Gastrointestinal dysmotility in the ICU

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Purpose of review
This review aims to provide a comprehensive overview of gastrointestinal dysmotility, particularly in critically ill patients within the ICU. It highlights the pathophysiology, prevalence, and clinical implications of conditions, such as oesophageal dysmotility, gastroparesis, ileus, and Ogilvie's syndrome. By examining current diagnostic and treatment approaches, the review emphasizes the importance of recognizing and managing gastrointestinal dysmotility to improve patient outcomes.

Recent finding
Recent literature indicates that up to 60% of ICU patients experience some form of gastrointestinal dysmotility, with those on mechanical ventilation being particularly at risk. The review identifies key contributors to gastrointestinal dysmotility, including inflammatory states, electrolyte imbalances, and the effects of certain medications. Nonpharmacological strategies, such as early enteral feeding, correcting electrolyte abnormalities, and mobilization are critical. Prokinetic agents have shown promise in alleviating feeding intolerance and improving gastric emptying, though their effects on overall mortality remain inconclusive.

Summary
Gastrointestinal dysmotility presents a significant challenge in critically ill patients, leading to various complications that hinder recovery. Understanding the underlying pathophysiology, coupled with effective diagnostic and treatment strategies, is essential for enhancing patient care. This review underscores the need for continued research and clinical focus on gastrointestinal motility disorders in the ICU to improve health outcomes for this vulnerable population.

Keywords
acute colonic pseudo-obstruction, critically ill patient, oesophageal dysmotility, gastrointestinal dysmotility, gastroparesis, ileus, Ogilvie's syndrome, prokinetics

INTRODUCTION

The gastrointestinal system is vital to the general health and well being of individuals, particularly in critically ill patients who often face numerous challenges. Gastrointestinal dysmotility, a common occurrence among individuals in the ICU, can significantly impact patient outcomes. Although mortality prediction scores do not currently include gastrointestinal dysmotility, their effects on mortality are important [1].

The prevalence of gastrointestinal dysmotility in the ICU is substantial, with up to 60% of patients being affected [2]. Notably, patients on mechanical ventilation and those with raised intracranial pressure post head trauma are particularly susceptible to abnormalities of gastric emptying, with up to 50 and 80% experiencing this issue, respectively [3,4]. The implications of gastrointestinal dysmotility are profound, and encompass complications, such as bacterial translocation, which may cause sepsis ventilator-associated pneumonia, and malnutrition, all of which can significantly impact patient recovery and well being [5,6].

The purpose of this study is to present a concise overview of the pathophysiology of gastrointestinal dysmotility, and physiology of gastrointestinal motility along with an exploration of current diagnostic and treatment approaches employed in the intensive care setting. Through this review, we hope to shed light on the significance of addressing gastrointestinal dysmotility in critically ill patients and offer insights into potential avenues for improving patient care and prognosis.

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KEY POINTS

- Gastrointestinal dysmotility affects up to 60% of ICU patients, particularly those on mechanical ventilation or raised intracranial pressure, leading to severe complications, such as infections and malnutrition.
- Common causes of gastrointestinal dysmotility include inflammatory states, medication effects (e.g. opioids), and electrolyte imbalances, complicating the management of critically ill patients.
- Diagnostic methods like gastric emptying scintigraphy are often impractical in the ICU, necessitating alternative approaches, such as bedside ultrasound to assess gastric residual volume.
- Effective management involves a combination of nonpharmacological interventions (e.g. electrolyte correction) and pharmacological treatments, primarily prokinetic agents, to enhance gastrointestinal motility.
- Addressing gastrointestinal dysmotility may improve outcome in critically ill patients, highlighting the need for better diagnostic and treatment protocols in intensive care settings.

PATHOPHYSIOLOGY AND CLINICAL EFFECTS OF GASTROINTESTINAL DYSMOTILITY

The exact cause of gastrointestinal dysmotility is still not clear, but common causes include various types of inflammatory/shock states associated with raised cytokines like septic shock, cardiogenic shock, burns, traumatic brain injuries, polytrauma, comorbidities (like diabetes mellitus type 2, Parkinson's disease, amyloidosis, etc), electrolyte abnormalities, advanced age, drugs like opioids, alpha-adrenergic agonists, and abdominal surgery [7▪]. Pathophysiology and associated clinical features are illustrated in Fig. 1.

In this article, dysmotility syndromes commonly observed in ICU, for example, oesophageal dysmotility, gastroparesis, ileus, and Ogilvie syndrome and their context in the recent literature will be discussed.

OESOPHAGEAL DYSMOTILITY

Oesophageal motility disorders can be classified as either primary (because of oesophageal disease motility) or secondary (from the tumour, compression, scleroderma, etc.). In this review, we will discuss only primary oesophageal motility disorder. These disorders are frequently found in patients with alcohol abuse, diabetes, critically ill patients on opioids, ketamine, benzodiazepines (drugs that inhibit oesophageal motor activity), etc. In ICU patients, both the amplitude and frequency of the contraction of the oesophagus for propulsion are reduced [2].

