Nutrition in the Surgical Patient

10

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Nutrition in the surgical patient is a multifactorial, complex subject. Beyond the decision to feed enterally or parenterally, a surgeon must consider specific patient characteristics that interfere with the delivery of nutrients for useful and purposeful digestion and metabolism. The patient with postoperative ileus, a previous bowel obstruction, short gut, an open abdomen after damage control, or discontinuous bowel, to mention only a few special circumstances, has energy requirements beyond what is provided by maintenance or resuscitative fluids. These examples comprise situations in which early feeding would inherently be of benefit. Certainly the patient with an enteric fistula deserves focused discussion as this patient population, more than the standard surgical patient or even the patient with an open abdomen after damage control, has the additional complexity of nutrient and digestive component loss.

Attention should also be given to the consideration of nutritional access as many patients with these special circumstances do not have the ability to take food orally. Surgeons must decide how they will provide nutrition to their patients and many times this requires surgical or endoscopic placement of lines and tubes that can be used to administer nutrients into the body. Timing of feeding and location of feed entry into the body are further decisions that the surgeon faces. This chapter serves to discuss and present data regarding the differences in parenteral, enteral, gastric, and post-pyloric feeding, and includes algorithms for instituting early nutritional support in the acute and traumatic patient populations.

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Rationale for and Types of Nutritional Support

The rationale for providing nutritional support is to prevent acute protein malnutrition, to modulate the immune response, and to promote normal gut function [1].

Enteral Versus Parenteral Nutrition

In the 1970s total parenteral nutrition (TPN) was introduced, but despite its availability, enteral nutrition (EN) was still more economical and convenient to provide. However, the practice at that time was to hold EN until the gut proved to be completely functional, which could take days or even weeks, for surgical and trauma patients. By the 1980s enough data had been collected to support the use of EN in these surgical populations. Enteral nutrient provisions were functional and processed effectively in the critically ill patient with maladapted gut mucosa [2, 3]. In fact it was shown in multiple studies that introducing enteral feeds into the gut stimulated immunologic response and competence [4–7]. The 1990s introduced data that TPN may be harmful in patients who could otherwise tolerate enteral feeds. There were more infections, including catheter-related sepsis, seen in the parenteral group [8, 9]. Meta-analyses confirmed that early enteral feeding, compared to parenteral nutrition, reduced postoperative infections and complications [10, 11].

Enteral Nutrition

Enteral nutrition is the preferred form of nutritional supplementation in surgical patients who have enteral access [12–14]. Absolute contraindications to enteral feeds include functional complications such as bowel obstruction, peritonitis, progressive ileus, massive gastrointestinal hemorrhage, and gastrointestinal ischemia associated with shock and

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vasopressors. Relative contraindications include proven intolerance to enteral nutrition and intolerance associated with short gut syndrome, high-output fistula, pancreatitis, and inflammatory bowel disease.

Early enteral feeding supports gastrointestinal structure and function, and in the critically ill surgical patient can reduce gut hyper-permeability, enhance gut blood flow, promote gastric emptying, and stimulate gut-associated immunity. Multiple studies have shown tolerance of trophic feeds in critically ill and mechanically ventilated patients, and in patients with recent bowel surgery [15]. While there are studies that show some increased infectious complications with early goal enteral feeds, there is more convincing data to the contrary [13, 14, 16]. Based on 14 Level 2 studies, early EN was shown to reduce infectious complications and mortality and is overwhelmingly recommended in mechanically ventilated patients after adequate resuscitation [17, 18].

Parenteral Nutrition

Total parenteral nutrition is appropriate in situations in which enteral feeds cannot be used. Its disadvantages include need for vascular access, infection of vascular access and associated bloodstream infection, sepsis, cost, need to monitor electrolytes and adjust formula, and hyperglycemia. Several types of amino acid-specific formulas for TPN are available and there is evidence to support the use of glutamine for both enteral and parenteral nutrition, regardless of the formula used [19, 20]. Glutamine shows decreased complications and increased survival when added as a supplement to TPN [21].

