel obstruction or bowel perforation and to identify reversible causes of prolonged postoperative ileus.

Imaging — We typically obtain plain abdominal films first, followed by abdominal computed tomography and then upper gastrointestinal contrast study, if necessary, to confirm diagnosis.

Plain abdominal films — Plain abdominal films are often the first type of diagnostic imaging obtained for the evaluation of abdominal distention, nausea, or pain. Supine and upright plain abdominal radiographs may show dilated loops of small bowel and colon in patients with postoperative ileus (image 1A-C) but should demonstrate air in the colon and rectum without a transition zone to suggest bowel obstruction and no evidence of free air to suggest perforation (unless recently in the postoperative period, where air may be noted in the peritoneal cavity for up to seven days) [89,90].

Computed tomography — When the combination of history, findings on physical examination, and plain radiography cannot distinguish ileus from small bowel obstruction, we

perform computed tomography (CT) of the abdomen. Abdominal CT with oral contrast has a sensitivity and specificity of 90 to 100 percent in distinguishing ileus from complete small bowel obstruction, although it is less reliable in distinguishing ileus from partial small bowel obstruction [90-93]. Moreover, abdominal CT can often identify secondary causes of ileus, such as a pelvic abscess or postoperative bleeding, and in cases of small bowel obstruction, it may identify a site of obstruction (image 2) or suggest the presence of strangulated, ischemic, or necrotic bowel [92,94]. (See "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Diagnostic evaluation'.)

The radiographic features of small bowel obstruction are discussed elsewhere and may overlap with those of ileus, especially in the case of partial small bowel obstruction (table 2). (See 'Differential diagnosis' below and "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Diagnosis'.)

Upper gastrointestinal contrast study — If the diagnosis remains uncertain after abdominal CT and the patient's condition persists, upper gastrointestinal contrast studies (enteroclysis) with water-soluble radio-opaque contrast material (eg, Gastrografin) should be considered. Enteroclysis is particularly useful for distinguishing ileus from partial small bowel obstruction, which more closely mimics ileus than complete small bowel obstruction, and for identifying the severity of partial obstruction. (See "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults", section on 'Alternative studies for special patient groups'.)

Laboratory — The laboratory evaluation should seek to identify reversible factors that may contribute to postoperative ileus or that might suggest another postoperative complication (table 3).

We obtain the following laboratories in patients suspected to have prolonged postoperative ileus:

- Complete blood count – Anemia may be due to postoperative bleeding. Elevated white cell count may signal intra-abdominal infection, intestinal ischemia, or intra-abdominal abscess.
- Electrolyte panel (including magnesium) – Hypokalemia worsens ileus; magnesium depletion can lead to hypokalemia.
- Creatinine and blood urea nitrogen – Uremia can lead to ileus.

- Liver function tests, amylase, lipase – Postoperative gallbladder dysfunction or pancreatitis can lead to ileus.

DIFFERENTIAL DIAGNOSIS

The evaluation of the patient with suspected prolonged postoperative ileus should exclude other surgical entities that can lead to postoperative abdominal distention and decreased bowel activity, such as small bowel obstruction and bowel perforation.

Physiologic ileus — Some degree of postoperative ileus is a normal physiologic response to abdominal surgery and typically follows a benign and self-limited course. Following abdominal surgery, "normal" physiologic postoperative ileus due to postoperative gut dysmotility was widely reported as lasting 0 to 24 hours in the small intestine, 24 to 48 hours in the stomach, and 48 to 72 hours in the colon [2,38,95-97]. However, this belief has been challenged; the duration of postoperative gastrointestinal dysmotility appears to be shorter than previously thought. Generally, gastric and small intestinal activity appears to return within hours of surgery, and colonic activity returns by postoperative day 2 or 3 [5,49,98-103]. When ileus extends beyond that, the patient is diagnosed as having a prolonged postoperative ileus, provided there are no signs of mechanical intestinal obstruction. (See 'Diagnosis' above.)