Diagnosis of oesophageal motility disorder requires eliciting an appropriate history like chest pain, difficulty in swallowing or chronic use of opioids, which is linked to spastic oesophageal contractions and poor relaxation of the lower oesophageal sphincter (LES) by Babaei *et al.* [8]. Diagnostic methods for oesophageal dysmotility include endoscopy, barium swallow, high-resolution manometry and functional lumen imaging probe [9▪▪]. However, the clinical use of these diagnostic methods in the ICU setting is limited.

Gastro-oesophageal reflux (GER) disease has been defined as a condition that arises when the reflux of the contents from the stomach causes symptoms and/or complications, according to Montreal Global consensus [10]. It has been suggested that nasogastric intubation, a frequent procedure for critically ill patients, is the cause of GER. A positive association has also been noted between the length of nasogastric intubation and the severity of erosive oesophagitis. Studies have demonstrated that reflux episodes in patients on mechanical ventilation are primarily caused by low or nonexistent LES pressure (LESP), frequently accompanied by a strain or cough [2].

GASTRIC DYSMOTILITY

Gastroparesis is characterized by a decrease in stomach motility that prolongs food retention in the stomach and causes related symptoms [11]. In addition to the usual symptoms of nausea, vomiting, early satiety, and postprandial fullness, gastroparesis patients frequently have epigastric pain, bloating, and belching. Mechanical obstruction of the gastrointestinal tract needs to be ruled out to make a diagnosis of gastroparesis [12]. Gastroparesis is diagnosed by confirming a delay in gastric emptying. Several complex factors contribute to the pathophysiology of delayed gastric emptying, such as impairments in duodenal motility, pyloric function, and gastric accommodation [13].

The conventional gold standard for determining the rate of stomach emptying is gastric emptying scintigraph [14]. The solid gastric meal retention of more than 10% at 4 h after ingestion is an established, reproducible, and validated criterion for diagnosis of delayed gastric emptying [15]. Although the relationship between gastric emptying rate and gastrointestinal symptoms has been controversial, studies using scintigraphy with a solid meal and gathering data for at least 3 h after ingestion showed a positive correlation between gastric

