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Prevention of Gastrointestinal Complications in the Critically Ill Patient

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ABSTRACT

Common pathophysiologic changes associated with critical illness directly contribute to the development of gastrointestinal (GI) complications. In addition, supportive interventions such as mechanical ventilation and vasopressors increase the risk of GI complications. Early, specific signs of GI complications are rarely present; therefore, because of late or missed diagnosis, morbidity and mortality related to these complications can be high. This article aims to review the pathophysiology of GI dysfunction and describe an approach to evaluate the abdomen in the critically ill patient. Risk can be limited by understanding individual patient characteristics, thoughtfully evaluating the risk-benefit profile of all interventions, and implementing preventive strategies.
Keywords: complications, critical illness, gastrointestinal

Gastrointestinal (GI) complications in the critically ill patient are common and carry a high risk for mortality. The potential etiologies are numerous and are related not only to patient characteristics such as age and underlying disease processes but also to interventions such as mechanical ventilation. Most critically ill patients are unable to provide a history or description of symptoms, which limits and/or delays timely recognition of GI dysfunction. It is therefore essential that critical care practitioners suspect GI complications, initiate preventive strategies, and diligently monitor the patient for evidence of complications. This article aims to review the pathophysiology of GI dysfunction and describe an approach to evaluate the abdomen in the critically ill patient. The risk of GI complications can be limited by understanding individual patient characteristics, thoughtfully evaluating the risk-benefit profile of all interventions, and implementing preventive strategies.

Prevalence of Gastrointestinal Complications

The overall prevalence of GI complications in the critically ill is difficult, if not impossible, to determine. Many authors have, however, reported the prevalence of and predictive factors for GI complications in specific patient populations. These data allow practitioners to evaluate risk in individual patients.

Chan et al[1] described GI complications in neurosurgical patients. Their retrospective report of 526 patients revealed 36 (6.8%) endoscopically or surgically identified GI complications. All patients had GI bleeding, and two also had perforation. Moreover, 11 patients died as a direct result of the GI complication. Preoperative coma was the only factor that predicted life-threatening complications.

In a prospective study of 11 508 cardiac surgery patients, D'Ancona et al[2] identified 147 GI complications in 129 patients (1.2%). Complications included gastroesophagitis (12.2%), upper GI hemorrhage (28.6%), perforated peptic ulcer (4.7%), cholecystitis (6.8%), pancreatitis (8.8%), intestinal ischemia (11.5%), colitis (12.2%), diverticulitis

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(3.4%), intestinal occlusion (1.1%), and lower GI hemorrhage (0.7%). Mortality was highest with intestinal ischemia (11/17, 64.7%). Patients with complications were older and had higher comorbidity (unstable angina, chronic renal failure, and peripheral vascular disease), morbidity (mechanical ventilation for >24 hours, intraaortic balloon pumping, bleeding, acute renal failure, stroke, and infection), and mortality rates (22.5% vs 4%). Six factors independently predicted GI complications: mechanical ventilation for more than 24 hours, postoperative renal failure, sepsis, valve surgery, preoperative chronic renal failure, and deep sternal wound infection.

Mesenteric ischemia is a catastrophic complication. In an analysis of 8709 cardiac surgery patients, Mangi et al[3] reported that 46 (0.53%) had GI complications requiring surgical evaluation. Thirty-one (67%) of these patients had mesenteric ischemia, with a mortality rate of 67.7% (21 patients). Predictors of death from GI complications were New York Heart Association class III and IV heart failure, smoking, chronic obstructive pulmonary disease, history of syncope, aspartate aminotransferase level higher than 600 units/L, direct bilirubin level more than 2.4 mg/dL, pH less than 7.30, and the need for more than 2 vasopressors.

Andersson et al[4] analyzed data prospectively collected from 6186 cardiac surgery patients. Forty-seven (0.8%) experienced GI complications, most commonly upper GI bleeding. The most lethal complication was mesenteric ischemia (10 patients, 80% mortality). Nine variables independently predicted major GI complications: age more than 80 years, active smoker, preoperative inotropic support, New York Heart Association class III and IV heart failure, cardiopulmonary bypass time greater than 150 minutes, postoperative atrial fibrillation, postoperative heart failure, reoperation for bleeding, and postoperative vascular complications.