Whenever possible, the gastrointestinal track should be utilized for nutritional support. The algorithm (Fig. 10.1) reviews the decision process for starting EN and for the administration of TPN. In general, TPN should be started by 7–10 days postoperatively if the patient is well nourished at baseline and unable to tolerate adequate EN. Unlike early enteral feeding, there is no clear benefit to early TPN. There is equally no difference in outcomes for patients who take enteral and parenteral nutrition in combination [22]. Patients with persistent ileus, bowel obstruction, short gut, high-output fistulas, and malabsorption may all benefit from TPN. Additionally, patients unable to tolerate EN or who are at risk for non-occlusive bowel necrosis (hypoperfusion, vasopressor, or paralytic requirements) may benefit from TPN. There is new data that indicates that the risk of infection with the parenteral route may have been overestimated as a recent randomized trial performed in the UK and involving 33 English intensive care units and 2400 patients [23]. This study showed no significant difference in the mean number of treated infectious complications or in the 30-day mortality among patients receiving early parenteral nutrition compared to patients receiving early EN. Another Australian randomized single-blind clinical trial involving 31 hospitals with 1372 patients even demonstrated significantly fewer days of invasive ventilation but not significantly shorter intensive care unit (ICU) or hospital stays with early parenteral nutrition when compared with no nutrition in the presence of relative contraindications for EN [24]. Parenteral nutrition thus remains a valuable and necessary tool in specific patient populations.

Determining Caloric Needs

Caloric needs can be calculated using one of many formulas such as the Harris–Benedict equation or measured with indirect calorimetry.

Harris–Benedict Equation

The Harris–Benedict equation estimates basal energy expenditure (BEE) to determine caloric requirements. The Harris–Benedict equations are specific to men and women based on weight, body mass index (BMI), and height and are as follows:

$$\text{Men:}\quad \text{BEE} = 66 + (13.7 \times \text{weight}) + (5 \times \text{height}) - (6.8 \times \text{age})$$

$$\text{Women:}\quad \text{BEE} = 665 + (9.6 \times \text{weight}) + (1.9 \times \text{height}) - (4.7 \times \text{age}).$$

Weight is in kilograms (kg), height in centimeters (cm), and age in years. The BEE represents energy requirements in the fasting, resting, and non-stressed state, so it may not be completely accurate in trauma or surgical patients. In the presence of metabolic stress, the BEE must be multiplied by an empirically derived stress factor; this factor may grossly overestimate the true caloric needs of the individual and remains the source of controversy in using this formula in the critically ill. Overestimation of caloric needs results in complications such as overfeeding, hypercapnia, hyperglycemia, and hepatic steatosis. The new multiplication constants to estimate the stressed caloric needs range from 1.2 to 1.6 times the BEE. These new recommendations better estimate the caloric needs of even the most stressed patient scenarios, such as burns.

Indirect Calorimetry

Indirect calorimetry is a tool used to measure resting energy expenditure (REE) and relies on the relationship of oxygen consumption and carbon dioxide production. Because of the