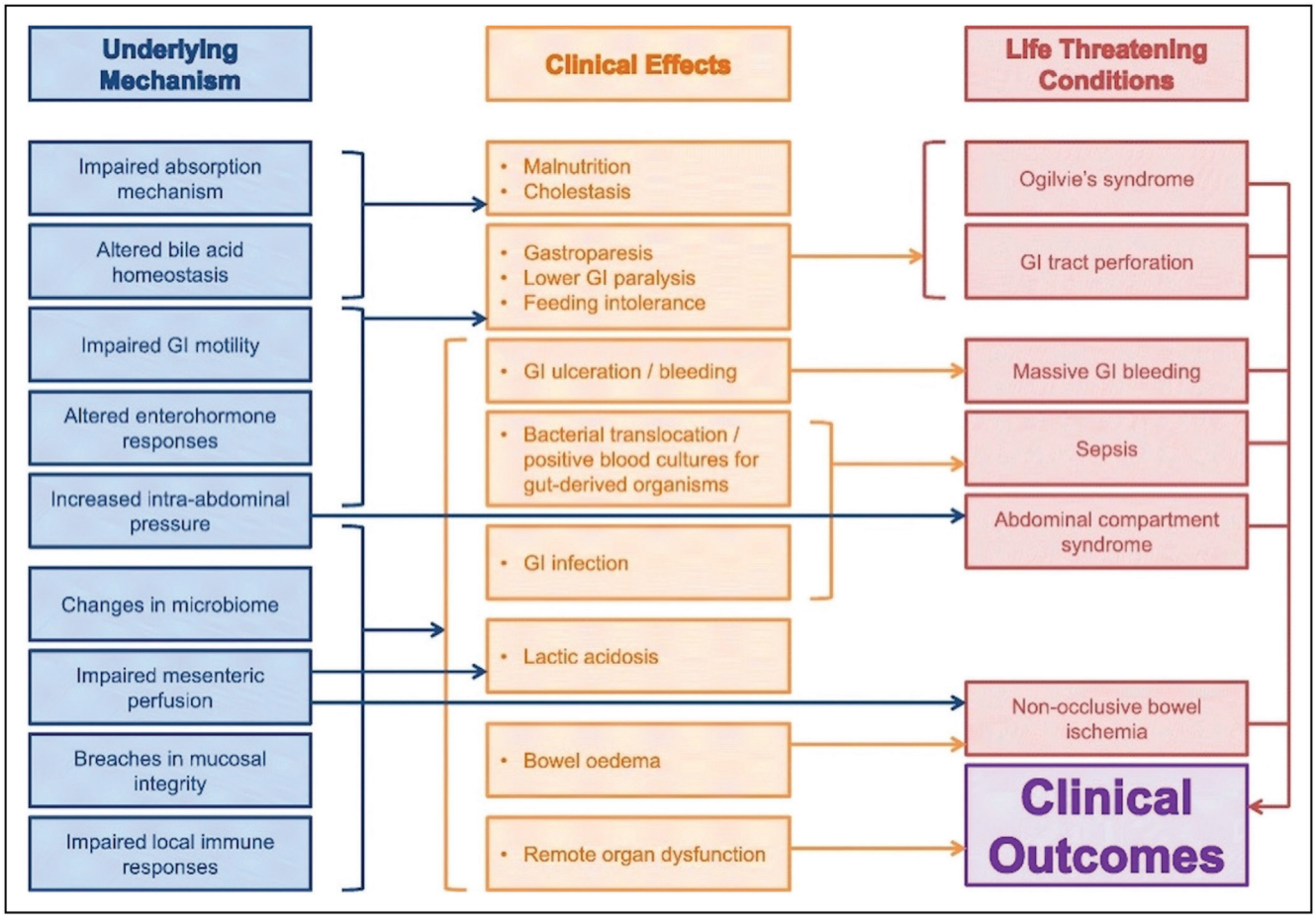

FIGURE 1. Pathophysiology and clinical features of gastrointestinal dysmotility. ACS, abdominal compartment syndrome; GI, gastrointestinal; IAP, intraabdominal pressure. The original image is under Creative Commons Attribution 4.0 International License from Reintam Blaser A *et al.* Working Group on Gastrointestinal Function within the Section of Metabolism, Endocrinology and Nutrition (MEN Section) of ESICM. Gastrointestinal dysfunction in the critically ill: a systematic scoping review and research agenda proposed by the Section of Metabolism, Endocrinology and Nutrition of the European Society of Intensive Care Medicine. *Crit Care* 2020;24(1):224.

emptying, the severity of nausea, vomiting, abdominal pain, and early satiety/fullness [16]. Qutbi *et al.* [17▪] have suggested that extending the evaluation to 4 h compared to 3 h has little impact on the ultimate diagnosis of delayed gastric emptying and may not be significantly useful. The current American College of Gastroenterology (ACG) clinical guideline for gastroparesis recommends gastric emptying scintigraphy as the first-line test for patients with signs and symptoms suggestive of gastroparesis. This test measures solid meal emptying over a minimum of 3 h [11]. For gastric emptying scintigraphy, Shah *et al.* [18] in their retrospective analysis have found similar results with 50% consumption of the standard scintigraphy meal. Orthey *et al.* have demonstrated that dynamic scintigraphy during gastric emptying scintigraphy can be used to measure duodenal bolus propagations following meal ingestion. Merely 12% of the antral contractions within the first 60 min after meal ingestion result in the propagation of duodenal boluses. This methodology seems promising when evaluating antropyloroduodenal coordination in patients exhibiting unexplained upper gastrointestinal dysmotility symptoms [19].

Often in a critically ill patient, performing tests like gastric emptying scintigraphy may not be possible, and hence direct measurement of gastric emptying is generally not done. Instead, intensivists typically rely on the measurement of gastric residual volume (GRV). GRV is the amount of fluid drained/aspirated from the stomach after enteral feed. The easiest way to measure GRV in a critically ill patient is by measuring nasogastric tube aspiration volume; however, it is not a risk-free procedure. Arunachala Murthy *et al.* [20▪] have found in ICU patients that even after adjusting for sickness severity, large GRVs were related to higher mortality and were more

prevalent in men and those who consumed formulas, which were energy-dense (>1.5 kcal/ml). In a Cochrane review of eight randomized control studies, Yasuda *et al.* [21] expressed uncertainty about the effect of GRV on clinical outcomes, including hospital stay duration, pneumonia, vomiting, and death. Basher *et al.* [22▪] in their pilot study have demonstrated high specificity (90%) and 80% efficacy of the noninvasive, risk-free electrical impedance approach for measuring gastric volume in an ICU setting. Bedside point-of-care ultrasound (POCUS) can be an excellent tool for GRV assessment in any ICU for a critically ill patient (Fig. 2). Ankalagi *et al.* studied 43 critically ill patients using serial ultrasound GRV measurements. The stomach residual volume was computed using the antral cross-sectional area (CSA), which is the product of the anteroposterior (AP) and craniocaudal diameters of the gastric antrum determined using ultrasonography in the right lateral decubitus position. Before the enteral feed was started, a baseline measurement was made. For the first 4 h, the ultrasound scan was repeated every hour. During this time, the patients were monitored for feed intolerance. They concluded that GRV can be measured using ultrasound to predict feed intolerance with an area under the receiver operative curve (AUROC) of 99.3% and a sensitivity of 100%, specificity of 99% at 4 h [23▪]. Brotfain *et al.* used a POCUS-based approach to prospectively analyse the measures of GRV and nasogastric tube positioning that were repeated by nurses in the ICU. The study showed a good association between the use of POCUS for nasogastric tube positioning and assessment of GRV and standard protocol of syringe aspiration, indicating that it is a secure, straightforward, and efficient tool for critical care unit nurses [24▪]. Although there are no established clinical characteristics that characterize upper gastrointestinal dysmotility, the ESPEN guidelines recommend that a GRV greater than 500 ml over 6 h should prompt stopping additional feeds, performing abdominal examination to rule out ileus or bowel obstruction, and administration of prokinetics should be considered [25]. ESPEN suggests delaying enteral nutrition if GRV is greater than 500 ml over 6 h and considering postpyloric feeding if gastric feeding intolerance is not solved with prokinetics [26▪▪].