The prevalence of, and predictive factors associated with, GI complications in other common intensive care unit (ICU) diagnoses such as acute respiratory failure, sepsis, and heart failure is less well defined. As in the studies of surgical patients mentioned above, GI complications are related to both underlying disease processes and therapeutic interventions. Stress-related mucosal damage (SRMD), altered gastric motility, mesenteric ischemia, and diarrhea have all been described in acute respiratory failure and mechanical ventilation.[5] Intraabdominal hypertension (IAH) is another serious GI complication in critically ill patients, particularly those with abdominal surgery, massive fluid resuscitation (>5 L/24 h), ileus, pneumoperitoneum or hemoperitoneum, and/or abdominal infection.[6]

Gastrointestinal Complications

Stress-related Mucosal Damage

Splanchnic hypoperfusion is a common pathophysiologic mechanism leading to mucosal ischemia and GI dysfunction. The GI vasculature is unable to autoregulate or compensate for reduced systemic blood pressure. Any low cardiac output state, vasopressor use, or mechanical ventilation will cause splanchnic hypoperfusion. This hypoperfusion leads to ischemia, reduced bicarbonate secretion, decreased upper GI motility, and acid back-diffusion.[7]

Mechanical ventilation affects splanchnic perfusion in several ways. First, positive end-expiratory pressure may decrease cardiac output and thus splanchnic perfusion. Second, positive end-expiratory pressure causes sympathetic activation. Catecholamines redistribute blood flow away from the splanchnic vascular bed, resulting in increased vascular resistance. The use of catecholamines for hemodynamic support creates the same effect. Mucosal ischemia results from an imbalance between oxygen supply and demand. Third, emerging evidence suggests that mechanical ventilation causes cytokine release and propagates a systemic inflammatory response that may damage the GI tract.[7,8]

Mucosal ischemia contributes to a decreased capacity to neutralize hydrogen ions. Intramucosal acidosis, cell death, and ulceration (SRMD) follow. Most critically ill patients have endoscopically detectable erosions within 24 hours of admission to the ICU. Ulceration or erosion due to SRMD does not contain inflammation or *Helicobacter pylori* as is seen in peptic ulcer disease, and the signs and symptoms of ulcerative disease are not present.[9] Clinically significant bleeding due to SRMD (erosions through the mucosa into the submucosa and muscularis propria) occurs in 3% to 4% of patients not given prophylaxis. Increased morbidity, mortality, and ICU length of stay result.[7]

Hypomotility

Impaired motility can cause a functional, nonmechanical obstruction, most commonly an adynamic ileus. Impaired motility is caused by intestinal ischemia, electrolyte imbalances such as hypokalemia, peritoneal injury, abdominal surgery, lower-lobe pneumonia, pancreatitis, cholecystitis, intraabdominal abscesses, and medications (opiates, dopamine, diltiazem, verapamil, and anticholinergics).

Distention due to the accumulation of gases and fluids begins almost immediately after an obstruction develops. Initially, the absorption of water and electrolytes is reduced. After 24 hours, sodium and water move into the bowel lumen, causing further distention and fluid loss. The fluid and electrolyte losses may be significant, contributing to hypovolemia, renal insufficiency, and shock.[10]

Intramural blood flow may be compromised with extreme distention, resulting in ischemia. When blood supply is impaired, bacterial invasion of the bowel wall can occur. This bacterial translocation may cause septicemia.[5,7,10,11]

Mesenteric Ischemia

Mesenteric ischemia occurs when 2 of the 3 branches of the abdominal aorta that supply blood to the stomach and intestines—the celiac axis and the superior and inferior mesenteric arteries—are compromised. Nonocclusive mesenteric ischemia may be caused by any low cardiac output state, vasopressor use, or vasculature compression due to IAH. Reperfusion after prolonged periods of hypoperfusion can also be damaging.

Common causes of acute occlusive mesenteric ischemia include aortic emboli, typically due to atrial fibrillation, mitral valve disease, or prosthetic heart valves. Emboli from the heart readily enter the superior mesenteric artery because of its direct line of flow from the aorta, causing ischemia of the small intestine. Thrombosis from atherosclerotic disease, trauma, or hypercoagulable states may also cause acute occlusive ischemia.

