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What is This?
Feeding the Hemodynamically Unstable Patient: A Critical Evaluation of the Evidence

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Early administration of enteral nutrients has been shown to improve outcome in acutely ill patients.1,2 Thus, early enteral feeding has become an integral component of the care of acutely ill patients. Many critically ill patients are hemodynamically unstable or need vasopressors/inotropes to maintain adequate blood pressure and cardiac output. Although many clinicians use enteral feeding in these patients, others feel that enteral feeding is contraindicated in this patient group. In healthy adults, enteral nutrients usually produce an increase in gut blood flow. However, in patients with low flow states and shock, the effect of enteral nutrients on gut blood flow is less clear. The presence of luminal nutrients may increase oxygen demand beyond that which can be satisfied by the available delivery, potentially leading to gut ischemia. The objective of this review is to critically evaluate the evidence for and against enteral feeding in hemodynamically unstable patients and in patients requiring vasopressor support.

Benefits of Early Enteral Feeding

Enteral infusion of nutrients prevents adverse structural and functional alterations of the gut barrier induced by injury, increases epithelial proliferation, maintains mucosal integrity, decreases gut permeability, improves gut blood flow, and improves local and systemic immune responsiveness.3–9 There is an increase in immunoglobulin A secretion, maintenance of the gut associated lymphoid tissue, improved mucous secretion, decreased bacterial translocation, increased trophic hormone secretion, and decreased infection. Apoptosis occurs in many clinical conditions that include ischemia-reperfusion injury. Early enteral feeding may also decrease apoptosis in the gut.9

Hyperemic Response to Enteral Nutrients

Enteral nutrients are important for maintenance of normal gastrointestinal tract structure and function. Luminal nutrients are trophic to the gut and important for maintenance of the villus structure, absorption, the gut barrier, and immune function. These effects are mediated via both direct and indirect (ie, hormonal) effects. Enteral nutrients also augment blood flow to the gastrointestinal tract, primarily via locally mediated vasodilation. Nutrient augmentation of blood flow aids oxygen delivery to the gut and absorption of nutrients after feeding. In contrast, gut blood flow is diminished during fasting or starvation. The effect of luminal nutrients on gut blood flow is known as the postprandial hyperemic response.10–17 Importantly, flow to the intestine after nutrient administration increases in all layers, including the mucosa, submucosa, and muscularis.17

The major dietary components responsible for mesenteric vasodilation during feeding are dietary peptides, simple carbohydrates, and long-chain fatty acids.15,16,18 High-fat meals cause a greater increase in intestinal blood flow than carbohydrate meals, and complex nutrient solutions containing protein, carbohydrate, and fat induce larger hyperemic responses than solutions containing the individual constituents.18 Arginine does not seem to vasodilate normal gut. However, arginine has been shown to increase gut blood flow during sepsis, presumably via conversion to nitric oxide (NO).19

Experimental Studies of Gut Oxygen Supply and Demand

Absorption of nutrients from the gut requires oxygen. Normally, nutrients vasodilate mesenteric blood vessels and increase oxygen supply. Under normal circumstances, oxygen supply is more than adequate to meet the increased demands of absorption. However, there is concern that demand may
outstrip supply in hemodynamically unstable patients and in patients requiring vasopressors or inotropes.

Gut blood flow is reduced in patients after various forms of critical illness, including sepsis, hemorrhage, multitrauma, cardiogenic shock, and burn injury. Importantly, gut blood flow remains depressed despite fluid resuscitation and return of blood pressure and cardiac output to normal values, indicating that factors other than intravascular volume and cardiac output determine local tissue blood flow. Decreased gut blood flow is associated with ischemic injury, bacterial translocation and multiple-organ failure. Gianotti et al \(^{20}\) studied intestinal blood flow and fungal translocation and reported that translocation was greatest in villi with intestinal blood flow and fungal translocation and improved survival after oral glutamine administration in a model of sepsis. Decreased bacterial translocation and improved survival in experimental sepsis with the use of enteral diets has been reported by a number of investigators.\(^ {27}\)

Purcell et al \(^ {28}\) evaluated the effect of enteral feeding on gut blood flow in animals after lung injury and ventilation with positive end expiratory pressure (PEEP). Lung injury was found to decrease cardiac output and portal vein and hepatic artery blood flow. Feeding reversed the decrease in portal vein and hepatic artery blood flow. Interestingly, ventilation with PEEP in lung injury was associated with a decrease in gut oxygen delivery but no change in oxygen consumption. Although feeding increased gut oxygen consumption, the concomitant increase in oxygen delivery resulted in a better oxygen delivery:consumption ratio in the fed \textit{versus} nonfed state. Purcell\(^ {28}\) and other investigators have found that gut blood flow increases within minutes of nutrient administration and needs relatively small volumes to produce the effect.

