Enteral Nutrition in the Critically Ill: Myths and Misconceptions

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Background: Nutritional support is an essential component of the management of critically ill and injured ICU patients. Optimal provision of calories and protein has been demonstrated to reduce morbidity, mortally, and length of ICU and hospital stay. Yet, a large proportion of ICU patients receive inadequate nutrition.
Objective: To provide an evidence-base assessment of factors leading to inadequate enteral nutrition support in critically ill patients.
Data Source: Search of PubMed database and manual review of bibliographies from selected articles.
Data Synthesis and Conclusions: A number of common myths and misconceptions appear to play a major role in limiting the provision of enteral nutrition in the critically ill. This article provides scientific data to debunk the most common myths and misconceptions related to enteral nutrition. (*Crit Care Med* 2014; 42:962–969)
Key Words: bowel sounds; enteral nutrition; ileus; myths; parenteral nutrition; surgery

Over the past three decades, the understanding of the molecular and biological effects of nutrients in maintaining homeostasis in the critically ill population has made exponential advances. Nutrition support is now considered an essential component of the management strategy of critically ill patients in the ICU (1–3). It has been well established that delivering early enteral nutritional (EN) support reduces disease severity, diminishes complications, decreases length of stay in the ICU, and favorably impacts patient outcome (1–10). Yet, a large proportion of ICU patients receive inadequate nutritional support (11–15). This appears to be driven by a number of widespread myths and misconceptions (11–14, 16). This article reviews the most common misconceptions with the goal of optimizing nutritional support in critically ill patients.

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MYTH NO. 1: STARVATION OR UNDERNUTRITION IS "OKAY"

Nutrition is essential for survival. Adequate nutrition is essential for the critically ill patient to support anabolism, ameliorate uncontrolled catabolism, maintain a competent immune system, and ultimately improve patient outcome. Nutrition support attenuates the metabolic response to stress, limits oxidative cellular injury, and favorably modulates the immune response (17–21). In a large observational study conducted in 167 ICUs, Alberda et al (15) demonstrated a strong association between the reduced provision of energy and protein and worse outcomes. Weijs et al (22) demonstrated that optimal nutritional therapy (calories and protein) in mechanically ventilated critically ill patients was associated with a 50% decrease in 28-day mortality. Similarly, Allingstrup et al (23) demonstrated that in severely ill ICU patients, a higher provision of protein was associated with a lower mortality. The energy deficit accumulated by underfeeding patients during their ICU stay has been shown to be an important factor in increasing the risk of adverse outcomes (24–26). It, however, needs to be recognized that these are observational studies that may be confounded by severity of illness; less sick patients who tolerate EN better are more adequately fed and have better outcomes. Despite this limitation, the Society of Critical Care Medicine (SCCM) and American Society of Parenteral and Enteral Nutrition (ASPEN) guidelines (1), as well as the Canadian (3, 27) and European guidelines (2), all recommend that EN be initiated within 48 hours in the critically ill patient who is unable to maintain volitional intake. It is important to emphasize that there is no known illness or disease that has been demonstrated to benefit from starvation. However, it is not uncommon for critically ill patients to be starved. In the SepNet point prevalence study conducted in Germany, 10% of ICU patients with sepsis received no nutritional support, whereas only 20% received EN exclusively (11).

The role of an initial strategy of trophic feeding (or permissive underfeeding) in the critically ill patient is controversial. The EDEN study randomized patients (n = 1,000) with acute lung injury to receive either trophic (20 kcal/hr) or full feeding (25–30 kcal/kg/d) for the first 6 days (28). After day 6, all patients who were still receiving mechanical ventilation received the full feeding protocol. There was no difference in