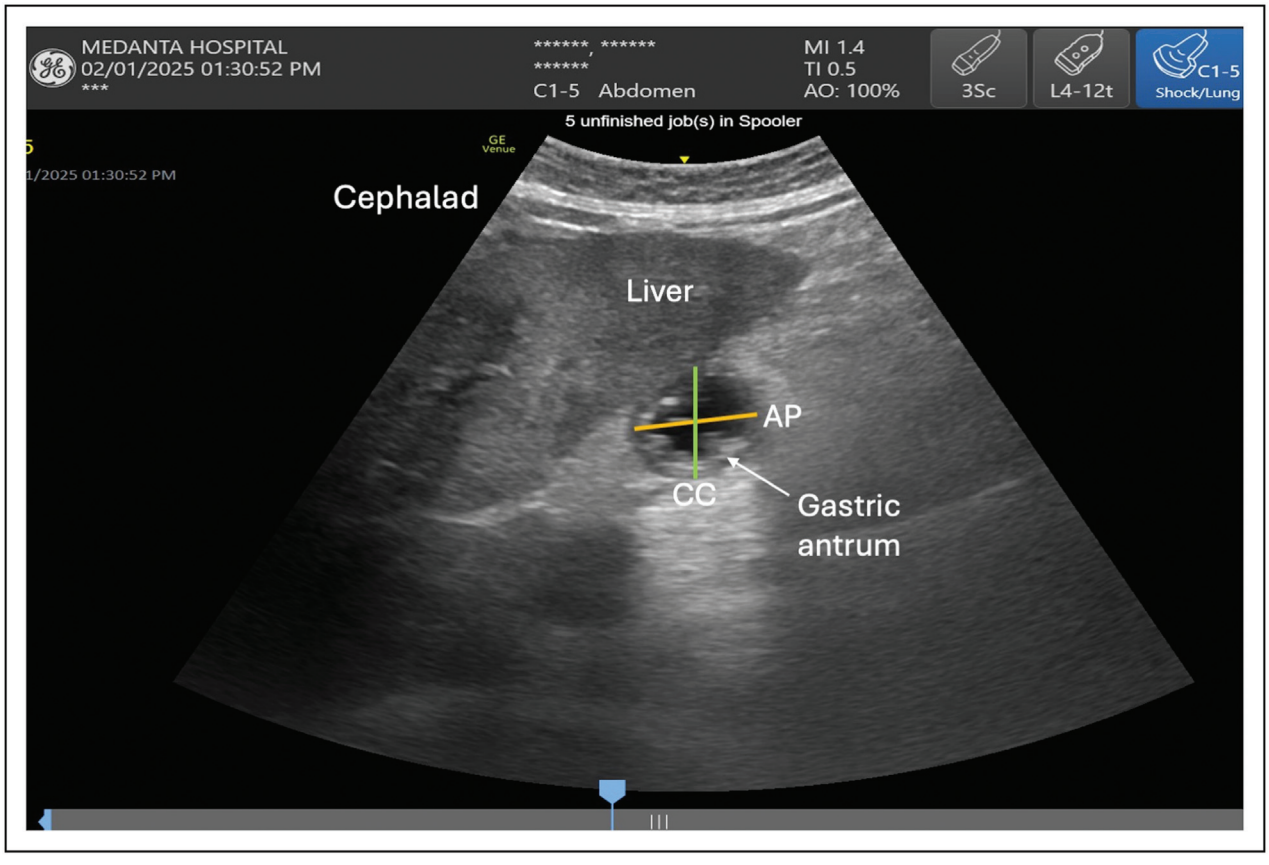

FIGURE 2. Gastric residual volume calculation. Antral cross-sectional area (ACSA; cm^2) = (AP × CC × π)/4, GRV (ml) = 27 + 14.6 ACSA − 1.28 * age (years). AP, anteroposterior diameter; CC, craniocaudal diameter. From Perlas A, *et al.* Validation of a mathematical model for ultrasound assessment of gastric volume by gastroscopic examination. *Anesth Analg.* 2013;116:357–363.