Kazamias et al \(^ {24}\) studied the effect of enteral nutrients on hepatic oxygenation and bioenergy status in a model of endotoxic shock. Dogs were administered endotoxin into the portal vein and randomized to enteral or placebo feeding via jejunostomy. Systemic blood pressure and gut blood flow declined after endotoxin. Enteral feeding improved hepatic artery and portal vein blood flow, superior mesenteric artery blood flow, intestinal mucosal microcirculatory flow, hepatic microcirculatory flow, hepatic and intestinal tissue oxygenation, and hepatic energy stores. Superior mesenteric artery blood flow and mucosal microcirculatory flow in the fed animals exceeded baseline levels. The investigators concluded that enteral nutrition reversed endotoxin-induced splanchnic ischemia. Gosche et al \(^ {25}\) in a model of \textit{Escherichia coli} sepsis, found a decrease in intestinal microcirculatory blood flow during bacteremia, which was restored to above-baseline values after glucose suffusion. In addition, Gianotti et al \(^ {26}\) reported decreased bacterial translocation and improved survival after oral glutamine administration in a model of sepsis. Decreased bacterial translocation and improved liver function\(^ {22}\) and survival in animals enteral feeding during hemorrhagic shock on sur-
Enteral feeding likely produces beneficial effects on the gut through a variety of mechanisms. It improves blood flow and tissue oxygenation, supplies substrate for energy and tissue repair, provides substrate for immune function, provides substrate for generation of vasodilating substances such as NO, regulates expression of genes involved in the proinflammatory response and endothelial activation, and provides antioxidant substances.

**Vasopressors**

Alterations in gut blood flow vary with the vasactive agent used and the underlying disease (ie, sepsis, hemorrhagic shock, cardiogenic shock). Thus, the effects of individual vasopressor/inotropic agents cannot be generalized and must be considered in the context of each agent and the specific disease process. Furthermore, the effect of a vasactive agent on total splanchnic blood flow may not parallel the effect on blood flow to the bowel and the distribution of flow within the layers of the bowel. For example, an agent may increase total splanchnic blood flow yet shunt blood away from the intestinal mucosa, causing mucosal ischemia. In addition, a combination of vasactive agents may result in alterations in splanchnic blood flow that may not be predicted from the effects of each agent alone. A full discussion of the effects of vasactive agents on the gut is beyond the scope of this review. In general, experimental and clinical studies demonstrate that dopamine increases gastrointestinal mucosal blood flow and gastric intramucosal pH (pHi), a surrogate marker of mucosal perfusion. Although dopamine agonists may increase splanchnic mucosal blood flow in healthy individuals, this effect has not been demonstrated in states of hemodynamic compromise. In patients with septic shock, dopamine decreases gastric pHi, whereas norepinephrine increases pHi. Neviere and colleagues assessed the effects of dopamine and dobutamine on splanchnic mucosal blood flow in septic patients by using gastric tonometry and laser Doppler flowmetry. Gastric mucosal blood flow increased and pHi increased with dobutamine, whereas the opposite effects were noted with dopamine. In a group of septic patients, Olsen and coworkers demonstrated that, although dopamine increased oxygen delivery, it did not increase pHi. Similarly, Meier-Hellman and coworkers demonstrated that dopexamine (a dopamine and β-2 adrenergic agonist) caused a dose-dependent reduction in pHi despite an increase in splanchnic oxygen delivery. In a porcine hemorrhagic shock model, Segal and colleagues demonstrated that treatment with low-dose dopamine hastened the development of gut ischemia. It is postulated that dopamine causes precapillary vasoconstriction with diversion of blood flow away from the gut mucosa. Norepinephrine, however, may increase splanchnic perfusion in patients with sepsis. In a volume-resuscitated endotoxic shock model, Treggiari and coworkers demonstrated that norepinephrine increased portal venous blood flow and jejunal mucosal blood flow as assessed by laser Doppler flowmetry. Norepinephrine will, however, decrease mucosal blood flow during hypovolemic states. Epinephrine has been reported to decrease splanchnic blood flow.

