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Management of Gastrointestinal Failure in the Adult Critical Care Environment

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ABSTRACT

Gastrointestinal failure represents a complex syndrome with various underlying causes. Effectively managing the diverse scenarios from practical, metabolic, and nutritional perspectives poses significant challenges, which this review aims to explore. Recent developments Acute gastrointestinal injury (AGI) has been characterized and has progressed into the gastrointestinal dysfunction score (GIDS), modeled after the Sequential Organ Failure Assessment (SOFA) score, ranging from 0 (no risk) to 4 (life-threatening).

However, a specific, reliable, and reproducible biomarker associated with it is still lacking. Assessing risk using the Nutrition Risk Screening (NRS) score is the initial step in addressing nutritional therapy. Nutritional management must be tailored to the severity of gastrointestinal failure and its clinical presentations, always focusing on preventing undernutrition and dehydration, as well as providing essential micronutrients. The incorporation of fibers in enteral feeding solutions has become more accepted and is even recommended based on microbiome research. Parenteral nutrition, whether used alone or in conjunction with enteral feeding, is warranted when the intestine cannot meet nutritional needs.

Conclusion

The variability of gastrointestinal insufficiency prevents a standardized nutritional approach for all critically ill patients, underscoring the importance of early detection and the need for personalized care.

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Received: October 14, 2025; **Accepted:** October 21, 2025; **Published:** October 28, 2025**Introduction**

Most healthcare professionals concur on the necessity of providing nutrition to patients and acknowledge the heightened morbidity and mortality associated with malnutrition [1]. However, the delayed consequences of acute underfeeding complicate this responsibility, often relegating it to nursing staff, similar to the tasks of early mobilization and pressure injury prevention. Patients in the ICU typically present with acute organ failure(s) and the management thereof. While cardiac, pulmonary, and renal failures are routinely evaluated and monitored, gastrointestinal failure remains inadequately addressed and acknowledged. This critical organ system has, until now, been omitted from the evaluation criteria of multiple organ failure scores, such as the SOFA score, which only includes the liver to represent the entire gastrointestinal tract [2].

This oversight results in a deficiency of diagnostic standardization and dependable clinical indicators [2]. In contrast to the bedside echocardiography utilized for cardiac assessment, blood gas analysis for pulmonary evaluation, or diuresis/creatinine levels for renal function. Furthermore, the gastrointestinal barrier constitutes the largest surface area of the body that interfaces with the external environment serving a vital role in both physical and microbiological defenses in humans [3]. To perform its various functions, the gastrointestinal tract demands 30–40% of the body's baseline energy expenditure rendering it particularly susceptible during episodes of hemodynamic shock [4].

In the Intensive Care Unit (ICU), the occurrence of gastrointestinal failure symptoms is notably high. Critically ill patients are generally older and frequently present with multiple comorbidities, such as cancer, diabetes, and other chronic illnesses, which often lead to preadmission malnutrition commonly associated with sarcopenia.

Their primary diagnosis typically involves one or more organ failures. A majority of these critically ill patients are placed on mechanical ventilation, sedated, and administered vasoactive medications. They are also given opioid analgesics and, at times, a considerable volume of crystalloid fluids for resuscitation purposes. Each of these elements contributes to the development of gastrointestinal failure. Furthermore, critically ill patients often undergo numerous imaging or invasive procedures, resulting in repeated fasting intervals [5,6].

Although a definition for gastrointestinal failure has been established, referred to as acute gastrointestinal injury (AGI), which is graded from I (low risk) to IV (the most severe stages), it remains a polymorphic syndrome. This syndrome arises from various factors, including impaired gastrointestinal motility (such as gastroparesis or lower gastrointestinal paralysis), disorders in enterocyte function (which may involve impaired absorption mechanisms and/or compromised mucosal barrier function), disrupted bile acid homeostasis, or inadequate mesenteric perfusion. All these pathophysiological mechanisms can act as both causes and consequences of critical illness. Depending on

their intensity, these mechanisms may result in life-threatening conditions, including Ogilvie's syndrome, gastrointestinal perforation, significant gastrointestinal hemorrhage, sepsis due to bacterial translocation, abdominal compartment syndrome, and nonocclusive bowel ischemia. Despite its role in enhancing communication, the AGI score has recently been supplanted by the gastrointestinal dysfunction score (GIDS).