Mechanical bowel obstruction — Distinguishing prolonged postoperative ileus from mechanical small bowel obstruction is of critical importance and can be challenging as they share many of the same signs and symptoms (table 2).

It is useful to note that commonly patients with early postoperative bowel obstruction have an initial return of bowel function and oral intake, which is then followed by nausea, vomiting, abdominal pain, and distention, whereas patients with ileus generally do not experience any return of bowel function. Small bowel obstruction may be more likely in the presence of potential areas of bowel herniation (eg, other prior incisions, known inguinal or femoral hernia, stomas, adhesions, other surgeries associated with internal hernias), masses (intraluminal or extraluminal), large tumor burden, or peritoneal metastases. Intense cramping pain, feculent emesis, or rapidly progressing pain or distention are more suggestive of bowel obstruction than prolonged postoperative ileus. Localized tenderness, fever, tachycardia, and peritoneal signs suggest bowel ischemia or perforation, which indicate the need for emergency surgical intervention.

Although postoperative ileus and noncomplicated bowel obstruction can each be initially managed conservatively, prolonged or worsening small bowel obstruction ultimately requires

surgery to prevent complications (eg, ischemia, necrosis, perforation). (See "Etiologies, clinical manifestations, and diagnosis of mechanical small bowel obstruction in adults" and "Management of small bowel obstruction in adults".)

Colonic pseudo-obstruction — Acute colonic pseudo-obstruction may be due to a variety of medical and surgical conditions and should also be considered in the differential diagnosis of postoperative ileus. (See "Acute colonic pseudo-obstruction (Ogilvie's syndrome)".)

MANAGEMENT

There are few data from well-designed trials to guide therapy of patients with prolonged postoperative ileus. All potentially reversible causes that can prolong postoperative ileus, both surgical and medical, should be corrected. Otherwise, supportive care includes removal of any inciting factors, maintenance and replacement fluid therapy, bowel rest and bowel decompression (as needed), and serial abdominal examination.

Because strategies aimed at curtailing prolonged postoperative ileus may not be as effective once it is established, they are best instituted before or shortly after surgery, before the postoperative ileus becomes prolonged. Such preventive measures, which are the essence of the modern enhanced recovery after surgery (ERAS) programs, are discussed in detail elsewhere. (See "Measures to prevent prolonged postoperative ileus".)

Correcting reversible causes — Secondary causes of ileus and nonobstructive bowel dysfunction, such as intra-abdominal leak, abscess, or retroperitoneal bleeding, should be investigated. Some of these may require surgical management or other interventions (table 3). (See 'Computed tomography' above.)

Electrolytes, including potassium and magnesium, should be checked daily and replaced as needed. (See 'Laboratory' above and "Clinical manifestations and treatment of hypokalemia in adults" and "Hypomagnesemia: Clinical manifestations of magnesium depletion".)

Supportive care — Supportive care is the mainstay of treatment for those with prolonged postoperative ileus.

- Pain management – Opioids should be used sparingly and supplemented with other agents such as nonsteroidal anti-inflammatory drugs or acetaminophen. (See "Measures to prevent prolonged postoperative ileus", section on 'Multimodal analgesia' and "Approach to the management of acute pain in adults".)

- Fluid therapy – Intravenous fluids should be administered to maintain normovolemia. Replacement intravenous fluids should be administered when there is a large volume of emesis or drainage from a nasogastric tube (one or more liters per day). (See "Maintenance and replacement fluid therapy in adults" and "Maintenance intravenous fluid therapy in children".)

- Bowel rest – The patient may be allowed sips of clear fluids, if tolerated. Once abdominal distention resolves and bowel sounds return, the patient can be started on a liquid diet. When the patient has taken adequate fluids, the diet can be advanced and intravenous fluid discontinued.

- Bowel decompression – For patients with moderate-to-severe or continuous vomiting, or significant abdominal distention, a nasogastric tube can be placed. (See "Inpatient placement and management of nasogastric and nasoenteric tubes in adults".)