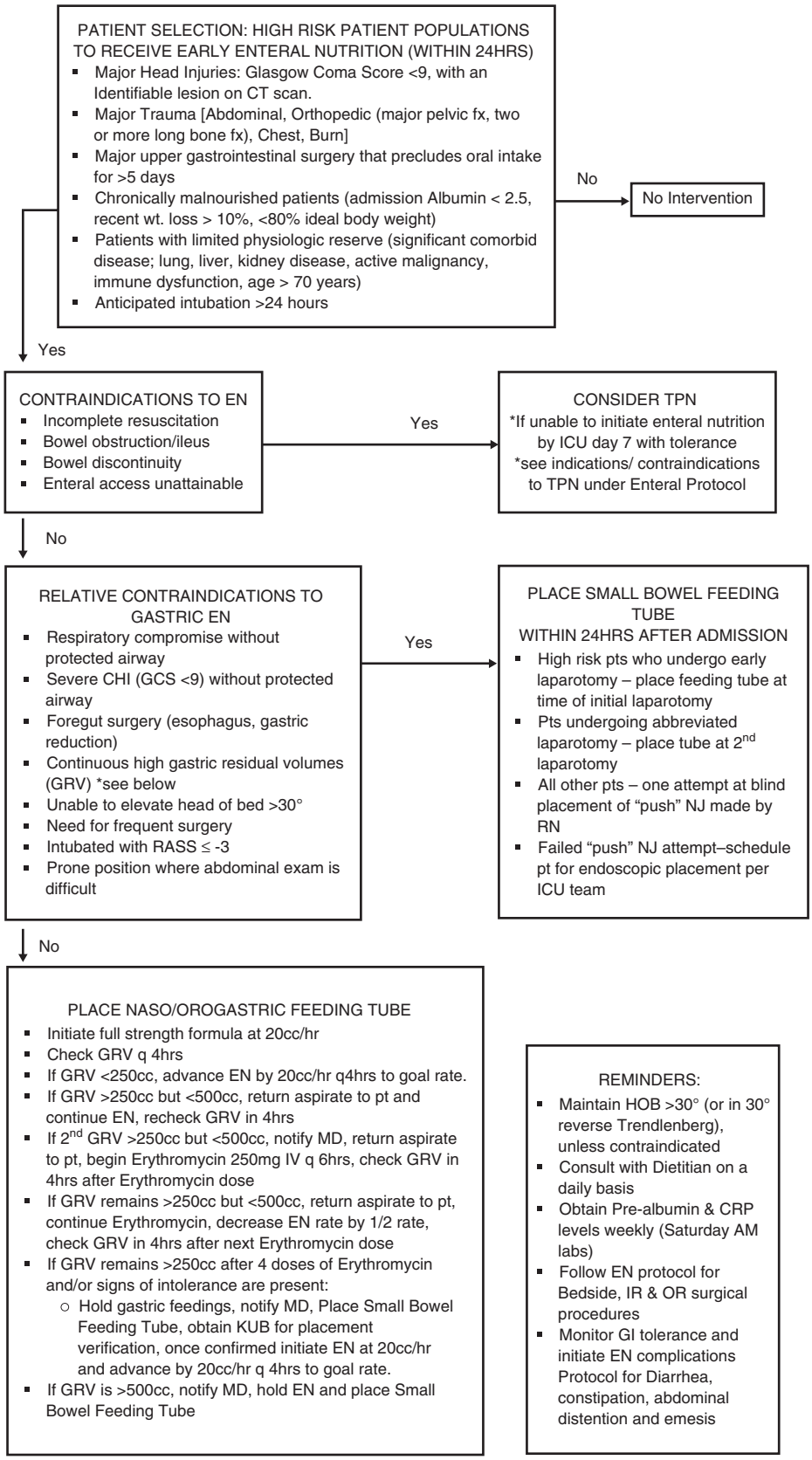

Fig. 10.1 Example of an enteral nutrition protocol algorithm

components necessary to calculate the REE, patients should be ventilated for best accuracy, although there is support to use it even in spontaneously breathing patients. It is recommended that steady state be achieved, defined as a change in either parameter of less than 10 % over 5 min or more [25]. The REE obtained should then be used to estimate the patient's baseline nutritional goal. Indirect calorimetry may be helpful when overfeeding would be undesirable (as in diabetes, obesity, or chronic obstructive pulmonary disease), underfeeding would be especially detrimental (renal failure, large wounds), physical or clinical factors promote energy expenditure that deviates from normal, drugs are used that may significantly alter energy expenditure (paralytic agents, beta-blockers, corticosteroids), patient response to calculated regimens is suboptimal, or body habitus makes energy expenditure predictions challenging (morbid obesity, quadriplegia).

The respiratory quotient is another derivative from the components of the indirect calorimetry. The formula is below:

$$\text{Respiratory quotient}\left(\text{RQ}\right)=\frac{V_{O2}}{V_{CO2}}=\frac{CO_2\,\text{production}}{O_2\,\text{consumption}}$$

The RQ is a gross measurement of substrate utilization [26]. When an RQ value ≥1 is obtained, CO_2 production may be increased by one of the two mechanisms: either a high proportion of non-protein calories are being supplied as glucose (carbohydrates have RQ of 1) or less commonly, the patient is being provided excess calories. Failure to wean with a persistently elevated PCO_2 on an arterial blood gas should prompt measurement of the RQ. An RQ of 0.85 provides optimal utilization, while <0.7 suggests gross underfeeding and ketone utilization.