A further challenge is the absence of dependable biomarkers. Various options have been suggested, including plasma citrulline concentration to estimate enteric mass [7], fatty acid-binding protein, tracer glucose absorption, or paracetamol absorption to offer a semi-quantitative assessment of absorption along with a few others [2,8,9]. Gastric residual volume (GRV), the most commonly used indicator of dysfunction, is not associated with any biomarker or imaging technique (although gastrointestinal ultrasound shows promise). Therefore, there is a need for a consistent and focused clinical evaluation of a range of gastrointestinal symptoms and signs. An increasing number of identified gastrointestinal symptoms correlates with higher mortality rates [10]. This review aims to present a comprehensive overview of the management.

Management

The initial step in management is to evaluate the patient's nutritional status within the first 48 hours of admission [1]. The Nutrition Risk Screening (NRS) score is a straightforward tool that has been validated in critical care environments: scores of at least 5 points are linked to an elevated risk of mortality [11].

The tolerance for enteral feeding and the gut's ability to meet nutritional requirements remains the gold standard for monitoring gastrointestinal function in critically ill patients. The GIDS includes the absence of oral food intake as an indicator of dysfunction. Food intolerance is diagnosed through clinical examinations conducted at least twice daily, based on recurrent or severe vomiting, regurgitation, abdominal distension, absent or abnormal bowel sounds, abdominal pain, the presence or absence of stool, stoma flow, severe diarrhea, increased intra-abdominal pressure (IAP), and/or gastrointestinal bleeding. Surprisingly, these readily available variables are not consistently documented in the patient's charts, leading to their unrecognized status. The assessment primarily relies on two numerical variables: the GRV and the IAP.

Gastric Residual Volume

Measurement is conducted by suctioning a gastric tube using a syringe or by attaching a drainage bag at the stomach level, observing for a duration of 15 to 120 minutes. Ultrasound imaging is appealing for evaluating gastric overfilling, screening for free fluid, excluding distension of digestive loops, and ensuring correct positioning of the nasogastric tube [12]. This commonly utilized variable has become contentious following a large randomized trial [13]. That did not show a difference in the incidence of ventilation-associated pneumonia in patients with a protected airway; furthermore, GRV measurement was linked to a decrease in nutrition delivery [14].

Nevertheless, elevated gastric residuals suggest a heightened risk of vomiting during the initiation of enteral nutrition and/or in patients exhibiting abdominal symptoms during enteral feeding. The management of high gastric residuals includes metoclopramide, a frequently used prokinetic agent, despite its relatively low efficacy, often combined with erythromycin for a sustained effect over 48 hours [15]. These medications promote gastric emptying. Postpyloric feeding may also be recommended

in cases of confirmed gastroparesis. Proper patient positioning is crucial, as a bed angle of less than 30° is associated with increased gastric aspirates [16].

Intra Abdominal Pressure

Intra-abdominal pressure (IAP) is assessed by instilling 50 ml of water into the bladder through a urinary catheter using a syringe. It is frequently overlooked, despite its high prevalence and its grading correlates with mortality rates [17]. The World Society of the Abdominal Compartment (WSACS) has published recommendations for medical management in three steps aimed at reducing IAP [18].

The therapeutic goals include the evacuation of intraluminal contents and the removal of intra-abdominal space-occupying lesions, aimed at enhancing abdominal wall compliance and optimizing fluid administration along with systemic and regional perfusion.

Monitoring intra-abdominal pressure (IAP) during the initiation and escalation of enteral nutrition will aid clinicians in identifying abnormal increases in IAP among patients suffering from severe abdominal conditions, hypoperfusion, and fluid overload.

While a slight rise in IAP should not automatically result in the cessation of enteral nutrition, values that reach 20 mmHg should serve as a caution against the initiation or advancement of enteral nutrition, prompting considerations for either reducing or halting enteral nutrition altogether. Initially, enteral nutrition administered at 35 ml/h was paused and later resumed at 10 ml/h (trickle feeding) two hours later when IAP levels decreased, while parenteral nutrition was commenced to meet the patient's needs. Laxatives are occasionally utilized as a preventive measure to shorten the time to defecation; however, their effectiveness is limited.