- Nutritional support – Patients with postoperative ileus who are unable to tolerate enteral nutritional support will require total parenteral nutrition until they can be transitioned to oral feedings. (See "Overview of perioperative nutrition support", section on 'Postoperative nutrition support'.)

- Postoperative mobilization – A retrospective study on more than 8600 individuals found a significant association between postoperative mobilization and postoperative ileus as part of a composite outcome in patients undergoing thoracic, colorectal, or orthopedic surgery, but not cardiac or other surgeries [104]. Another study confirmed the beneficial effects of an early postoperative mobilization in patients undergoing abdominal surgery [105]. Therefore, early postoperative mobilization of surgical patients is recommended.

Serial abdominal examination — The abdominal examination should be performed several times per day to evaluate the amount of distention and patient discomfort. Repeated or more detailed imaging studies may be needed if ileus cannot be differentiated from bowel obstruction or if conservative measures do not improve the patient's condition in 48 to 72 hours. (See 'Imaging' above.)

Gastrografin is a water-soluble, hyperosmolar oral contrast agent commonly used for computed tomography (CT) imaging. Although Gastrografin has been used therapeutically to treat adhesive small bowel obstruction [106], it does not accelerate recovery of prolonged postoperative ileus after elective colorectal surgery. Based upon available evidence, we do not recommend using Gastrografin in the treatment of prolonged postoperative ileus beyond diagnostic purposes. (See "Gastrografin for adhesive small bowel obstruction", section on 'Exclusions'.)

Gastrografin has been studied in two small randomized trials of patients who had prolonged ileus after surgery. In one trial, orally administered Gastrografin accelerated time to flatus or stool (18.9 versus 32.7 hours) and time to resolution of abdominal distension (52.8 versus 77.7 hours) but did not affect time to resolution of nausea and vomiting (64.5 versus 74.3 hours) or time to tolerance of oral diet (75.8 versus 90.0 hours) [107]. The other trial reported no significant differences in mean time to resolution of postoperative ileus between the Gastrografin and control groups (9.1 versus 10.3 days) [108].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Bowel obstruction".)

SUMMARY AND RECOMMENDATIONS

- **Postoperative ileus** – Postoperative ileus refers to obstipation and intolerance of oral intake following abdominal or nonabdominal surgery. It is due to nonmechanical factors, primarily inflammation of the intestinal smooth muscle, leading to disruption of the normal coordinated propulsive motor activity of the gastrointestinal tract. (See 'Introduction' above and 'Normal gastrointestinal motility' above and 'Etiology of postoperative ileus' above.)

- **Prolonged postoperative ileus** – Some degree of postoperative ileus is a normal and physiologic response to abdominal surgery. However, when ileus is prolonged (more than three to five days), it leads to patient discomfort, dissatisfaction, morbidity, and prolonged hospitalization, and it must be differentiated from mechanical bowel obstruction or other postoperative complications. (See 'Prolonged postoperative ileus' above.)

- **Epidemiology and risk factors** – The incidence of prolonged postoperative ileus varies widely in the surgical literature and depends upon the definition of postoperative ileus that is used and the type of surgery. The most important risk factors for prolonged postoperative ileus include prolonged abdominal or pelvic surgery, lower gastrointestinal surgery, open surgery, delayed enteral nutrition/nasogastric tube placement, intra-abdominal inflammation, postoperative complications, and possibly increased body mass index (table 1). (See 'Epidemiology and risk factors' above.)

- **Diagnosis** – Prolonged postoperative ileus is a clinical diagnosis that is made when one or more of the following symptoms or signs persist for more than three to five days

(depending on the nature of the surgery and what is considered "typical"). (See 'Diagnosis' above.)