the number of ventilator-free days (primary outcome), 60-day mortality, and other secondary endpoints between groups. Follow-up of these patients showed no difference in physical function (as assessed by the SF-36 questionnaire), psychological and cognitive function, and quality of life at 12 months (29). The patients enrolled in the EDEN trial were "well-nourished" overweight patients (mean body mass index, 30 kg/m^2) (28). Moderate obesity has been shown to be protective during critical illness, the so-called obesity paradox (30). The findings of the EDEN study may therefore not be generalizable to critically ill patients who are malnourished. The results of the EDEN trial are similar to a smaller study ($n = 200$) conducted by Rice et al with a similar study design and patient population to the EDEN trial (31). In the absence of a proven benefit of permissive underfeeding, critically ill patients should receive 20-25 kcal/kg/d and 1g/kg/d protein. The benefits of higher quantities of protein are controversial, with the SCCM/ASPEN guidelines recommending 1.2–2.0 g/kg/d (1), with some authorities recommending up to 2.5 g/kg/d (32). However, increased provision of protein has not been demonstrated to limit muscle wasting, loss of lean body mass, or improve clinical outcomes.

"Bowel rest" was popularized in the 1970s for the treatment of active Crohn disease, colitis, acute and chronic pancreatitis, diverticulitis, and a number of other gastrointestinal (GI) disorders (33–39). It was postulated that EN would enhance inflammation in these disorders while "bowel rest" would prevent further damage by removing the stimulus of luminal antigens and stimulation of bowel function. However, the concept of bowel rest is seriously flawed. Starvation does not inhibit bowel function, and this approach is akin to inducing asystole to rest the heart. Starvation decreases splanchnic blood flow and results in profound structural and functional changes to the GI tract (see *Myth No. 2: Parenteral Nutrition Is Safe* section). Furthermore, recent data suggest that EN particularly with a lipid- and protein-rich formula has a profound anti-inflammatory effect on the GI mucosa (the gut-brain immune axis) (40). The luminal presence of a lipid- and protein-rich nutrition triggers a vagal reflex via peripheral cholecystokinin-1 receptors, which reduces local and systemic activation of peripheral nicotinic acetylcholine receptors on inflammatory cells (40). These data suggest that bowel rest is unlikely to be beneficial. Indeed, EN has been demonstrated to improve the outcome of the GI disorders for which bowel rest was considered the standard of care (41–43).

MYTH NO. 2: PARENTERAL NUTRITION IS SAFE

It is now widely accepted that the GI tract is the preferred route of delivering nutritional support (44). Furthermore, consensus guidelines strongly recommend "enteral over parenteral nutrition" (PN) in critically ill patients (1–3). The institution of early EN in critically ill medical and postoperative patients has been demonstrated to improve outcome (4, 6). Yet, PN continues to be widely used in patients who can be fed enterally. The adverse sequela associated with PN results from the "double hit" of not directly feeding the bowel, as well as the metabolic, immunologic, endocrine, and infective complications associated with infusing a solution with a high glucose concentration and fat globules into a patient's systemic venous system (45). PN bypasses the gut and liver. EN stimulates the release of a wide variety of enterohormones that play a crucial role in regulating gut function and metabolic pathways. Furthermore, the portal system supplies the liver with a rich source of nutrients and hormones, which are essential for hepatic function. Lack of enteral feeding results in GI mucosal atrophy, bacterial overgrowth, increased intestinal permeability, and translocation of bacteria and/or bacterial products. In a large cohort of critically ill patients, Grau et al (46) demonstrated that PN was strongly associated with the development of liver dysfunction, whereas early EN was protective. EN has a major effect on the gut-associated lymphoid tissue (GALT), which is the source of most mucosal immunity in humans. PN results in rapid and severe atrophy of this tissue (47–50). In addition, to its effects on the GALT, PN impairs humoral and cellular immunologic defenses. PN is associated with impaired leukocyte chemotaxis, impaired phagocytosis, impaired bacterial and fungal killing, and an attenuated inflammatory response (51–53). PN is associated with increased free radical formation (54); this complication may be of considerable importance in critically ill patients.