ILEUS

Paralytic ileus is the most frequent clinical sign of small intestine dysmotility in critically ill patients [2]. Ileus is defined as a lack of regular physiological intestinal motility in the absence of mechanical obstruction, making it unable to propel its contents farther inside the gastrointestinal tract [27]. Abdominal surgery, sepsis, pancreatitis, peritonitis, narcotic usage (by activation of mu-opioid receptors in the gastrointestinal tract by commonly used opioids like morphine, fentanyl, tramadol, etc.), anticholinergic use, hypokalaemia, hypomagnesemia, hyperglycaemia, acidosis, hypoxia, hypothermia, renal failure, and mechanical ventilation are common clinical entities that predispose to ileus [28].

The role of an abdominal computed tomography (CT) scan is imperative in distinguishing between a mechanical obstruction and an ileus. A CT abdomen with oral and intravenous contrast will help identify the possible location of obstruction and rule out other disorders of the abdomen.

After elective colorectal surgery, 10–24% of patients experience postoperative ileus (POI). Koch *et al.* have postulated in their retrospective study that the probability of POI rose by 1.4 times for every extra litre of intravenous fluid administered during the first 72 h [29]. Similarly, Shim *et al.* [30], in a retrospective analysis of the Korean database on robot-assisted radical cystectomies, have found that patients had a longer length of hospital stay and POI with increased intravenous fluids. One of the mainstays of haemodynamic resuscitation for critically unwell patients is fluid resuscitation. Overzealous fluid resuscitation can have negative effects on several organ systems. When there is an inflammatory response that changes capillary permeability, as occurs during sepsis, fluid overload is more likely to occur and worsen or precipitate ileus in critically ill. De-resuscitation has been postulated as the final phase of intravenous fluid therapy in critically ill patients. There are various ways to achieve de-resuscitation, like diuretics or ultrafiltration [31]. In a more recent systematic review and meta-analysis by Messmer *et al.* on aggressive fluid de-resuscitation in individuals suffering from septic shock who were critically ill, the authors did not find any evidence that active fluid de-resuscitation was better than standard care in terms of patient-centred outcomes, fluid balance, or mortality in patients suffering from septic shock. This was primarily because of heterogeneous de-resuscitation techniques and the small sample size of the studies [32].

OGILVIE'S SYNDROME

Also known as acute colonic pseudo-obstruction (ACPO) is a large intestine functional condition characterized by dysmotility of the colon that leads to distension without any mechanical obstruction. The underlying pathophysiology of ACPO remains unknown despite technical advances in studying the physiology of colonic motility, including spatiotemporal mapping and high-resolution manometry [33▪]. The prevailing theory holds that it results from the colon's enhanced sympathetic and diminished parasympathetic activity, which impairs peristalsis [27]. The common causes of ACPO include congestive cardiac failure, myocardial infarction, trauma, burns, cerebrovascular accident, dementia, multiple sclerosis, infections like Herpes zoster, pneumonia, surgery (abdominal, pelvic, gynaecological, etc), malignancy and medications (opioids, antidepressants, anticholinergics) [33▪].

Common symptoms include the inability to pass flatus or stool; however, some may present with diarrhoea. Abdominal distention is common, but worsening fever with pain in the abdomen should alert a clinician for suspected perforation and peritonitis. A plain X-ray of the abdomen may be used as a bedside screening tool for suspected ACPO. A CT scan with rectal, intravenous, and oral contrast is advised as the preferred diagnostic technique. Rectal contrast enhances diagnostic accuracy. As an alternative to CT, fluoroscopy with rectal contrast may have an additive therapeutic impact [33▪]. Acute colon obstruction and toxic megacolon are distinguished from ACPO on imaging by the haustrations, which are maintained in ACPO [10]. Although the left colon may also be impacted, the cecum, ascending colon, and transverse colon are most frequently involved [27]. Perforations in individuals with caecal diameters less than 9 cm have been reported, notwithstanding the minimal chance of perforation in patients with cercal diameters less than 12 cm [34].