- Abdominal distention, bloating, and "gassiness"
- Diffuse, persistent abdominal pain
- Nausea and/or vomiting
- Delayed passage of or inability to pass flatus
- Inability to tolerate an oral diet

- **Imaging** – Imaging and laboratory evaluation are important to exclude other causes of ileus and surgical entities that can lead to postoperative abdominal distention and decreased bowel activity that may require surgical management or other intervention, such as small bowel obstruction, bowel perforation, intra-abdominal abscess, or retroperitoneal bleeding (table 2). (See 'Diagnostic evaluation' above and 'Differential diagnosis' above.)

  Plain radiographs demonstrating air in the colon and rectum, with no transition zone or free air, support a diagnosis of postoperative ileus and may be adequate for distinguishing postoperative ileus from small bowel obstruction. However, if there remains any suspicion for small bowel obstruction or another diagnosis, we perform computed tomography (CT) of the abdomen, and if CT is still nondiagnostic, we perform an upper gastrointestinal tract contrast study, such as enteroclysis. (See 'Imaging' above.)

- **Management** – All potentially reversible causes that can prolong postoperative ileus, both surgical and medical, should be corrected (table 3). Otherwise, supportive care includes pain control that minimizes opioid use, judicious intravenous fluid, electrolyte therapy, and dietary restriction, and selective placement of a nasogastric tube for gastrointestinal decompression for those with persistent nausea/vomiting. The patient should be closely monitored with serial abdominal examinations for improvement or worsening of their condition. Additional imaging studies are warranted if conservative measures do not improve the patient's condition in 48 to 72 hours. (See 'Management' above.)

- **Prevention** – Because strategies aimed at curtailing prolonged postoperative ileus may not be as effective once it is established, they are best instituted before or shortly after surgery, before the postoperative ileus becomes prolonged. Such preventive measures, which are the essence of the modern enhanced recovery after surgery (ERAS) programs, are discussed in detail elsewhere. (See "Measures to prevent prolonged postoperative ileus".)

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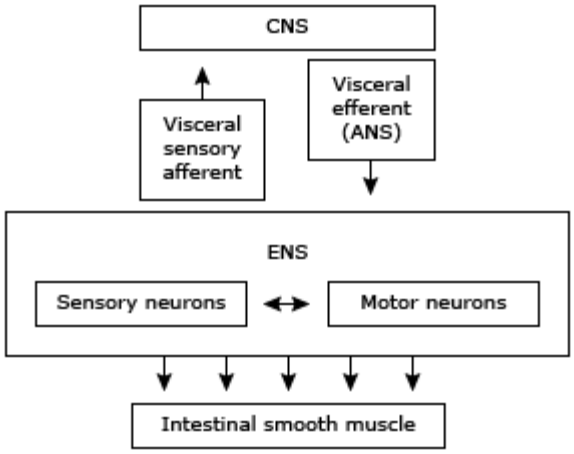
Topic 8042 Version 34.0

GRAPHICS

Schematic of gastrointestinal greater nervous system



CNS: central nervous system; ANS: autonomic nervous system; ENS: enteric nervous system.

Graphic 50829 Version 3.0

Nonsurgical causes of ileus

Pharmacologic agents
Opioids
Antihypertensive agents: Non-dihydropyridine calcium channel blockers (verapamil>diltiazem), clonidine
Antineoplastic agents: Bortezomib, busulfan, pegylated liposomal doxorubicin, methotrexate, paclitaxel, thalidomide, vinblastine, vincristine
Gastrointestinal agents:
▪ Antidiarrheal/antispasmodic: Alosetron, loperamide, diphenoxylate-atropine, hyoscyamine
▪ Phenothiazine antiemetics: Prochlorperazine, promethazine
Other: Oral iron preparations, zoledronic acid
Drugs with significant anticholinergic properties, including:
▪ Selective serotonin reuptake inhibitor antidepressants (paroxetine>fluoxetine)
▪ Tricyclic antidepressants (eg, amitriptyline, imipramine, desipramine, nortriptyline)
▪ Antipsychotics (eg, clozapine, haloperidol, olanzapine, quetiapine)
▪ Parkinson disease medications (eg, benztropine, carbidopa-levodopa, entacapone)
▪ H_1 antihistamines, first generation (eg, diphenhydramine, chlorpheniramine, cyproheptadine, meclizine)
▪ Muscle relaxants (eg, baclofen, cyclobenzaprine, tizanidine)
▪ Overactive bladder (OAB) medications (eg, oxybutynin, solifenacin, tolterodine)
▪ Atropine products
A method for estimating additive anticholinergic effects of various drugs is provided in a separate table. This is not a complete list of all medications that can contribute to nonsurgical ileus.
Medical conditions
Pancreatitis
Gastroenteritis
Spinal cord injury
Myocardial infarction
Stroke
Pneumonia