How to Handle Practical Issues Abnormal Gastrointestinal Motility Constipation

Constipation occurs more often than diarrhea with its incidence ranging from 5 to 83% yet it is diagnosed less frequently [19,20]. It is characterized by the failure to pass stools for over 4 days. This condition is more prevalent in patients receiving enteral nutrition compared to those on oral feeding, and it can lead to severe complications affecting other organs, especially the lungs [16].

Constipation increases the likelihood of developing abdominal compartment syndrome. Key risk factors include dehydration, medications that slow gastrointestinal motility (such as myorelaxants, opioids, antihypertensive drugs, and iron/calcium supplements), as well as prolonged bed rest. Current management strategies involve the use of laxatives and enemas. In cases of persistent paralytic ileus, neostigmine may be administered either intravenously or subcutaneously [21].

Preventing constipation can avert organ dysfunction, as demonstrated by a randomized trial involving patients on mechanical ventilation: a notable reduction in the SOFA score was observed in those who received laxatives from the outset [22]. Dietary fiber plays a crucial role in prevention [23]. While a lack of fiber correlates with a decrease in both the number and diversity of microorganisms. Furthermore, insoluble dietary fibers enhance intestinal transit by increasing bulk, thereby reducing the necessity for laxatives. Recent reviews and meta-analyses

suggest that fibers should be incorporated into any enteral feeding regimen for the sake of gut and overall health, including in cases of inflammatory bowel disorders [24-25].

Diarrhea

Diarrhea presents a significant challenge for nurses and poses a risk to patients by jeopardizing skin integrity and potentially leading to malabsorption [26]. The prevalence of diarrhea reported ranges from 2% to 68%. Preventive measures focus on utilizing enteral feeding solutions that contain fiber [25]. The primary contributor to diarrhea is the prescription of antibiotics, which affects over 70% of patients in intensive care units [27].

The mortality rate associated with antibiotic-induced diarrhea, including cases involving Clostridium positive patients, fluctuates between 10% and 56%. The pathophysiological mechanisms underlying the various types of diarrhea such as inflammatory (including ulcerative colitis, Crohn's disease, and coeliac disease), infectious, and secretory entail numerous alterations in ion and solute transporters, along with the activation of cyclic nucleotide and Ca²⁺-signaling pathways [28].

The current approach to managing diarrhea has remained relatively unchanged for the past three decades, primarily involving the replacement of lost fluids and electrolytes through oral or intravenous rehydration, as well as the cautious administration of medications that reduce intestinal motility (μ -opioid agonists) or fluid secretion [29].

In cases of diarrhea resulting from enteric infections, various antibiotics are prescribed based on the specific pathogenic organism involved [30]. The use of antimotility medications has been restricted due to concerns regarding the risks of ileus, ischemic colitis, and bacterial overgrowth. Numerous meta-analyses and clinical studies have indicated that probiotics can help prevent or shorten the duration of diarrhea [31,32].

Fiber plays a crucial role in the management of diarrhea, especially soluble fibers such as guar gum and others. A dietary fiber intake of 15-30 g/day is recommended for both patients and healthy individuals, as it provides short-chain fatty acids to the gut mucosa, enhances the reabsorption of electrolytes and water in the colon, inhibits the proliferation of pathogenic bacteria, and normalizes transit times, with supplementation from partially hydrolyzed guar gum.

Short Bowel Syndrome

In individuals diagnosed with short bowel syndrome (SBS) who have undergone jejunostomy, it is crucial to implement nutritional therapy to avert complications linked to a high-output stoma (HOS), which is deemed clinically significant when the output surpasses 2000 ml/day [33,34]. The potential complications arising from HOS encompass dehydration, imbalances in electrolytes (specifically sodium and magnesium), and undernutrition.

Although a healthy large bowel can absorb, following an adaptive phase, up to 1000 kcal daily in SBS patients (as opposed to the typical 150 kcal per day), the majority of nutrient absorption occurs within the initial 100 cm of the jejunum. Vitamin B12 and bile salts are absorbed in the final 100 cm of the ileum, magnesium is absorbed in the terminal ileum and proximal colon, while water and sodium absorption takes place throughout the entire bowel [35].