MANAGEMENT OF GASTROINTESTINAL DYSMOTILITY

Nonpharmacological management

Correcting electrolyte imbalances, avoiding opioid agonists and anticholinergic medications, mobilizing patients, and, whenever feasible, initiating early enteral feedings are all part of the fundamental care of ileus. Patients receiving small peptide formulae had higher levels of prealbumin, higher albumin growth, and higher daily protein intake than patients receiving standard polymeric formulae in critically ill patients with acute gastrointestinal injury, according to a meta-analysis by Wang *et al.* comparing studies from 1980 to 2022. Additionally, their stays in the hospital and ICU were shorter; however, no difference was seen in all-cause

Table 1. Pharmacological agents for gastrointestinal dysmotility in the ICU

Pharmacological group	Drugs	Description	Evidence
5-HT4R agonist	Prucalopride	US FDA approved for chronic idiopathic constipation in adults. Selective agonist of 5-HT4R receptors, which significantly reduces cardiovascular risk. It induces relaxation of human colonic circular smooth muscle and contraction of colonic longitudinal smooth muscle. Only oral formulation, with 30% plasma protein binding. Mostly excreted unchanged, primarily in urine. Half-life: 18–20 h Majority of its negative effects being self-limiting and usually detected on the first day of treatment.	Might be the first line of treatment for gastrointestinal disorders, such as treatment of functional constipation and gastroparesis [40]. Meta-analysis by Ali *et al.* included eight studies in the analysis. They interpreted that using a dosage of 1–4 mg/day, prucalopride improved gastric emptying and the gastroparesis cardinal symptom index over placebo in two 4-week trials. Oral prucalopride 2–4 mg/day significantly improved the number of bowel movements and symptoms of chronic constipation in seven 12-week trials. The placebo group did not significantly improve symptoms. The most frequently reported side effects of prucalopride were headache, nausea, diarrhoea, and abdominal pain [41].
	Felcisetrag	Not yet US FDA approved. Still under trial	Chedid *et al.* evaluated intravenous Felcisetrag versus placebo and found that it significantly improved gastric emptying in gastroparesis along with small bowel and colonic transit [42]. In phase 2 study of Felcisetrag for postoperative gastrointestinal dysfunction after bowel surgery, Boeckxstaens *et al.* [43] have found that although the drug was well tolerated, it failed to show any clinically meaningful difference in time to recovery of gastrointestinal functions when compared to placebo.
	Velusetrag	Not yet US FDA approved. Still under trial	Velusetrag is also well tolerated and accelerates gastric emptying in people with idiopathic or diabetic gastroparesis [44].
	Tegaserod	US FDA approved in 2002 for irritable bowel syndrome-associated constipation. Tegaserod was withdrawn in 2007 over the concern of possible associated cardiovascular ischemic events. US FDA has now re-approved the use of the drug in women less than 65 years old with no history of cardiovascular disease. 5-HT4 receptor agonist, 5-HT2B receptor antagonist Apart from 5HT4 receptor actions, 5HT2B antagonism can lead to inhibition of both 5-HT-mediated gastrointestinal motility and visceral hypersensitivity. Oral formulation only, with 98% plasma protein binding Two-third of the oral drug is excreted unchanged in faeces, rest one-third excreted as metabolites in urine. Half-life: 4.6 to 8.1 h To be taken 30 min prior to a meal to increase absorption.	Shah *et al.* [45] reported that Tegaserod 6 mg b.i.d. reduces constipation in patients with irritable bowel disease.

Table 1 (*Continued*)

Pharmacological group	Drugs	Description	Evidence
Dopapime-2 receptor antagonist	Metoclopramide	US FDA approved indications: nausea/vomiting in GERD, gastroparesis, and chemotherapy patients. Inhibits D2 and 5-HT3 receptors in postrema area of brain to relieve symptoms of nausea and vomiting. Decreases lower oesophageal pressure, increases gastrointestinal motility (inhibits D2 receptor, agonist for 5-HT4 receptors and antagonism of muscarinic receptor inhibition) Oral bioavailability: 30–100% 30% plasma protein bound Metabolized by cytochrome P450 in liver, majority excreted in urine. Half-life: 5–6 h Overdose may lead to extrapyramidal symptoms	Mainstay option for treating gastroparesis. Metoclopramide has been found to be more efficacious than domperidone [46▪▪]. In a study on the risk of pneumonia linked to stroke in patients with dysphagia, the placebo group experienced a significantly higher number of pneumonia episodes (RR 5.24, 95% CI 2.43–11.27; $P < 0.001$) compared to the metoclopramide group [47]. When comparing the safety profile of these two drugs, 5% of patients receiving domperidone experienced clinically significant adverse events, such as QTc prolongation (95% CI 3.32–8.62). 15% of patients (95% CI 7.48–26.61) treated with metoclopramide experienced restlessness, an extrapyramidal adverse event. This was a seven-fold increase in comparison to patients receiving a placebo (OR: 7.72; 95% CI: 1.27–47.05) [48▪▪].
	Domperidone	Not US FDA approved for any indication. It is used in many countries for gastroparesis, gastroesophageal reflux disease and constipation. Prokinetic effect is by dopamine receptor blocking property. Antiemetic effect by dopamine receptor antagonism at postrema area in brain and at gastric level. 91–93% protein bound Extensively metabolized in liver by cytochrome P450 enzyme system. Excreted majorly through faeces. Half-life: 7–9 h	Metoclopramide remains the only FDA-approved drug for gastroparesis [49▪▪].
Muscarinic receptor antagonist	Neostigmine	Off label for treating acute colonic pseudo-obstruction Cholinesterase inhibitor Neostigmine is an acetylcholinesterase inhibitor that exerts strong muscarinic effects, stimulating intestinal smooth-muscle contractions and enhancing peristalsis. Given intravenous, 2 mg over 5 min or continuous infusion of 0.4–0.8 mg/h for 24 h. 15–25% plasma protein bound Hydrolyzed by cholinesterase and by microsomal enzymes in liver Half-life: 42–60 min	Neostigmine has been employed for treating gastrointestinal dysmotility, as it has been shown to enhance gastrointestinal motility. An RCT (ChiCTR2200058305) has been planned to evaluate the use of neostigmine in gastrointestinal dysmotility in acute pancreatitis [50▪].
	Acotiamide	Not yet US FDA-approved. Still under trial	Acotiamide is linked to decreased antral pressures following enteral intake [51].
Ghrelin receptor agonists	Relamorelin	Not yet US FDA-approved. Still under trial	Relamorelin is a ghrelin receptor agonist that speeds up stomach emptying and relieves discomfort, nausea, bloating, and fullness in diabetic gastroparesis patients. Furthermore, it stimulates nodose afferents and the dorsal motor nucleus of the Vagus neurons [52,53].