Recently, vasopressin has gained popularity as a "vasopressor" agent in patients with septic shock. Vasopressin is normally involved in the regulation of plasma osmolarity. However, plasma concentrations of vasopressin are increased dramatically in hypovolemic and cardiogenic shock, and it seems that endogenous vasopressin is important in maintaining blood pressure in these conditions via its vasoconstricting action. Low levels of circulating vasopressin have been reported in patients with septic shock. Vasopressin is an intriguing vasopressor because it has little pressor activity in normal subjects in low doses (ie, 0.04 units/min) but markedly increases arterial pressure in septic patients with intractable hypotension. In addition, vasopressin enhances the pressor response to catecholamines. Experimental studies have demonstrated that vasopressin causes intestinal vasoconstriction that may enhance the gastrointestinal mucosal injury characteristic of sepsis. Furthermore, emerging data suggest that low-dose vasopressin may cause severe gastric mucosal acidosis in patients with sepsis.

In hypovolemic, cardiogenic, and obstructive shock, vasopressor agents increase blood pressure by shunting blood from the gut and other peripheral organs (ie, bone marrow, skin, kidneys) to the central circulation. These "nonessential" organs are more sensitive to vasoconstriction than are central "essential" organs (ie, heart, brain). Thus, the effect of vasoconstrictor medications and hypotension in these forms of shock is to decrease gut blood flow. Blood flow to the gut after various forms of shock is not immediately reversed with volume resuscitation, perhaps because of increased levels of endogenous or exogenous vasoconstrictor agents (ie, norepinephrine, endothelin I, angiotensin II, vasopressin) or a decrease in endogenous vasodilators (ie, NO), or because of an ischemia-reperfusion injury to the microcirculation.

Because nutrients vasodilate mesenteric blood vessels, they may help maintain gut blood flow in patients who are hemodynamically unstable or receiving vasopressors (ie, gut resuscitation). We first addressed the interaction between enteral nutrients and vasopressors on gut blood flow by studying the effect of enteral feeding during high-dose phenylephrine (an α-adrenergic agonist) administration in a rat model. Ultrasonic flow probes were placed on the superior mesenteric artery and animals randomized to receive enteral formula or IV fluids. Blood flow was measured, and animals were infused with increasing doses of phenylephrine. Phenylephrine decreased mesenteric...
blood flow by 52% in animals not receiving enteral feeding, whereas blood flow decreased only 28% in enterally fed animals (Fig. 1). In similar experiments, we subsequently found that enteral nutrient administration improved mesenteric blood flow and renal blood flow during infusion of vasopressin.\(^49,50\) Figure 2 shows the significant increase in mesenteric blood flow that occurred in animals receiving both enteral nutrition and vasopressin at low and moderate doses. It is notable that at the highest dose of vasopressin studied, the nutrient benefit was not maintained. Doses of vasopressin studied caused increases in mean arterial pressure of 15%, 25%, and 35%, respectively. We later found that carnosine, a dietary peptide with inotropic and vasodilatory effects, also improved mesenteric blood flow during vasopressin administration.\(^51\) None of the animals in any of these studies developed intestinal infarction. Overall, these studies indicate that enteral nutrients improve gut blood flow, especially during vasopressor administration.

**NO and the Gut**

A number of the inflammatory mediators released during sepsis initiate the transcription and translation of inducible NO synthase, resulting in the generation of NO from arginine. NO binds to heme-containing proteins such as guanylate cyclase, which increases the synthesis of guanosine 3',5'-cyclic monophosphate (cyclic GMP).\(^52,53\) Cyclic GMP mediates a variety of processes that include smooth muscle relaxation (ie, vasodilation) and inhibition of platelet aggregation. The fall in mean arterial pressure and fall in systemic vascular resistance in sepsis is partially mediated via the release of NO.\(^54\) Nitric oxide synthase can be blocked by arginine analogs\(^55\) and methylene blue.\(^56\) These agents have been shown to increase blood pressure in patients with sepsis.\(^57–60\)

On the other hand, NO is important for maintenance of microvascular blood flow in patients with sepsis.\(^61\) A number of sepsis models have demonstrated that NO synthase inhibition decreases microvascular flow and increases tissue injury in organs such as the intestines, liver, and kidneys,\(^19,62–64\) suggesting that intestinal production of NO is important for maintaining gut blood flow during periods of hemodynamic instability. Arginine has been shown to increase gut blood flow during sepsis, presumably via conversion to NO.\(^19\)