Calculating TPN

Components of TPN include dextrose, fatty acids, amino acids, electrolytes, vitamins, and trace minerals. Dextrose is the carbohydrate at a caloric density of 3.4 kcal/g. Dextrose solutions of 50 or 70 % dextrose are readily available, but any carbohydrate percentage and volume can be mixed according to the patient's need. Protein provides 4 kcal/g and is provided as amino acids. Standard amino acid solutions contain a balance of essential and nonessential amino acids and are available as either 10 g/100 ml or 15 g/100 ml. Fat emulsions are 2.0 kcal/cm^3 of 20 % lipid and are the source of essential fatty acids, linoleic, linolenic, and arachidonic acids. The electrolyte cations, which include sodium, potassium, magnesium, phosphorus, and calcium, are mixed into the TPN solution using one of several anions. Acid–base status may be affected by the amount of chloride or acetate used in providing sodium and potassium. The concentrations of calcium and phosphorus are limited to avoid precipitation of a calcium phosphate salt. Vitamins included are A, C, D, E, and B vitamins, including folate, but not vitamin K, which must be added separately. Mineral product is added to provide copper, chromium, manganese, zinc, and selenium. The basic steps in calculating TPN are as follows: (1) establish the kilocalories and protein desired, (2) select the appropriate amino acid formula and quantity, (3) calculate 10 % of kcal as lipid emulsion, and (4) tally the kcal from amino acids and fat and subtract from goal, which is the amount of dextrose kcal needed. Divide this number by 3.4 to get the grams of dextrose required [27].

Types of Formulas

The primary categories of enteral formulas include polymeric, elemental, immune-enhancing, and specialty formulas.

Standard Enteral Diet versus Immune-Enhancing Diets

Both basic and clinical research suggests that the beneficial effects of enteral nutrition can be amplified by supplementing formulas with specific nutrients that exert immune-enhancing effects, including glutamine, arginine, nucleotides, and omega-3 fatty acids. There are numerous prospective randomized controlled trials comparing immune-enhancing enteral diets to standard enteral diet and most, but not all, demonstrate improved outcomes. The majority of trials are in trauma and cancer patients, though a few trials include mixed ICU and septic ICU patients.

Pharmaconutrition

The concept of pharmaconutrition allows the separation of nutritional support from the provision of key nutrients that may modulate the inflammatory and immune response associated with critical illness. This came about after the realization that the greatest benefit in clinical outcomes was from studies utilizing specific nutrients [16]. This is likely due to their effects on the enteric inflammatory response and the way in which they work to block inflammatory stimulation. Any event that stimulates a gastrointestinal inflammatory response and a change in gut perfusion alters the way that the gastrointestinal tract utilizes nutrients. Providing intraluminal alimentation to stressed mucosa of the gut improves intestinal transit [28]. Pharmaconutrients alone or as supplementation have been shown to decrease infectious complications and complication-associated length of hospital stay [29].

Glutamine is the primary fuel source for the enterocyte and is preferred to glucose as a fuel source in times of stress [30]. It is released from muscle during the stress response and then exploited as a signal mechanism, promoting immune regulation and cellular protection, and as a nutrient and source of energy [31]. But in addition, glutamine has anti-catabolic and antioxidant properties that enhance its use and its receipt at enterocytes. Furthermore it increases plasma concentration of arginine [32]. Although glutamine can be provided both enterally and parenterally, it demonstrates the most benefit of barrier to infection and control of the immune response when given enterally [32]. Meta-analysis and prospective randomized trials for trauma and burn patients showed benefit of glutamine in these patient populations in terms of decreasing infectious complications and enhancing the gut's use of other enteric nutrients [33–37]. Based on the available data, glutamine, despite the administration route, appears to lower infectious complications, decrease hospital length of stay, and enhance nutrient use in the critically ill patient [38, 39]. Heat-shock proteins, which serve as molecular regulators of denatured proteins, are induced by glutamine, which may be another way in which glutamine modulates the cyto-protection and inflammatory response [40–42]. Equally important is the lack of data showing adverse effect of using glutamine in either form.