Ischemia initiates a cascade of events culminating in ulceration, increased capillary permeability, and tissue necrosis. Erosion and ulceration occur as described above. Because of increased capillary permeability, fluid moves from the blood vessels into the bowel wall and peritoneum, contributing to hypovolemia and hypoperfusion. The microvasculature is exposed to the bacteria-rich contents of the intestines; therefore, gangrene, peritonitis, and sepsis may occur.[5,7,10]

Diarrhea

Diarrhea in the critically ill patient is difficult to diagnose. Reported prevalence rates vary widely, due in large part to the lack of a standard definition. Etiologies include intolerance of enteral feedings, microbial contamination of formulas, malnutrition, hypoalbuminemia, infectious processes, acute diverticulitis, and ischemic bowel disease.[12] In addition, virtually all medications are known to cause diarrhea. Drugs commonly used in critical care that may cause diarrhea include antidysrhythmics, antibiotics, antihypertensives, and potassium supplements. Medications formulated as elixirs, for example, acetaminophen, furosemide, and metoclopramide, may contain significant amounts of sorbitol, which can cause diarrhea.[13]

Diarrhea secondary to antibiotic administration occurs between the 5th and 10th day of treatment. The likely mechanism is the overgrowth of competitive microorganisms such as *Clostridium difficile*. Mucosal destruction, widespread inflammation, and the formation of pseudomembranes (pseudomembranous colitis) occur. Recognition and treatment are essential as toxic colon dilation and perforation can occur.[13] Patients with fever and diarrhea who have received an antibiotic (especially a cephalosporin, clindamycin, or ampicillin) within the past 3 weeks should have a stool specimen analyzed for the *C difficile* toxin.[14]

Although not directly related to increased mortality, diarrhea is distressing for patients and staff. It can impair the delivery of enteral nutrition, contribute to skin breakdown, contaminate wounds, and increase nursing workload. In addition, diarrhea can cause acid-base, hemodynamic, and fluid and electrolyte imbalances.

Intraabdominal Hypertension

Interest in intraabdominal hypertension (IAH) has increased dramatically in recent years. The normal pressure in the abdominal cavity (a closed compartment) can be elevated for many reasons, and it is now well understood that increased intraabdominal pressure (IAP) can result in abdominal compartment syndrome. Causes include intraabdominal bleeding, ileus, pancreatitis, mesenteric ischemia, massive volume replacement, and the use of

vasopressors.[15] Abdominal compartment syndrome, a potentially life-threatening situation, occurs when organ function is compromised.

Critically ill patients typically have microvascular derangements that result in capillary leakage and tissue edema. In the abdominal cavity, the bowel will expand with tissue edema, causing increased IAP. The increased pressure further compromises the vasculature and a vicious cycle ensues. As IAP surpasses 20 mm Hg, abdominal compartment syndrome is likely to occur. Congestion and edema of the intestinal wall and mesenteric tissue, mesenteric ischemia, reduced hepatic blood flow, metabolic acidosis, bowel death, and liver dysfunction are the pathophysiologic changes that occur within the GI system. Systemically, blood flow is decreased to all major organs, even those outside the abdominal cavity. In addition, preload decreases and cardiac output drops, further compromising organ and tissue perfusion. Work of breathing increases because of elevation of the diaphragm, increased intrathoracic pressure, and reduced lung volumes. Neurologic injury is possible owing to increased intracranial pressure and cerebral edema. Coagulopathy and immunosuppression may occur as a result of hypoperfusion of the liver and bone marrow.[16]

Until recently, IAH did not have any standard definition. Estimates of prevalence were therefore varied and reported to be between 18% and 81%, depending on the standard used and the patient population. In 2004, the World Society of the Abdominal Compartment Syndrome published consensus definitions and recommendations. Normal IAP in critically ill adults is 5 to 7 mm Hg. IAH is defined as sustained or repeated elevation of 12 mm Hg or more. The exact level requiring intervention has not been established. Moderate elevations (20–40 mm Hg) in symptomatic patients warrant consideration of intervention. Pressure is measured via the bladder, either through assembled components or a commercially available monitoring system.[6] An example is shown in Figure 1.

To evaluate the effects of IAH on organ function, Malbrain et al[17] enrolled 265 patients in a prospective, epidemiologic study. Fourteen ICUs in 6 countries enrolled patients in the study. There was no difference in ICU admission diagnosis (medical or surgical) and the presence of IAH, which (IAP > 12 mm Hg) was present in 85 (32.1%) of the patients. IAH on admission was associated with severe organ dysfunction during the ICU stay. Eleven patients (4.2%) developed abdominal compartment syndrome, defined for this study as an IAP higher than 20 mm Hg accompanied by failure of at least 1 organ. Abdominal surgery, fluid resuscitation, ileus, and liver dysfunction independently predicted IAH. The occurrence of IAH during the ICU stay was an independent risk factor for organ failure and mortality.