Moreover, a multidisciplinary approach that includes psychological support is vital to ensure optimal outcomes and enhance quality of life [36,37]. HOS in short bowel syndrome may be anatomical (resulting from surgical resection that leaves less than 200 cm of proximal short bowel) or functional (such as due to intra-abdominal sepsis). The treatment strategy involves restricting oral fluid intake (isotonic beverages of 500–1000 ml/day) and providing intravenous hydration [compensating for losses with NaCl 0.9% (1:1 for outputs exceeding 1500 ml/24 h) and KCl supplementation], along with the use of antimotility agents (such as loperamide and codeine phosphate) and antisecretory medications (including proton pump inhibitors and octreotide). In all scenarios, including when surgical restoration of bowel continuity is contemplated, it is essential to address various deficiencies (such as magnesium, vitamins B12/A/D/E/K, and zinc) and to meet nutritional requirements through artificial nutrition (a combination of enteral and parenteral nutrition). If fat malabsorption, steatorrhea, or pruritic bilious output is observed, cholestyramine should be added to the regimen. The formation of calcium bilirubin ate gallstones can be mitigated by maintaining enteral feeding, minimizing periods of oral fasting, and reducing the use of narcotic and anticholinergic medications.

Abnormal Lymphatic Drainage

Chylous losses, whether thoracic or ascitic, represent a rare condition that leads to nutritional, immune, and metabolic deficiencies. Chylothorax and chylous ascites are characterized by a triglyceride concentration exceeding 120 mg/dl (1.35 mmol/l) in pleural fluid and ascitic fluid. Additionally, a cholesterol concentration below 200 mg/dl (5.2 mmol/l) in pleural fluid serves as another indicator.

The causes of chylothorax and chylous ascites include traumatic disruptions, such as those following esophagectomy or major abdominal surgery, as well as obstructive factors like tuberculosis, malignancy, or cirrhosis. These disruptions result in the leakage of thoracic or intestinal lymph into the abdominal cavity, leading to the accumulation of a milky fluid that is rich in triglycerides [38,39]. The medical management of chylous ascites focuses on nutritional strategies aimed at reducing chyle flow, which may facilitate the spontaneous closure of the fistula.

The recommended dietary approach includes high-protein and low-fat nutrition, incorporating medium-chain triglycerides (MCTs) to minimize chyle production. In some cases, total parenteral nutrition with MCT may be necessary. Other treatment options, such as therapeutic paracentesis, somatostatin, and octreotide, are not covered in this text [40].

When to Rest and When to Feed

Most contraindications to enteral feeding have become relative, except in cases where there is a complete cessation of gastrointestinal function. However, the use of enteral nutrition necessitates careful monitoring. Indeed, aside from complete cessation conditions, critically ill patients may experience varying degrees of gastrointestinal dysfunction (GID), as evidenced by a recent large observational study [2].

Total Stop

In situations involving gut obstruction, perforation, or bowel ischemia, the standard protocol is nil per so, which requires no further explanation other than the initiation of parenteral nutrition until the issue is resolved.

Bowel Necrosis

Bowel necrosis can infrequently arise in the context of jejunal feeding [41]. It may manifest in patients who do not exhibit risk factors for enteric ischemia. Those receiving enteral nutrition may experience nonspecific symptoms, severe shock, and ultimately multiorgan failure: these individuals are candidates for immediate enteral resection and a period of bowel rest lasting a few days.

Acute Mesenteric Ischemia

Acute mesenteric ischemia (AMI) represents a potentially fatal condition that typically necessitates ICU admission: during this episode, enteral nutrition must be entirely halted until the affected vessels are recanalized.

A randomized trial involving 183 AMI patients compared the reintroduction of enteral nutrition to parenteral nutrition within the first week following revascularization, revealing several benefits associated with the enteral nutrition approach [42]. Including a significantly reduced need for prolonged parenteral nutrition, fewer infections, decreased respiratory complications, and a quicker restoration of bowel continuity in cases requiring resection.