Table 1 (*Continued*)

Pharmacological group	Drugs	Description	Evidence
Selective NK1 receptor antagonists	Tradipitant	Not yet US FDA-approved. Still under trial	Tradipitant may be beneficial in two ways for gastroparesis: First, these drugs reduce afferent mechanisms that cause emesis by either directly targeting the brain regions accountable for nausea and vomiting, by inhibiting the impact of substance P within the central emetic circuitry or by modulating NK1 receptors on vagal afferents. Second mechanism in relieving gastroparesis involves modification of the functional interaction between the acetylcholine and NK1R systems, which is responsible for stimulating smooth muscle contractions in the stomach, by NK1 receptor antagonists [54]. In a multicentre, double-blind, placebo-controlled experiment, including 152 persons with gastroparesis treated for 4 weeks, Tradipitant, 85 mg daily, was investigated. The Gastroparesis Cardinal Symptom Index (GCSI) daily diary and additional patient-reported questionnaires were used to measure symptoms. In 46.6% of patients on Tradipitant compared to 23.5% of patients on placebo, there was a greater than 1-point improvement in the GCSI score. Additionally, there was a significant decrease in the number of nausea-free days and nausea scores, especially in patients with significant baseline nausea and vomiting scores [55].

CI, confidence interval; OR, odds ratio; RR, relative risk; US FDA, United States Federal Drug Agency.

mortality [35▪▪]. In an earlier trial, Jakob *et al.* randomized patients to a semi-elemental diet versus a standard diet for evaluating gastrointestinal tolerance of enteral nutrition in critically ill. The investigators were not able to find any variation between the two groups' diarrhoeal incidence rates [36]. Qiu *et al.* evaluated fat-modified enteral nutrition containing medium-chain triglycerides, carnitine and taurine to a standard enteral feeding in 144 ICU patients. They found that feeding intolerance was significantly less with the fat-modified diet. In the interventional feed group, the incidence of abdominal distension was 26.8%, while in the control feed group, it was 43.8% [37].

Pharmacological management

Prokinetic agents are the first line of pharmacological management of gastrointestinal dysmotility in a critically ill patient. Prokinetic therapy hastens the emptying of the stomach along with accelerating gut transit and thus improves the delivery of enteral nutrition, especially in those with gastric dysmotility and enteral feed intolerance [38]. Peng *et al.*, in a meta-analysis and systematic review of 10 RCTs with a total of 846 participants, found that prokinetics were found to have a positive effect on feeding intolerance in most trials in critically ill patients (10 of 13, 76.92%). Prokinetic drugs may shorten hospital length of stay [mean difference −3.21, 95% confidence interval (CI) −5.35 to −1.06; $P = 0.003$; low certainty] and ICU stays (MD −2.03, 95% CI −3.96 to −0.10; $P = 0.04$; low certainty) in critically ill people receiving gastric feeds. Prokinetics, however, could not improve all-cause mortality or reported adverse event outcomes [39]. ESPEN guidelines suggest that the first line of prokinetic treatment for critically ill patients with gastric feeding intolerance should be intravenous erythromycin. As an alternative, prokinetic therapy might involve intravenous metoclopramide or a combination of metoclopramide and erythromycin [26▪▪]. Pharmacological agents are discussed in Table 1.