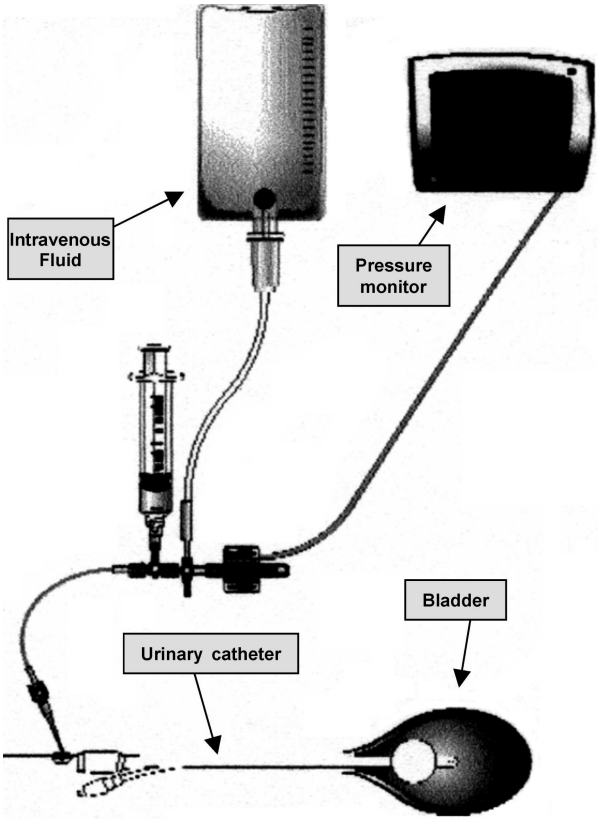

Figure 1: Intraabdominal pressure monitoring with assembled components.
Used with permission from Wolfe T, Kimball E, Bernstone T. Intra-abdominal pressure monitoring: the key to avoiding abdominal compartment syndrome. *AACN News.* 2004;21(9):14–18.

Prevention Strategies

Primary prevention strategies for SRMD, hypomotility, mesenteric ischemia, diarrhea, and IAH include a thorough assessment, risk factor recognition, assurance of adequate GI perfusion, and stress ulcer prophylaxis. Assessment is challenging because many clinical manifestations of GI complications are nonspecific. For example, multiple disorders cause abdominal pain, GI bleeding, and diarrhea. Practitioners must also be able to recognize factors in the history and physical examination

that indicate risk for GI complications. The history and physical examination may be limited, as critically ill patients are often unable to provide an accurate history or subjective data regarding symptoms. The absence of early, specific clinical signs places patients at risk for late diagnosis of a serious complication. Imaging studies may be necessary for accurate evaluation.

Assess Abdominal Pain

Abdominal pain is a sensitive, nonspecific indicator of GI pathology. There are 3 broad categories of abdominal pain: visceral, parietal, and referred.

Visceral pain occurs when hollow organs such as the gallbladder or stomach contract forcefully or are distended. Solid organs such as the liver can also be painful when distended. Visceral pain is poorly localized and is often present near the midline. Epigastric pain may be caused by the liver, biliary tree, stomach, duodenum, or pancreas. Periumbilical pain may be from the small intestine, proximal colon, or appendix. Hypogastric pain may be caused by the colon, bladder, or uterus. Visceral pain is often described as gnawing, burning, cramping, or aching. When severe, associated symptoms include sweating, pallor, nausea, vomiting, and restlessness.

Parietal pain originates in the parietal peritoneum and is caused by inflammation. It is described as steady and aching and is more precisely localized over the involved structure. Parietal pain is aggravated by moving or coughing.

Referred pain is felt in sites that are distant from the involved organ but innervated by the same spinal levels. Referred pain radiates or travels from the initial site. It is usually well localized and may be superficial or deep. Common sites for referred pain include the back for pancreatic or duodenal disorders and the right shoulder or right posterior chest for biliary tree disorders.[18] Pain patterns associated with selected GI complications are described in Table 1.