Arginine is another modulator of immune response of the enteric system. It is produced both endogenously from glutamine and the urea cycle, and obtained from the diet. When there is normal physiology without ongoing stress response, arginine serves to enhance immune function, contribute to wound healing, and stimulate anabolic hormones. L-arginine is a substrate for nitric oxide, which itself enhances the inflammatory response. L-arginine and its pathway to creating nitric oxide is a potential target for modification of immune activation. Specifically in trauma patients it has been shown that the release of IL-4, IL-10, and transforming growth factor beta increases arginase I expression, which corresponds to increased immune cell arginase activity and decreased plasma arginine and citrulline levels [43, 44]. By shunting arginine use in this way, it can no longer be used as a substrate for nitric oxide synthase dimerization and nitric oxide production. Therefore, administration of supplemental arginine in the critically ill patient may reduce the amount of nitric oxide produced in the post-injury period. Arguing against this data is work from another group suggesting that arginine supplementation increases nitric oxide production, thereby amplifying the systemic inflammatory response syndrome (SIRS) response and increasing mortality in the trauma or critically ill patient [45, 46]. There exists data supporting and refuting the use of arginine supplementation for both enteral and parenteral routes of administration [47–50]. It is clear, however, that arginine supplementation in elective surgical patients is beneficial. A recent meta-analysis by Drover et al. demonstrated a significant decrease in postoperative complications and hospital length of stay when patients undergoing gastrointestinal surgery received pre-, peri-, or postoperative arginine supplementation [51]. The effect was greatest when the supplementation included arginine as well as omega-3 fatty acids and nucleotides.

Nucleotides play an active role in cellular proliferation and immune modulation and are building blocks for several intrinsic cellular molecules. They are produced de novo and by salvage pathways. T cell proliferation and appropriate recognition of antigen are thought to be dependent on the presence of nucleotide because it has been shown that artificial decrease in interleukin-2 is corrected by addition of supplemental nucleotide [52]. They are either purine or pyrimidine derived with a ribose and one or more phosphate groups [53]. Similar to glutamine and arginine, intravenous (IV) and enteral forms are available. Infusions of nucleotides decrease bacterial translocation and decrease graft rejection [52, 54]. These references also show that parenteral doses of nucleotides, administered with TPN, decrease associated gut atrophy.

Omega-3 fatty acids are the active components of fish oils and have significant anti-inflammatory properties [55], the mechanism of which is likely a combination of functions including arachidonic acid displacement from cellular membranes, production of prostaglandins, and reduced activation of various nuclear factors [56]. Specifically, they target and down-regulate NF-kB and AP-1 [54] on the nuclear membrane and they down-regulate iNOS, thereby reducing production of nitric oxide. While there are no studies of critically ill patients who received only omega-3 fatty acid and no additional supplementation, there are three prospective randomized studies that included omega-3 fatty acid in the supplementation package and had a significant improvement in respiratory function of their critically ill patients [57–59].

Beyond activation of the immune system, the critically ill and traumatic patient suffers damage at the cellular level secondary to the effects of oxidation-induced injury. Antioxidants have been found to catalyze the breakdown of the substances that are implicated in causing this damage. Superoxide dismutase, catalase, and glutathione peroxidase have been identified as antioxidants; cofactors include selenium, zinc, manganese, and iron. Supplementation of these substances decreases the inflammatory response and halts oxidative stress [60–62]. Similar to nucleotides, it has been shown that the number of days on mechanical ventilation and overall mortality can be reduced by supplementation of antioxidants and their cofactors [62–64].

The value of vitamin supplementation has also been studied and it has been suggested that intravenous ascorbic acid addition in patients with severe sepsis is safe and results in reduction of pro-inflammatory markers such as C-reactive protein and procalcitonin although the effect on patient outcomes has not been proven [65]. The same findings could not be extended to other vitamins such as Vitamin D supplementation in Vitamin D deficient patients with sepsis [66].

Despite numerous studies demonstrating benefit from supplemental nutrients, a recent prospective randomized trial comparing enteral and parenteral glutamine and antioxidants in critically ill patients with established organ failure, parenteral and enteral glutamine supplementation resulted in a nonsignificant trend toward increased mortality. Therefore, the administration of glutamine is no longer recommended for patients with organ failure. [67]. A post hoc analysis did, however, suggest that glutamine may be safe for trauma and burn patients when administered prior to the development of distant organ injury [68].