Relative Stop

Inflammatory Bowel Disease

Bowel rest is a longstanding concept dating back to the 1980s. It is theoretically appealing, as one might anticipate that an inflamed intestine would heal more effectively if relieved of mechanical stress, intestinal secretions, and the antigenic challenge posed by food. A randomized trial involving 47 patients with severe acute colitis demonstrated that bowel rest, when compared to oral feeding, resulted only in a decrease in stool weight [43]. Without affecting surgical needs or other outcome measures. In the most severe cases of Crohn's disease, bowel rest continues to be utilized in instances of exacerbated painful symptoms.

Severe Diarrhea

Severe diarrhea (such as that caused by *C. difficile*) is another manifestation of inflammatory bowel disease, which may necessitate bowel rest, along with a combination of parenteral and enteral nutrition.

Chylous Leaks

Chylous leaks necessitate the temporary cessation of enteral fat intake, eventually transitioning to parenteral nutrition [44]. Medium-chain triglycerides (MCT) are utilized as an energy source in conjunction with amino acids and glucose. A gradual reintroduction of enteral feeding is implemented through a semi-elemental diet that includes MCT. An oral diet is reinstated once this approach has significantly diminished chyle production.

No Restriction

In the context of enhanced recovery after surgery (ERAS), bowel rest plays a minimal role in the management of surgical patients: rather, there is a strong recommendation to initiate gut feeding as soon as feasible [45,46]. Employing oral nutrition supplements for several days may complement this approach [47]. The same prompt initiation is advised for any intubated medical or surgical patient [1].

Nutritional Options

Enteral and/or Parenteral?

Recent large-scale randomized trials have demonstrated that there is a balance between enteral nutrition and parenteral nutrition when similar energy targets and rapid feeding progression are employed,

even in cases of septic shock [48,49]. The ESPEN-ICU guidelines emphasize that the enteral route should be attempted first to achieve the nonnutritional advantages of gut feeding: ultimately, the route is of little significance, and any method should be utilized to prevent acquired malnutrition. The nonnutritional advantages [1]. Encompass supporting mucosal health (by enhancing blood flow, releasing trophic agents such as cholecystokinin and bile salts), boosting systemic immunity by stimulating the gastrointestinal and mucosal-associated lymphoid tissue (GALT and MALT) [50], nourishing the microbiota, and being more economical than parenteral nutrition. However, since gastrointestinal dysfunction is common and leads to inadequate feeding progression and, ultimately, malnutrition, the delivery should be closely monitored. Patients with gastrointestinal dysfunction are often ideal candidates for supplemental parenteral nutrition to prevent exacerbating the energy and protein deficit [51].

Micronutrients (Vitamins and Trace Elements)

The absorption of micronutrients is hindered in cases of gastrointestinal dysfunction, leading to uncertainty regarding the bioavailability of micronutrients delivered enterally. This uncertainty is particularly pronounced during the initial phase of acute illness and impacts all types of micronutrients.

Consequently, since numerous acute hospital admissions are linked to previous inadequate nutritional intake, it is reasonable to implement a strategy that provides a combination of moderate doses of micronutrients via the intravenous route, alongside thiamine. In chronic intestinal disorders, the levels of various micronutrients are often diminished. It is essential to actively monitor for deficiencies in vitamins B7, B12, D, as well as trace elements Cu and Fe, utilizing blood tests for evaluation [52-54]. In cases of inflammatory bowel diseases, deficiencies in vitamins B12, A, D, E, K, and trace elements such as Se and Zn have frequently been documented [55]. Following surgical bypass of the duodenum, such as in bariatric procedures, lifelong therapeutic administration of vitamin B12 may be necessary due to malabsorption issues [56]. For patients suffering from prolonged intestinal disorders, it is crucial to systematically identify and address the aforementioned micronutrient deficiencies.

Conclusion

Each form of gastrointestinal dysfunction or failure necessitates tailored management; however, all cases require vigilance regarding the potential onset of malnutrition, which can be monitored through actual nutritional intake. In the context of the balance between enteral and parenteral nutrition each with its own benefits and complications evaluating the nutritional status and gastrointestinal function upon admission is a critical initial step in determining the appropriate strategy.

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