Heyland et al (55) performed a meta-analysis of PN (compared with no nutritional support) in critically ill patients. These authors demonstrated that PN almost doubled the risk of dying (relative risk [RR], 1.78; 95% CI, 1.11–2.85). The SCCM/ASPEN guidelines state that "Enteral nutrition is the preferred route of feeding over parenteral nutrition (PN) for the critically ill patient who requires nutrition support therapy (Grade: B)" and that "if early EN is not feasible or available the first 7 days following admission to the ICU, no nutrition support therapy should be provided" (1). However, if there is evidence of protein-calorie malnutrition at admission and all attempts at providing EN fail, the guidelines suggest that "it is appropriate to initiate PN as soon as possible following admission and adequate resuscitation" (1, 56–59).

Recently, the concept of supplemental PN has been popularized in patients who have a "short-term contraindication" to EN or until full caloric goals are achieved (60). In a landmark study, Casaer et al (61) randomized 4,640 ICU patients to either early (within 48 hr) or delayed (day 8) supplemental PN. In this study, for every outcome measure investigated, the patients who received early PN did worse. This included a group of patients who received no concomitant EN (62). Furthermore, they demonstrated a dose-response relationship; the more PN the patients received, the greater the likelihood of harm. Heidegger et al (63) randomized ICU patients to supplemental PN (between day 4 and day 8) or EN alone. Although the published data suggested a lower rate of nosocomial infections in the patients receiving supplemental PN, an intention-to-treat analysis failed to show this benefit (64, 65). Furthermore, there was no difference between groups for any of the secondary outcomes. Doig et al (66) randomized 1,372 patients with "relative contraindications" to early EN to early PN or

pragmatic standard of care. There was no difference in 60-day mortality, ICU, or hospital length of stay although the early PN group required 0.47 fewer days of ventilation. The study was conducted over 5 years in 31 hospitals in Australia and New Zealand; the number of screened patients nor the reasons why EN was contraindicated were not provided. However, 62% of those enrolled in this study were surgical patients; it is likely that EN was not contraindicated in many of these patients (see *Myth No. 7: EN Is Contraindicated in Patients Without Bowel Sound and/or a Postoperative Ileus* section). The results of these three studies fail to demonstrate a benefit from supplemental PN (61, 63, 66). Furthermore, although limited short-term PN may not be harmful (or beneficial), larger amounts of PN appear to be associated with harm (62). The 2013 Canadian Clinical Practice Guidelines "strongly recommend that early supplemental PN and high intravenous glucose not be used in unselected critically ill patients" (27).

MYTH NO. 3: EN CONTRAINDICATED WITH VASOPRESSORS

Many critically ill patients are hemodynamically unstable and/or require vasopressors/inotropes to maintain adequate blood pressure and cardiac output. Vasopressors improve hemodynamics by shunting blood from the gut and other peripheral organs (i.e., bone marrow, skin, and kidneys) to the central circulation. These "nonessential" organs are more sensitive to vasoconstriction than are central "essential" organs (i.e., heart and brain). Thus, the effect of vasoconstrictor medications and hypotension is a decrease in gut blood flow. It has, therefore, been postulated that because these patients have limited oxygen delivery and that by increasing GI oxygen demand with enteral feeding, intestinal ischemia will develop. However, these propositions are based on evidence from animal models where the mesenteric artery was occluded and in patients with atherosclerotic occlusion of the mesenteric arteries (67). Based on this information, many clinicians believe that EN will cause bowel ischemia and is contraindicated in patients receiving pressors. Anecdotal cases reports of mesenteric ischemia in trauma patients receiving vasopressor agents (see below) are often cited to support this belief (68). However, this theory is incorrect. Indeed, both experimental and clinical studies demonstrate that EN increases gut blood flow and protects against bowel ischemia. In the experimental and clinical setting, enteral infusion of nutrients prevents adverse structural and functional alterations of the gut barrier, increases epithelial proliferation, maintains mucosal integrity, decreases gut permeability, improves gut blood flow, and improves local and systemic immune responsiveness. These effects are mediated via both direct and indirect (i.e., hormonal and neuronal) effects (69–71).