Hypokalemia
Diabetes (neuronal loss with progressive disease)
Diabetic ketoacidosis
Acute intermittent porphyria
Botulism
Severe burns
Parkinson disease
Epilepsy

Graphic 96322 Version 2.0

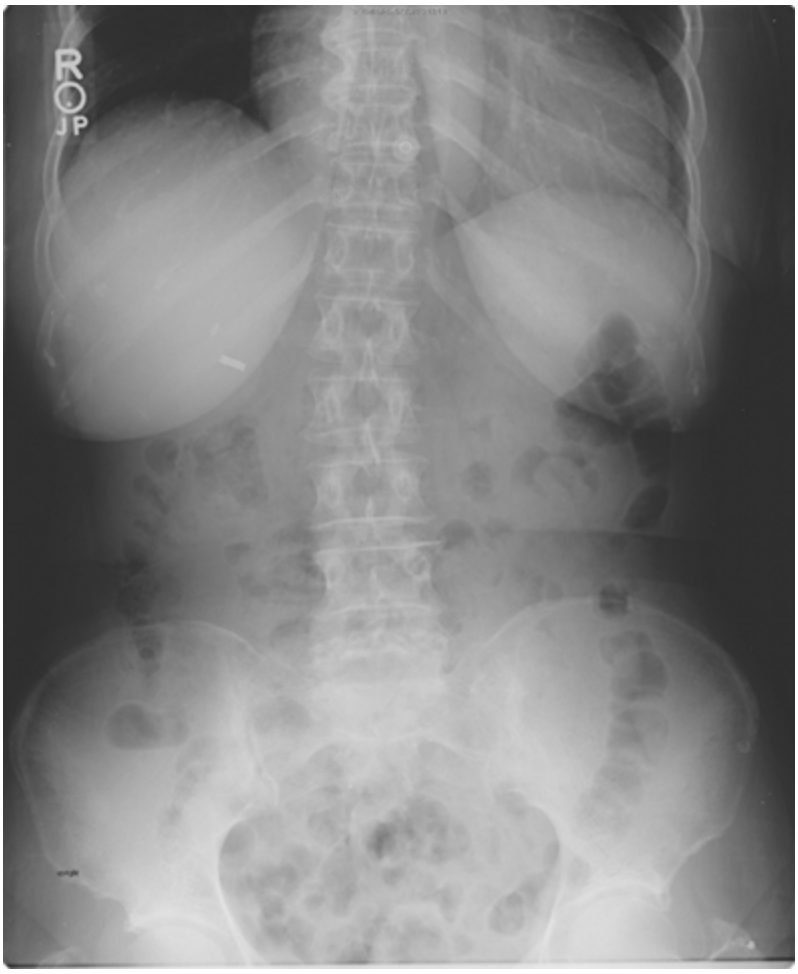
Comparison of ileus and small bowel obstruction

Sign or symptom	Ileus	SBO
Abdominal distention	May be present	May be present
Bowel sounds	Usually quiet or absent	May be high pitched, may be absent
Obstipation	May be present	May be present
Pain	Mild and diffuse	Moderate to severe, colicky
Peritoneal signs	Absent	May be present
Radiography	Dilated loops of bowel, paucity of colonic gas	Dilated loops of bowel, differential air-fluid levels, paucity or absence of colonic gas
Fever, tachycardia	Absent	Should raise suspicion
Vomiting	May be present	May be present, may be bilious or feculent

SBO: small bowel obstruction.