Optimal Route of Delivery of Enteral Nutrition

Access can be divided into gastric (and duodenal) and jejunal with push, endoscopic, radiologic, and surgical options all available. For patients to be fed gastrically, a soft, non-sump nasogastric tube can be placed. There are also blindly placed nasojejunal tubes. If blind placement is unsuccessful, an endoscopically placed nasojejunal tube is an option. Nasojejunal feeding may be done indefinitely, but if the need for long-term access becomes apparent, either a percutaneous endoscopic gastrostomy (PEG) or a PEG with a jejunal extension limb (PEG-J) can be placed. For those patients identified as candidates for jejunal feeds and undergoing laparotomy, either a standard open jejunostomy or a needle catheter jejunostomy (NCJ) can be placed.

The largest study examining the safety of needle catheter jejunostomies in patients undergoing major elective and emergency abdominal operations documented an incidence of major complications of 1 % and minor complications of 1.7 % [69]. When feeding jejunostomy-related complications in trauma patients were reviewed by Holmes et al. [70] the overall major complication rate was 4 %. However, the majority of complications occurred in patients with a Witzel tube jejunostomy (10 %), with only a 2 % rate with NCJs. In fact, the only difference between patients with and without major complications was the type of feeding access. Major complications included small bowel perforation, volvuli with infarction, intraperitoneal leaks, and non-occlusive small bowel necrosis. The first three of these complications can be minimized by improved technique and the latter minimized by more judicious feeding.

Gastric Versus Small Bowel Feeding Controversy

While gastric and post-pyloric nutrition have been compared, statistically no difference is noted in the time to reach caloric goal, length of stay in the ICU, or length of ventilator time between the two [71]. There is a consistent delay in initiating gastric feeds when compared to post-pyloric feeds in surgical patients, but again, the ultimate outcomes data do not differ. The early initiation of pro-kinetic agents may also be of benefit. In fact gastric feeds and post-pyloric feeds can achieve the same caloric supplementation in the same amount of time in the critically ill patients [72]. It has also been shown that initiating early enteric feeds (within 36 h) improves survival and decreases infectious complications [73].

If feeds are provided past the ligament of Treitz, enteral feeds do not need to be held for the operating room [74]. This is important in the surgical population where frequent trips to the operating room might otherwise greatly hamper uninterrupted full caloric nutrition in these patients. Aspiration during intubation remains a risk for patients who have been gastrically fed [75]. This same risk does not appear as evident even for patients who have continuous jejunal tube feeds running during their operations. There is no difference in aspiration risk in gastric or post-pyloric feeds with respect to aspiration risk or residuals [76]. Furthermore, there does not seem to be a significant difference in rates of pneumonia or ICU mortality among adult ICU patients fed intra-gastric or through a jejunal tube [77].

Additionally the question of gastrointestinal prophylaxis in the patient who is ventilated and fed into the small bowel is significant. Gastric pH must be addressed in any patient intubated more than 48 h and undergoing non-gastric nutritional support. This is to prevent stress ulceration, which is a known complication of ICU patients. Because gastric tubes can be placed nasally and blindly by push technique easier than jejunal tubes, the natural tendency is toward placing nasogastric (NG) tubes for decompression and to pass a nasojejunal tube and feed it even if gastric. There may be a need for recommendations on post-pyloric feeds in ICU-level patients secondary to their frequent trips to the operating room, need for continuous uninterrupted feeds to prevent malnutrition, and prevention of aspiration. Equally one could argue for gastric feeds with head of bed elevation, which might cut the number of stress ulcers and reduce the number of procedures and sedation that ICU patients are getting for placement of endoscopic tubes. The type of stress ulcer prophylaxis is another matter of debate. A systematic review of 14 trials enrolling a total of 1720 patients in 2013 favored the use of proton pump inhibitors over histamine 2 receptor antagonists in critically ill patients [78]. The former were found to be more effective in preventing clinically significant upper gastrointestinal bleeding. Nonetheless the heterogeneity of the trials included did limit the strength of that recommendation.

Effectiveness of Nutritional Delivery

Once the provision of nutrition has been started at goal, it is equally important to measure the effectiveness of that nutrition. Several ways of assessing caloric use in the critically ill and surgical patient have been described. Updated BMI, 12-h

urinary urea nitrogen, prealbumin, and C-reactive protein (CRP) levels are obtained weekly after recording a baseline measurement and starting nutrition. Indirect calorimetry is also available as required for further assessment. The urinary urea nitrogen serves to estimate the protein need and loss in patients who have a creatinine clearance greater than 50 ml/min. A normal range is 6–24 g/day. A negative result indicates excessive muscle shunting for energy. (Total urinary nitrogen is more accurate in the critically ill, but is less readily available [79]. In addition, spinal cord-injured patients must be excluded because loss is tremendous and ongoing [80]).