In endotoxic and septic shock models, 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 (70, 72, 73). These experimental data have been confirmed by clinical studies. Revelly et al (74) evaluated EN in nine patients requiring hemodynamic support by catecholamines 1 day after cardiac surgery. Patients were fed with postpyloric feeding tubes. During enteral feeding, cardiac index, indocyanine green clearance, and glucose absorption increased while gastric tonometry remained unchanged. Similarly, Berger et al (75) demonstrated "close-to-normal" paracetamol area under the curve (a test of intestinal absorption) in hemodynamically unstable cardiac surgery patients receiving EN. Overall, these studies indicate that enteral nutrients improve gut blood flow with preservation of the bowel absorptive capacity during vasopressor administration. The benefits of early EN in critically ill patients treated with vasopressors are supported by a multicenter study, which demonstrated a lower hospital mortality in patients fed within 48 hours (34% vs 44%, $p < 0.001$) (76). In this study, the benefits of early EN were greatest in the sickest patients and those receiving multiple vasopressors.

MYTH NO. 4: EARLY EN IS NOT IMPORTANT IN PATIENTS RECEIVING MECHANICAL VENTILATION

The initiation of EN is often delayed in patients receiving mechanical ventilation. It is likely that nutrition is not considered a priority and thus pushed to the "back burner" while more acute issues take precedence. Furthermore, many may cite older guidelines that stated that it was acceptable for mechanically ventilated patients to go a week without nutrition. In the German Competence Network Sepsis (SepNet) study, mechanical ventilation was a strong predictor for the failure to provide EN (11). Artinian et al (6) performed a retrospective analysis of a prospectively collected large multicenter ICU database to determine the impact of early enteral feeding on the outcome of mechanically ventilated critically ill medical patients. In this study, early EN (within 2 d of admission) was associated with a significant reduction of ICU and hospital mortality. Barr et al (77) demonstrated that the implementation of an evidence-based nutritional management protocol significantly shortened the duration of mechanical ventilation. In this study, EN was associated with a reduced risk of death.

MYTH NO. 5: EN IS CONTRAINDICATED WITH HIGH GASTRIC RESIDUAL VOLUME

Many clinicians monitor gastric residual volumes (GRV). The presumption is that GRV measurements are accurate and useful markers for the risk of aspiration and pneumonia. Enteral feeding is then interrupted when the GRV exceeds 150 mL. There is, however, no data to support this practice. High GRVs (i.e., > 400 mL) do not necessarily predict aspiration, and low GRVs (i.e., < 100 mL) are no guarantee that aspiration will not occur. Interrupting EN when the GRV exceeds 100–200 mL has not been shown to decrease the prevalence of aspiration. McClave et al (78) randomized critically ill ventilated patients to two management strategies using a GRV more than 200 mL or GRV more than 400 mL for interrupting gastric feeding. In this study, the prevalence of aspiration was similar between groups. Similarly in a prospective multicenter study, Montejo et al (79) randomized patients to a control group (GVR

> 200 mL) or an intervention group in which tube feeds were held when the GRV exceeds 500 mL. In this study, there was no difference in the risk of pneumonia, ventilator-free days, organ failure, or mortality between groups. More recently, Reignier et al (80) randomized mechanically ventilated patients to a group in which the GVR was not monitored and a group in whom tube feeds were held when the GRV exceeded 250 mL. These investigators demonstrated no difference in the risk of pneumonia between groups; however, the proportion of patients receiving their caloric goal was higher in the no-GRV group. It should, however, be noted that in this study, patients with abdominal surgery within the past month; a history of esophageal, duodenal, pancreatic, or gastric surgery; a history of GI bleeding; and contraindications to prokinetic agents were excluded. These data suggest that there is poor relationship between GVR and the risk of aspiration. Monitoring GVR may not be necessary in patients at low risk for aspiration and may only serve to reduce the amount of nutrition provided.