Graphic 52448 Version 4.0

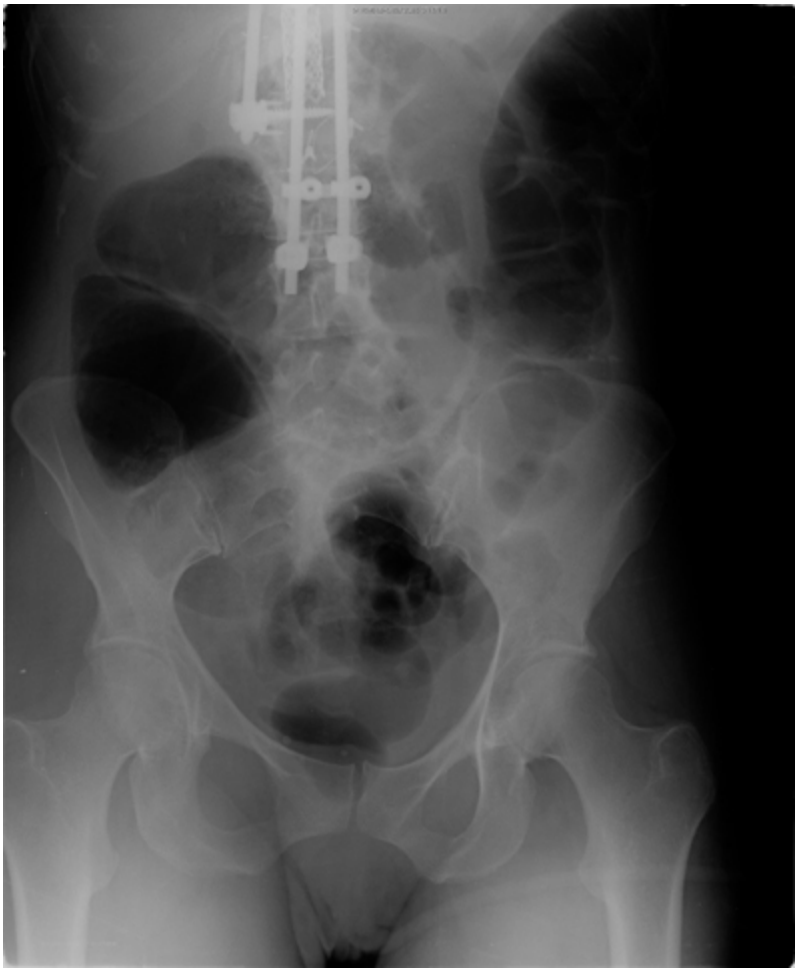
Normal bowel gas pattern on plain abdominal radiograph



Plain abdominal radiograph shows a normal pattern with gas throughout nondilated loops of small and large bowel.