Management of acute colonic pseudo-obstruction

American Society for Gastrointestinal Endoscopy guidelines have suggested that conservative management should be the first choice for patients without ischemia, peritonitis, significant abdominal pain, or caecal diameter less than 12 cm. It involves identifying and correcting contributing factors. For patients who are not candidates for conservative therapy, have failed it after 72 h, or are at risk for perforation, neostigmine (2 mg over 3–5 min) is

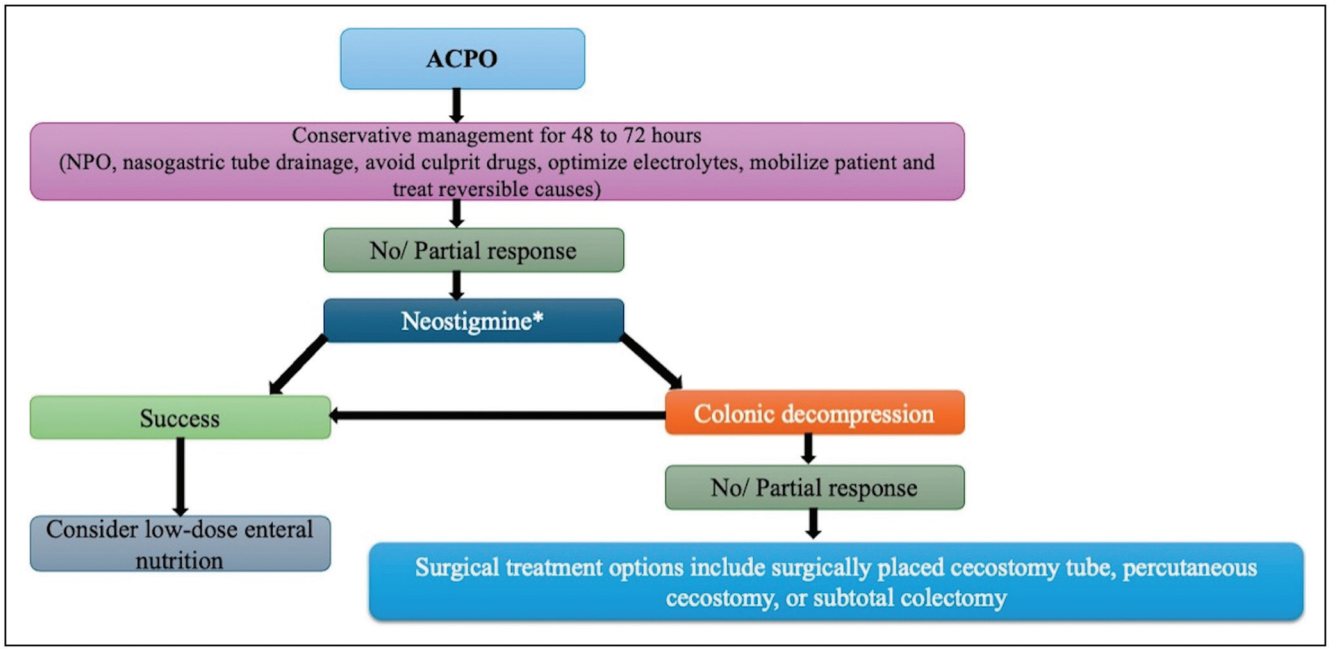

FIGURE 3. Acute colonic pseudo-obstruction management. NPO, Nil per oral. *Neostigmine should be given only if no contraindication.

recommended with cardiovascular monitoring. If the initial dose of neostigmine is ineffective, a second dose is suggested. Colonic decompression is suggested for those who are not suitable for conservative therapy or who have failed it (up to 72 h) and can undergo endoscopy, colonic decompression with a decompression tube is suggested. In cases of overt perforation or signs of peritonitis, surgical intervention is recommended [56]. Figure 3 outlines the management of ACPO.

CONCLUSION

In conclusion, gastrointestinal dysmotility is a significant concern in critically ill patients, particularly ICU patients. The implications of gastrointestinal dysmotility are profound, leading to complications, such as bacterial translocation, infections, ventilator-associated pneumonia, and malnutrition, all of which can significantly impact patient recovery and well being. Understanding the physiology and pathophysiology of gastrointestinal motility and current methods for diagnosing and treating gastrointestinal dysmotility in the ICU is critical for improving patient care and outcomes. Nonpharmacological management, including correcting electrolyte imbalances, avoiding opioid agonists and anticholinergic medications, mobilizing patients, and initiating early enteral feedings, is essential. Pharmacological management, particularly prokinetic agents, are crucial in hastening gastric emptying and improving gut transit. Overall, addressing gastrointestinal dysmotility in critically ill patients is vital for enhancing patient care and prognosis, and ongoing research and clinical practice should continue to focus on optimizing diagnostic and treatment approaches.

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Conflicts of interest

There are no conflicts of interest.

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