MYTH NO. 6: POSTPYLORIC FEEDING REDUCES THE RISK OF ASPIRATION

As an extension of the myth that the GRV is associated with the risk of aspiration pneumonia, many clinicians believe that all critically ill patients should receive postpyloric feeding. Cleary, there are some critically ill patients who have impaired gastric motility (especially patients with diabetes) who cannot tolerate early gastric feeds in whom EN is tolerated if delivered beyond the pylorus (81). However, there is little consensus regarding the issue as to whether the routine use of postpyloric feeding decreases the risk of aspiration pneumonia. We performed a meta-analysis comparing the risk of pneumonia in patients fed gastrically versus postpylorically (82). In this meta-analysis, the risk of pneumonia was unrelated to the route of feeding. Ho et al (83) reported similar findings. However, a meta-analysis by Alhazzani et al (84) demonstrated a small reduction in the risk of pneumonia with small bowel feeding without affecting mortality, ICU length of stay, or duration of mechanical ventilation. We suggest placement of an orogastric tube and early (within 12 hr of ICU admission) initiation of EN in all mechanically ventilated patients. In those patients who demonstrate intolerance to gastric feeding (abdominal distension, regurgitation), we suggest the use of prokinetic agents (81, 85, 86). Should this approach fail, we would then place a postpyloric feeding tube. In patients with known gastric dysmotility and those who are nursed supine (e.g., extracorporeal membrane oxygenation patients), we would suggest early placement of a postpyloric feeding tube.

MYTH NO. 7: EN IS CONTRAINDICATED IN PATIENTS WITHOUT BOWEL SOUND AND/OR A POSTOPERATIVE ILEUS

In 1905, Cannon (87) was the first clinician to formally suggest a relationship between abdominal auscultation and bowel function. Remarkably, abdominal auscultation has become part of the standard physical examination of patients, and yet, no studies have validated the value of this maneuver. Historically, ICU nurses have been trained to auscultate each of the four abdominal quadrants for the presence of bowel sounds, with the presence of bowel sounds indicating that it is safe to feed patients. Similarly, the return of bowel sounds after abdominal surgery has been regarded as an indicator of the resolution of postoperative ileus and an indicator that it is safe to commence EN. However, the absence of bowel sounds does not mean that the bowel is not working. Bowel sounds result from air moving through the small intestine. The presence of bowel sounds requires swallowing of air and gastric emptying. Many seriously ill patients have little movement of air from the stomach to the small intestine and therefore have decreased bowel sounds. The absence of bowel sounds after operation seems to result from "the emptiness of the gut." When fluid and air is injected into the duodenum, sounds can be heard immediately (88).

Waldhausen et al (89) measured GI myoelectric and clinical patterns of recovery after laparotomy. Small bowel myoelectric activity returned immediately after surgery, whereas it took on average 2.4 days for the return of bowel sounds and 5 days for the passage of flatus. These authors were unable to find any correlation between bowel myoelectric activity and bowel sounds. These data suggest that ausculting for bowel sound has limited clinical utility and should not be used to guide the initiation of EN. Indeed, multiple clinical trial have shown improved outcome with the early initiation of tube feeds following abdominal surgery in spite of the absence of bowel sounds or the passage of flatus. Current guidelines recommend that "in the ICU patient population, neither the presence nor the absence of bowel sounds nor evidence of the passage of flatus or stool is required for the initiation of enteral feeding" (1).

MYTH NO. 8: EN IS CONTRAINDICATED FOLLOWING GI SURGERY

Classic surgical teaching suggests that due to reflex inhibition, the alimentary tract becomes inactive after abdominal surgery (88). The period of inactivity or postoperative ileus is thought to last for 3–5 days during which time the patient is tided over by gastric aspiration and parenteral fluids (88). It has been assumed that the postoperative ileus precludes enteral feeding. Furthermore, it has been suggested that bowel distention following enteral feeding would disrupt the anastomoses. Consequently, EN is frequently withheld from postoperative abdominal surgery patients, particularly those with fresh GI tract anastomoses. This approach is detrimental to patients and without scientific evidence. Motility studies demonstrate return of small bowel peristalsis within hours after laparotomy providing support for early postoperative EN (89, 90). Over 30 years ago, Moss (91) demonstrated the benefits of immediate EN following laparotomy and colorectal excision. In this study, a full-strength elemental diet was delivered into the duodenum immediately postoperatively. Using radiolabelled albumin, he demonstrated that 94% ± 4% of the albumin was absorbed with achievement of a positive protein balance by 5 hours postoperatively. Furthermore, barium motility studies performed on the first postoperative day demonstrated clinically adequate peristalsis.