Graphic 81896 Version 4.0

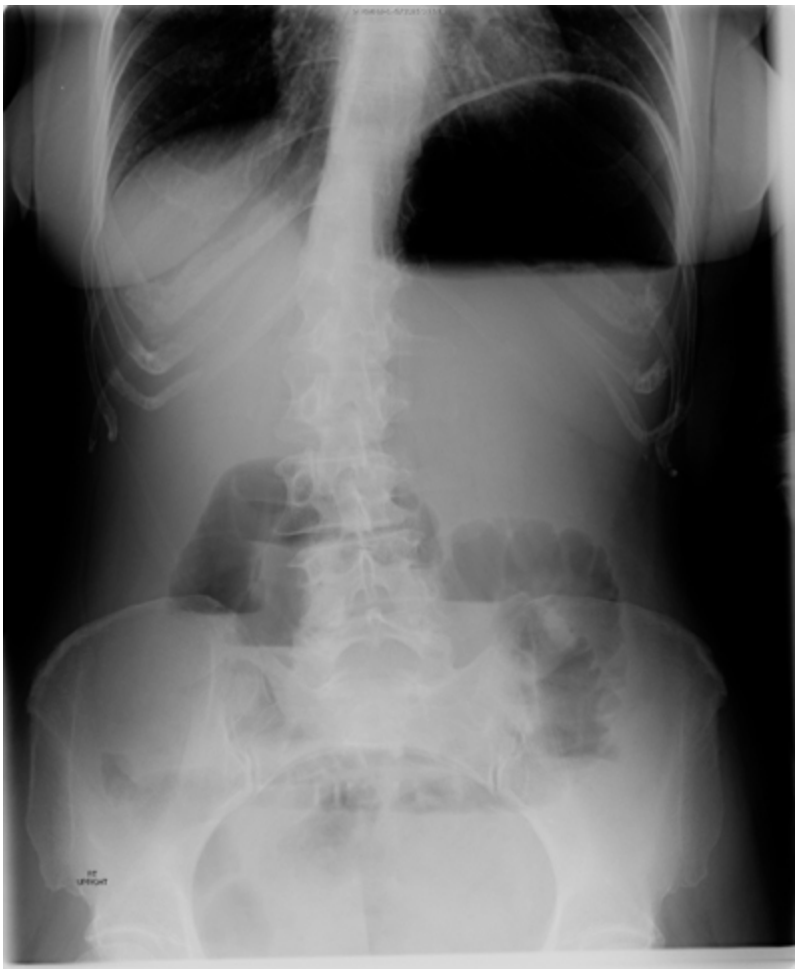
Postoperative ileus on plain abdominal radiograph



Plain abdominal radiograph shows a pattern suggestive of ileus with dilated colon on the right in a patient with spinal fixation devices.

Graphic 56266 Version 4.0

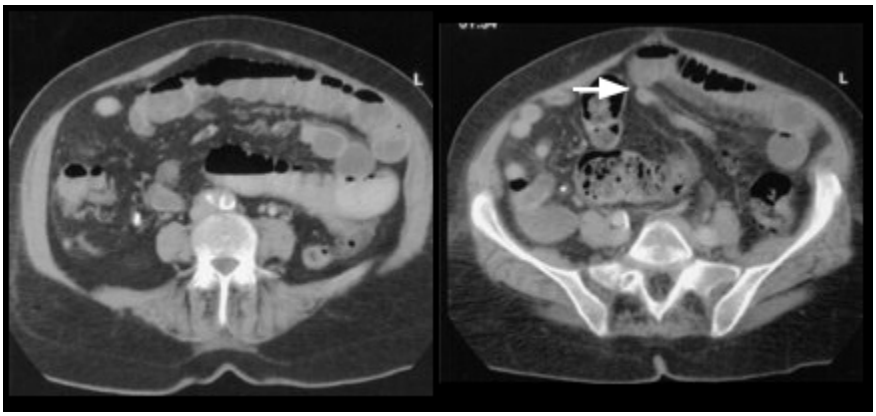
Small bowel obstruction on plain abdominal radiograph



Plain upright abdominal film shows a distended stomach and slightly dilated loops of small bowel with air fluid levels and a paucity of colonic gas, consistent with small bowel obstruction.

Graphic 68029 Version 4.0

Adhesive small bowel obstruction on computed tomography



Computed tomography (CT) of the abdomen demonstrates small bowel obstruction due to adhesive disease. The transition point is shown (arrow).

Graphic 50457 Version 4.0

Potentially treatable causes of ileus

Abdominopelvic abscess
Anastomotic leaks
Anticholinergic drugs
Antihistamines
Appendicitis
Cholecystitis
Hemoperitoneum or retroperitoneal hemorrhage
Hypokalemia
Hypomagnesemia
Opiates
Pancreatitis
Sepsis
Uremia

Graphic 70101 Version 1.0

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