Assess GI Bleeding

Bleeding in the esophagus, stomach, or duodenum (upper GI bleeding) is frequently caused by ulcers, SRMD, Mallory-Weiss tears, varices, or cancer. Bleeding from the jejunum, ileum, colon, or rectum (lower GI bleeding) is often caused by polyps, inflammatory disease, cancer, mesenteric ischemia, or hemorrhoids. Active bleeding is recognized by 1 or more of the following 3 clinical signs:

- Hematemesis—Bloody vomitus, either bright red, fresh blood or dark, grainy digested blood (resembling coffee grounds).
- Melena—black, sticky, tarry stools caused by digestion of at least 60 mL of blood in the GI tract. Melena typically occurs if blood loss is from the esophagus, stomach, or duodenum; it may be apparent from blood loss lower in the GI tract if transit is slow.
- Hematochezia—fresh, bright red blood passed from the rectum, usually originating from the colon, rectum, or anus. Blood loss may originate higher in the GI tract if transit is rapid. Upper GI bleeding in excess of 1 L can cause hematochezia.

Occult bleeding may be recognized by trace amounts of blood in the stool or gastric secretions, slowly decreasing hemoglobin and hematocrit levels, or unexplained elevations in the blood urea nitrogen level with stable creatinine. The blood urea nitrogen level will increase as a result of the digestion of blood in the GI tract. These findings should trigger testing for occult blood in the upper GI drainage or the stool. Occult blood is detectable with a guaiac test.[18] The severity of blood loss can be estimated by the signs and symptoms described in Table 2.

Perform Imaging Studies

Using imaging studies to evaluate the abdomen is ideal considering the limitations associated with history and physical examination. Unfortunately, transportation of patients to the radiology department may present an unacceptable risk. Bedside studies include plain radiographs to assess for obstruction or perforation. Ultrasound can also be performed at the bedside and is the preferred study if cholecystitis or cholangitis is suspected.

For patients who can leave the ICU, computed tomography is the first choice for imaging the GI system. Pancreatitis, hemorrhage, perforation, abscess, ischemia, and obstruction can be visualized with this technique. The use of oral contrast permits better visualization of the viscera. Intravenous contrast is useful for solid organ assessment and the identification of infectious processes, inflammatory processes, ischemia, and hemorrhage. Computed tomographic angiography or magnetic

Table 1: Assessment Findings Associated With Common Gastrointestinal Complications[10,16,18,19]

Problem	Pain Location and Description	Associated Signs and Symptoms
Paralytic ileus (hypomotility)	• Periumbilical pain, cramping • Pain subsides as motility decreases • Minimal tenderness or rigidity	• Abdominal distention • Vomiting (gastric contents, bile) • Increased peristaltic waves and high-pitched, "tinkling" bowel sounds • Tympany on percussion • Palpable dilated bowel loops • Singultus is common • Dehydration, hypovolemia, and metabolic acidosis will occur within 24 h of complete obstruction
Mesenteric ischemia –	• Periumbilical then diffuse pain, cramping, then steady • Pain becomes more steady and severe as ischemia progresses to necrosis or perforation	• Vomiting • Abdominal distention • Hyperactive, then absent bowel sounds • Bloody diarrhea • Leukocytosis • Metabolic acidosis • Shock • Bruit with systolic and diastolic component over partial arterial occlusions
Diarrhea	• Infection by viruses or bacteria: periumbilical cramping pain • Drug induced: little or no pain	• Stools watery without blood, pus, or mucus • Nausea, vomiting, and slight fever • Stools loose and watery • Nausea
Intraabdominal hypertension		• No specific signs or symptoms, and clinical examination is inaccurate • Recognition requires a high index of suspicion, and intraabdominal pressure monitoring is recommended

resonance imaging may be necessary to identify early ischemia. Upper and lower endoscopy can be both diagnostic and therapeutic in patients with GI bleeding. Although endoscopy can be performed at the bedside, patient instability may limit its use.[21]

Recognize Risk Factors

Several important risk factors are common to most critically ill patients. Low cardiac output states, the use of vasopressors, and mechanical ventilation are all associated with mucosal damage, nonocclusive mesenteric ischemia, and IAH. Examples of specific risk factors include atrial fibrillation as a potential cause of occlusive mesenteric ischemia and hypomotility due to electrolyte imbalances.