**Immune-Enhancing Diets**

Blood flow to the gut is affected by the content of the diet. Dietary peptides, long-chain fatty acids, and simple carbohydrates produce maximal vasodilation (see section on the Hyperemic Response to Enteral Nutrients). A variety of available enteral formulas are augmented in nutrients aimed at improving immune function. Such nutrients include arginine, glutamine, \(\omega-3\) polyunsaturated long-chain fatty acids, and antioxidants. Interestingly, immune-enhancing diets have been reported to increase splanchnic blood flow to a greater degree than standard nutritional formulas.\(^65,66\)

**Postoperative Enteral Nutrition**

Immediate enteral feeding through gastric and small intestinal feeding tubes is routinely used to treat postoperative patients in many clinical centers. In our systematic review of early enteral feeding in patients after abdominal surgery,\(^1\) bowel infarction did not occur. On the other hand, early...
Enteral feeding was associated with decreased infections and length of stay. In a study of early enteral feeding in patients after gastrointestinal surgery by Lewis et al., bowel infarction did not occur. Interestingly, the early-fed group had improved anastomotic healing, decreased infections, and decreased mortality.

Clinical studies indicate that early enteral feeding is safe in patients after gastrointestinal surgery and liver transplantation. For example, Braga et al. evaluated enteral compared with parenteral nutrition support in 257 postoperative patients. Patients underwent surgery on the stomach, pancreas, and esophagus. Enteral feeding was associated with less hyperglycemia, improved electrolyte balance, improved bowel function, better gut oxygenation, and decreased nutritional costs. Overall, groups were similar for complications, length of hospital stay, and mortality. However, in the subgroup of malnourished patients, there was a tendency toward lower complications and significantly shorter hospital stay with enteral compared with parenteral nutrients. The majority of studies that compare enteral to parenteral nutrition in postoperative patients demonstrate decreased infections in the enteral groups but similar length of stay and mortality. Importantly, there does not seem to be any increase in complications.

Revelly et al. evaluated enteral nutrition in 9 patients requiring hemodynamic support with catecholamines 1 day after cardiac surgery. Patients were fed with postpyloric feeding tubes. During enteral feeding, cardiac index increased (from 2.9 to 3.3 L/min/m²), mean arterial pressure decreased (78 to 70 mm Hg), indocyanine green clearance increased (527 to 690 mL/min), and gastric tonometry remained unchanged. There were increases in glucose level, insulin level, and glucose rate of appearance. The investigators concluded that enteral feeding increased cardiac index and splanchnic blood flow in patients requiring catecholamine support after cardiac surgery. Berger et al. studied intestinal absorption in hemodynamically stable and unstable patients (needing high-dose inotropes and vasopressors with or without an intra-aortic balloon pump) after cardiac surgery. Absorption of paracetamol was reduced on day 1 in all patients after gastric administration but not with postpyloric delivery. Absorption on day 3 was near normal in all patients. Absorption was normal in patients with low cardiac outputs receiving postpyloric nutrition. Enteral nutrition was well tolerated in all patients.

Enteral Nutrition in the Trauma or Burn Patient

Reduced gut blood flow is common after traumatic shock. Yet, enteral nutrition support has been shown to reduce septic morbidity after major trauma. Moore et al. randomized 75 trauma patients undergoing emergent celiotomy with an abdominal trauma index >15 to a control group or enteral-fed group (using a needle catheter jejunostomy). Nitrogen balance was improved in the early enteraly fed patients. In addition, septic morbidity was lower in the enteral-fed group. In a meta-analysis of trauma patients, early enteral feeding was safe and associated with reduced septic complications compared with patients fed via the IV route. Kompan et al. randomized multiple trauma patients with shock to early enteral feeding or delayed enteral feeding. After 6 hours of resuscitation, the early group began receiving enteral nutrition. The lactulose/mannitol test was used to assess gut permeability. Patients started on early enteral feeding demonstrated decreased permeability and less severe organ failure. However, in this small study, there was no difference between groups for length of intensive care unit stay or time of mechanical ventilation. In our review of early feeding in trauma patients, enteral feeding was well tolerated and associated with decreased infections and length of stay.

Normal healthy adult intestinal function in the fasting state is characterized by the cyclic occurrence of a migrating motility complex (MMC). The presence of this complex requires intact intrinsic neural and muscle function. To investigate the presence of MMCs in trauma patients, Moore et al. placed multisensor manometry catheters into 10 trauma patients who presented in shock and who had undergone laparotomy. All patients were fed through a needle catheter jejunostomy. The presence of MMCs before feeding predicted better tolerance to feeding. Unfortunately, few objective data on tolerance were provided. In a study of 12 critically ill mechanically ventilated patients (half with trauma), Dive et al. reported decreased gastric contractions but preserved duodenal MMC activity. They did not evaluate tolerance of small bowel feeding. It remains unclear whether this test is a good predictor of patients at risk for bowel necrosis. However, this may be a good test for future studies of feeding tolerance and patients at risk for bowel compromise.