In a canine model, Kawasaki et al (92) compared the effects of EN versus PN on GI motility after open abdominal surgery. They demonstrated that EN hastened recovery of GI motility. Therefore, early postoperative enteral feeding may be an effective way to decrease the duration of postoperative ileus. This is based on the fact that enteral feeding stimulates reflexes that produce coordinated propulsive bowel activity and increase the secretion of GI hormones that increase bowel motility (93). Hence, early EN appears to be an effective means of treating an ileus with starvation only serving to prolong the ileus.

The GI tract produces approximately 6 L of fluid per day, and it is illogical to propose that an additional liter or so of tube feeds will cause excessive distention of the bowel with anastomotic dehiscence. Furthermore, wound healing is critically dependent on an adequate supply of protein; starvation with protein catabolism is likely to increase the risk of wound dehisce. In an animal model, Moss et al (94) demonstrated that early enteral feeding doubled the bursting pressure of the colorectal anastomosis, with the anastomoses containing significantly higher concentration of collagen and collagen precursors than those of the unfed controls. Multiple experimental studies have demonstrated that early EN following bowel surgery is associated with improved wound healing, greater wound strength, and higher wound hydroxyproline and collagen accumulation (95–100). This may explain the lower risk of anatomic leaks and fistulas in bowel surgery patients who receive early enteral as opposed to delayed feeding or PN (5, 9, 101).

The experimental data demonstrating the benefit of early EN are supported by a large number of studies which have demonstrated the safety and improved outcomes associated with early EN in patients who have undergone both small and large bowel surgery. In 2001, we published a meta-analysis of 15 randomized controlled trials that compared early with delayed EN in postoperative patients (4). We demonstrated that early EN was associated with a significantly lower risk of infection (RR, 0.45; 95% CI, 0.3–0.66) and reduced length of hospital stay (mean 2.2 d; 95% CI, 0.81–3.63 d). More recent meta-analyses have reproduced these findings (5, 10). These data clearly demonstrate that early EN following GI surgery is feasible and that this intervention improves patient outcomes.

Concern has been raised that early enteral feeding may cause bowel ischemia following abdominal surgery (68). This is a very rare complication that was reported predominantly between 1986 and 2000 with isolated cases reported subsequently (102–108). Most of these patients had sustained traumatic injuries, and almost all had undergone a laparotomy with surgical placement of a jejunostomy tube (68, 102–104). Small bowel necrosis is very rare in postoperative patients who are initiated on early enteral feeding. Nevertheless, enteral feeding should be advanced slowly in patients at risk (severe abdominal trauma, large burns), and they should be discontinued in patients who developed abdominal complaints, such as pain, distention, and vomiting, until the status of bowel integrity can be evaluated. A semielemental formula may be advantageous in these patients.

MYTH NO. 9: EN IS CONTRAINDICATED IN PATIENTS WITH AN OPEN ABDOMEN

Decompressive celiotomy has reduced the mortality of patients with abdominal compartment syndrome (109). The management of these patients is challenging with the approach to the route and timing of nutritional support being controversial. Many patients are kept "nil per os" or receive PN on the assumption that these patients cannot be fed enterally due to bowel wall edema and bowel dysfunction. However, clinical studies have demonstrated that early EN is feasible in patients with an open abdomen and that this approach is associated with improved outcomes (110–112). Collier et al (101) demonstrated that early enteral feeding (within 4 d of celiotomy) was associated with earlier closure of the abdominal cavity and less fistula formation when compared with the delayed initiation of EN.