Initiate Stress Ulcer Prophylaxis

The authors of the Surviving Sepsis Campaign Guidelines thoroughly reviewed the literature and recommended the use of histamine$_2$

Table 2: Signs and Symptoms of Blood Loss[20]

Amount of Blood Loss	Signs and Symptoms
<15% (<1000 mL)	Blood pressure normal, heart rate normal or <100 beats/min supine, urine output normal, capillary refill normal, may be anxious
15–29% (1000 mL)	Blood pressure normal when supine, postural hypotension, heart rate 100–120 beats/min, capillary refill >3 s, increased respirations, urine output 25–30 mL/h, weakness, may have mental status changes
30–39% (1500–2000 mL)	Systolic blood pressure (90 mm Hg, heart rate >120 beats/min; cool, pale skin; increased respirations, urine output 10–15 mL/h, mental status changes
>40% (>2000 mL)	Mean arterial pressure <50 mm Hg, heart rate >140 beats/min, confused, lethargic, cold clammy skin, urine output minimal

receptor antagonists for prophylaxis. These agents have been demonstrated to work better than sucralfate. New data suggest that proton pump inhibitors suppress acid production more completely and they are gaining widespread use for prophylaxis. They are not all approved by the United States Food and Drug Administration for this purpose and direct comparison of proton pump inhibitors with histamine$_2$ antagonists has not been reported. The relative efficacy of proton pump inhibitors is therefore unknown, and more studies are needed to assess their safety and effectiveness. More recently, SRMD prophylaxis protocols

Table 3: General Prevention Strategies[5,7,25]

Goal	Strategies
Optimize cardiac output and tissue perfusion	• Manage fluid balance; transfuse as indicated; monitor possible sources of fluid loss • Evaluate effectiveness of fluid resuscitation • Monitor for and treat dysrhythmias • Minimize/eliminate environmental stressors • Monitor determinants of tissue oxygen delivery • Position patient for optimal perfusion • Maintain oxygenation and ventilation
Limit use of vasopressors	• Accurately measure systemic blood pressure; set appropriate parameters • Manage fluid balance; monitor possible sources of fluid loss • Evaluate effectiveness of fluid resuscitation • Treat underlying causes of hypotension • Monitor tissue perfusion
Decrease mechanical ventilation days	• Use research-based weaning protocols • Limit sedation as appropriate and implement daily interruption of sedation protocols • Initiate early mobilization • Prevent ventilator-associated pneumonia • Allow spontaneous breathing whenever possible

Table 4: Specific Strategies for Common Complications[2,7,8,12,13,17,22,24,26]

Complication	Strategies
Stress-related mucosal damage	• Initiate stress ulcer prophylaxis with a regimen that considers individual patient risk factors and disease state
	• Consider off-pump bypass surgery as an alternative to cardiopulmonary bypass
	• Monitor for and treat coagulopathies
Hypomotility	• Maintain electrolyte balance, especially potassium and magnesium
	• Promote peristalsis with frequent position changes and early mobilization
	• Initiate bowel program for patients with limited mobility and/or opiate use
	• Consider use of prokinetic agents such as metoclopramide or erythromycin to enhance gastroduodenal motility
	• Maintain effective decompression of gastrointestinal tract when indicated
Mesenteric ischemia	• Convert atrial fibrillation when possible or provide anticoagulation
	• Strictly monitor anticoagulation
	• Avoid or treat hypercoagulable states
	• Monitor intraabdominal pressure in at-risk patients
Diarrhea	• Use low-osmolarity formulas with fiber
	• Limit use of high-sorbitol elixirs, magnesium-based antacids, laxatives, and stool softeners
	• Ensure early recognition and treatment of *Clostridium difficile* infection
	• Monitor for hypoalbuminemia
	• Follow aseptic technique when handling enteral feeding systems
Intraabdominal hypertension	• Obtain baseline intraabdominal pressure in at-risk patients
	• Monitor for renal, pulmonary, cardiovascular, and neurologic signs indicating the development of abdominal compartment syndrome

recommend taking into account individual risk factors and disease states.[22–24] A summary of strategies used to limit the risk of GI system complications is described in Tables 3 and 4.

Summary

GI complications contribute to morbidity and mortality in the critically ill patient. Common pathophysiologic changes in critical illness are direct risk factors for the development of GI dysfunction. Risk is also increased by supportive interventions such as mechanical ventilation and vasopressors. It may seem then, that the development of GI complications is a given in critically ill patients requiring this level of support. However, clinicians can limit complications as they understand individual patient characteristics, thoughtfully evaluate the risk-benefit profile of all interventions, and implement preventive strategies.

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