Early enteral nutrition also improved outcome in burn patients. Interestingly, Jenkins et al. fed patients with burn injuries continuously, even during surgical procedures, and reported decreased infections in the fed versus a nonfed group.

Early Enteral Feeding of Medical Patients

Numerous studies indicate that bacteremia/sepsis causes mucosal arteriolar vasoconstriction and microvascular hypoperfusion of the intestine, despite vasodilation of systemic vessels. Diminished production of NO and an excess production of angiotensin II, endothelin I, and increased sympathetic tone have been implicated as a cause for the vasoconstriction. Rank et al. studied gut perfusion in 60 septic patients within 24 hours of shock onset. After resuscitation with fluids and inotropes, subjects were randomized to receive N-acetylcysteine (NAC).
or placebo. NAC improved heptosplanchnic flow and function. Unfortunately, these investigators administered the NAC IV. Thus the effect of enteral NAC on gut blood flow remains unknown. However, the study suggests that administration of some nutrients, especially nutrients with antioxidant and NO stimulating properties, may improve gut blood flow during sepsis.

Most studies of early enteral feeding have been performed in surgical patients. Nevertheless, studies performed in critically ill patients have indicated that early enteral feeding is safe. For example, Galban et al reported early enteral feeding in 176 septic critically ill patients. Sepsis is a disease in which gastrointestinal blood flow is reported to be diminished. Enteral feeding was initiated within 24 hours of sepsis. There were no reports of bowel ischemia or infarction, and the formulas were reported to be well tolerated. Gadek et al evaluated early enteral feeding in 146 patients with acute respiratory distress syndrome (ARDS). Most patients were medical patients and began receiving enteral nutrition within 24 hours of the diagnosis of ARDS. Enteral feeding was well tolerated, and there were no reports of bowel ischemia or infarction. Caparros et al evaluated early enteral feeding in 220 critically ill patients. Approximately 50% of the patients were medical, 10% surgical, and 40% trauma. Approximately 13% had sepsis, 6% septic shock, and 13% ARDS on entry into the study. Ninety-seven percent were receiving mechanical ventilation. Although gastrointestinal intolerance (ie, diarrhea, elevated gastric residual volume, abdominal distention) was common, none of the patients were reported to develop bowel ischemia or necrosis. The authors concluded that enteral nutrition was well tolerated. Atkinson et al evaluated early enteral feeding in 390 critically ill patients. Seventy percent of patients were medical. There were no reports of bowel ischemia or necrosis.

**Enteral Feeding–Associated Bowel Necrosis**

Early enteral administration of nutrients to critically ill patients is commonplace. Rarely do these patients develop bowel necrosis. Myers et al reviewed 16 years’ experience with needle catheter jejunostomies (NCJs) for enteral feeding in surgical patients. Of 938 patients who received NCJs, bowel necrosis developed in only 3 patients (0.15%). Two of the patients were septic with recurrent hypotensive episodes, and 1 was receiving high-dose vasopressin for variceal bleeding. Eddy et al reported on 122 NCJs placed in trauma patients. None of the patients developed bowel necrosis. Schunn et al reviewed 1359 patients receiving jejunal tube feeding. Small bowel necrosis was rare and developed in only 4 (0.2%) patients. All were abdominal surgical patients with multiple complications. Smith-Choban et al reported 5 of 143 (3.4%) bowel necroses in surgical patients receiving NCJs. Marvin et al reported 13 of 4311 (0.3%) bowel necroses in trauma patients. Twelve were being enterally fed before diagnosis, 4 of 13 were receiving vasopressors, 8 of 13 had closed head injuries, 11 of 13 were receiving inotropes (dopamine or dobutamine), and all were receiving mechanical ventilation. Lawlor et al reported small bowel necrosis in 3 of 386 (0.7%) surgical patients being fed with jejunal tubes. Holmes and colleagues reported small bowel necrosis in 3 of 222 (1.4%) trauma patients receiving jejunal feedings. In our experience with early enteral feeding of >10,000 postoperative patients, we have noted small bowel necrosis in only a handful of patients. In approximately half of these patients, necrosis was related to placement of a feeding tube through the bowel wall (which may have compromised local blood flow). Thus, feeding-associated bowel necrosis is a rare but potentially lethal event.