MYTH NO. 10: EN IS CONTRAINDICATED IN PATIENTS WITH PANCREATITIS

In patients with acute severe pancreatitis, classic teaching suggested that "total parenteral nutrition should be initiated promptly and should judiciously replace nutrient deficits and provide the extra energy imposed on the patient by the inflammatory process" (113). It was claimed that this approach was essential to "rest the pancreas" and that PN reduced mortality (113). EN was considered an absolute contraindication as it would stimulate the pancreas and worsen pancreatic inflammation. Randomized clinical trials comparing EN versus PN in patients with moderate and severe pancreatitis have, however, proven these recommendations to be wrong. Meta-analyses have demonstrated that EN as compared with PN reduces infectious complications (particularly pancreatic abscesses), organ failure, length of hospital stay, and mortality (43, 114, 115). Nutritional support should be viewed as an active therapeutic intervention that improves the outcome of patients with acute pancreatitis. EN should begin within 24 hours after admission and following the initial period of volume resuscitation and control of nausea and pain. Patients with mild acute pancreatitis should be started on a low-fat oral diet. In patients with severe acute pancreatitis, EN may be provided by the gastric or jejunal route (114).

MYTH NO. 11: PATIENTS MUST BE FED SEMIRECUMBENT AT 45°

In an article published in 1999, Drakulovic et al (116) demonstrated a lower frequency of clinically suspected ventilator-associated pneumonia (VAP) in 39 intubated patients randomized to the semirecumbent (45°) as opposed to the supine body position (47 patients). In this study, the risk of pneumonia was highest for patients receiving EN in the supine body position. Based on this small single-center study, it became standard of care to nurse all ICU patients in a semirecumbent 45° position particularly when receiving tube feeds. Indeed, the Centers for Disease Control and Prevention (117), the Agency for Healthcare Research and Quality (118), and the Institute for Healthcare Improvement (119) suggest elevating the head of the bed to

45° above horizontal to reduce gastroesophageal reflux and the prevalence of nosocomial pneumonia. The results of the study by Drakulovic et al (116) have, however, not been reproduced. Van Nieuwenhoven et al (120) randomized 112 intubated patients to the semirecumbent position with a target backrest elevation of 45° and 109 patients to a supine position with a backrest elevation of 10°. Average elevations were 9.8° and 16.1° at day 1 and day 7, respectively, for the supine group and 28.1° and 22.6° at day 1 and day 7, respectively, for the semirecumbent group. The target semirecumbent position of 45° was not achieved for 85% of the study time, and these patients more frequently changed position than supine-positioned patients. There was no difference in the risk VAP or any other outcome variable between groups. In an observational study of 66 ventilated patients, Grap et al (121) reported a mean backrest elevation of 21.7° with no association between backrest elevation and the Clinical Pulmonary Infection Score. Rose et al (122) performed 2,112 backrest elevation measurements in 371 patients in 32 ICUs. Backrest elevation more than or equal to 45° was recorded in 5.3% of instances and elevation of between 30° and 45° in 22.3% of instances (122). In this study, the mean backrest elevation was 23.8°. These studies suggest that nursing a patient semirecumbent at 45° is not feasible and attempts to do so may not reduce the risk of VAP. When the head of the bed is inclined at 45°, the patient often slides down; most of the weight of the upper body is applied on the sacral area, and this position becomes uncomfortable for the patient. Furthermore, experimental models suggest that the semirecumbent position may enhance the flow of mucous into the lungs with an increased risk of bacterial colonization and pneumonia (123). Although maintaining a patient supine (0°) probably increases the risk of pneumonia, there is no strong evidence that elevation of the head of the bed between 10° and 30° is associated with a greater risk of pneumonia than a semirecumbent 45° position.

CONCLUSION

We demonstrate that numerous myths and misconceptions abound which act in concert to delay and limit the provision of optimal nutritional support in critically ill patients. With few exceptions, early EN is feasible and improves the outcome of critically ill ICU patients. Such treatment should be considered the standard of care, and the early initiation of EN should be used as an indicator of the quality of care delivered in ICUs.

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