We have extensively reviewed the literature related to enteral feeding–associated bowel necrosis. The cause for bowel necrosis in most patients receiving enteral feeding is unclear and the pathophysiology seems complex. However, a number of relationships between feeding and bowel necrosis can be extracted from the literature. Virtually all patients were surgical patients, and bowel necrosis occurred in the postlaparotomy period. Patients underwent bowel manipulation and received feeding with NCJs or Witzel tube jejunostomies. Bowel necrosis was higher with Witzel tube jejunostomies than NCJs. Feeding-associated bowel necrosis is extremely rare in patients receiving gastric feeding. Interestingly, we found 1 report of bowel necrosis occurring in a patient who did not undergo abdominal surgery. The patient was a multiple trauma patient with closed head injury who was being fed via the gastric route but received high-dose clonidine for alcohol withdrawal. Clonidine can impair gut motility and blood flow and was felt to be the cause of the bowel necrosis in this patient. Bowel necrosis was reported in 10 of 2114 (0.5%) burn patients. However, the relationship to enteral feeding was not clear in the report. We found no reports of feeding-associated bowel necrosis in medical critically ill patients. Occlusive vascular events were absent in most reports. Hypotension and use of vasopressors were rarely reported in patients before bowel necrosis. Low-flow states were rare and most patients were hemodynamically stable before diagnosis of bowel necrosis. Bowel necrosis is also extremely rare in unfed patients who survive resuscitation from hemorrhagic shock, suggesting that hypotension alone is not a primary cause. Hypotension occurred in most patients after they developed bowel necrosis rather than before and appears to be a consequence rather than a primary cause of feeding-associated bowel necrosis. Feeding-associated bowel necrosis does not seem to be directly related to early aggressive enteral nutrition support in incompletely resuscitated patients.
Most cases occurred in patients fed enterally in a delayed fashion (ie, 5 to 10 days after injury) and occurred in patients who were hemodynamically stable and not receiving vasopressors and inotropes. Feeding-associated bowel necrosis rarely occurred with early enteral feeding. In a retrospective review, we found a similar incidence of bowel infarction in patients who were fed IV and those fed enterally. Thus, the relationship of feeding to bowel necrosis is unclear. Some cases may result from the underlying injury and not the enteral feeding. Hypotheses for feeding-induced bowel necrosis include increased energy demands in a compromised gut, imbalance between oxygen demand and supply, hypersensitivity to vasopressors such as angiotensin II and vasopressin, induction by endotoxin and other enterotoxins, toxic effects of specific nutrients, use of vasoconstrictors that limit gut perfusion, bacterial overgrowth, and progressive gut distention that limits blood supply.

Feeding-associated bowel necrosis presents as abdominal pain, abdominal distention, nausea or vomiting, high gastric output, ileus, fever, leukocytosis, sepsis, hypotension, and shock. Many believe that it is the bowel distention that impairs gut blood flow, resulting in ischemia and infarction. Thus, feeding should be immediately discontinued in patients who present with the above findings until the cause can be investigated and treated. Overall, small bowel necrosis is very rare in patients who are initiated on early enteral feeding. Most cases result from mechanical factors, and there is no clear evidence that early feeding is the causative agent. Nevertheless, enteral feeding should be discontinued in patients who develop hypotension and abdominal complaints such as pain, distention, and ileus until the status of bowel integrity can be evaluated. Although no procedure, however simple, is without risk, we believe that the benefits of early enteral feeding greatly outweigh the risks. However, it is essential that the clinician be aware of the complications of enteral feeding and react appropriately to them.

**CONCLUSION**

The available data indicate that enteral nutrition restores splanchic perfusion and oxygenation in the hemodynamically unstable patient and in subjects receiving vasopressor/inotrope support. Despite this evidence, we believe it prudent to delay initiation of feeding until the patient has been fluid resuscitated and has an adequate perfusion pressure. This goal should be obtained within 6 hours of hospitalization in most patients. Once the patient meets these criteria, we advocate the initiation of enteral feeding at low rates (ie, 25 to 30 mL/h) even if the patient requires continued vasopressor/inotropic support. If the patient suddenly develops hypotension or signs of abdominal distention, we advocate withholding enteral feeding until intravascular volume and perfusion pressure are reestablished and intestinal integrity is ensured. This approach does not apply to the patient with obstructive splanchnic vascular disease in whom enteral feeding may cause further bowel ischemia.

**REFERENCES**