C-reactive protein is an acute-phase protein that directly correlates with injury and ongoing inflammatory states. Elevation above 15 mg/dl indicates that the liver is unable to synthesize other types of proteins such as albumin, prealbumin, and transferrin. It therefore can be used to measure whether there is still acute inflammatory response preventing anabolism, appropriate, expected use of nutrients, and healing.

Prealbumin has a 2–4-day half-life, and its level indicates anabolic activity. Normal response during the critical phase would be an increase of 0.5–1 mg/dl/day.

Indirect calorimetry measures expired carbon dioxide to extrapolate energy consumption in the ventilated patient. Patients must be on a FiO_2 of less than 60 % with a PEEP of less than ten. The usefulness of the measurement is apparent for patients where over- or underfeeding would be clinically undesirable based on their known medical comorbidities [81].

Consequences of Inadequate Feeding

Though the precise caloric requirements for critically ill patients is not well defined and is dependent on numerous factors, it is well recognized that adequate caloric intake is important. In a prospective observational study of critically ill patients, an increase of 1000 cal/day significantly reduced mortality, with the most pronounced effects in those patients with a BMI less than 25 or greater than 35 [17]. In a recent study of more than 7000 intubated ICU patients, there was a significant association between the percent of prescribed calories received, and 60-day mortality [82]. Patients receiving more than two-thirds of prescribed calories were less likely to die than those receiving less than one-third of prescribed calories. The optimal percent of prescribed calories was approximately 80–85 %.

Early delivery of adequate calories to critically ill surgical patients, however, can prove challenging. Vasopressor use, bowel in discontinuity after damage control surgery, and ileus can all impede adequate early delivery of feeds. Nutritional adequacy is defined as the actual 24-h caloric or protein intake/prescribed 24-h caloric or protein intake and has been studied in the trauma adult and pediatric populations [83]. For both patient age groups, adequacy was ≤ 60 %. Therefore early placement of feeding access and a focus on the importance of early nutritional delivery are paramount. In fact, adequacy of nutrition in the ICU seems to play an important role in discharge destination. In a recent study by Yeh et al. of critically ill surgical patients, inadequate macronutrient delivery was found to be associated with lower rates of discharge to home [84].

Open abdomens and recent bowel anastomosis are not contraindications to early feeding [85]. In a recent meta-analysis of early versus traditional postoperative feeding in patients with bowel anastomosis, there was a significant reduction in total postoperative complications in patients receiving some type of nutritional support (either enteral feeds or diets) within 24 h of surgery, even if it was provided proximal to the anastomosis [86]. The use of enteral glutamine during shock may also be safe [87].

In an attempt to improve nutritional adequacy, the PEP uP Protocol has been proposed by Heyland et al. [88]. In a single center feasibility trial, enteral feeds were started at 25 ml/h, motility and protein supplements were started immediately, and the target was a 24-h volume of enteral nutrition rather than an hourly rate. If a patient missed feeds, "makeup" feeds were provided. They found a significant improvement in caloric and protein delivery, with no increase in complications.

On the other hand, there are some studies that suggest caution needs to be exerted in intensely feeding certain populations with critical illness. A prospective randomized trial conducted by Braunschweig et al. showed increased mortality in ICU patients with acute lung injury who are provided with more than 75 % of their estimated energy and protein needs per day as non-volitional infusional EN when compared with patients who received standard EN. It was postulated that intense nutrition leads to that effect by interfering with autophagy and altering gut microbiota [89]. These results were not replicated in a separate Australian study and further trials are warranted. [90]

Parenteral Supplementation of Enteral Nutrition

If critically ill patients are not receiving adequate enteral nutrition and adequate delivery of calories and protein is important, the question arises as to whether supplemental TPN should be added until full needs are met by the enteral route. This was recently investigated by Casaer et al. in a prospective randomized multicenter trial [91]. All patients received early EN but were randomized to either early (<48 h) or late (>day 7) parenteral nutrition. Survival was equal between groups but the late parenteral group had fewer ICU infections and a greater likelihood of being discharged alive.

Though the study demonstrated that the early use of supplemental TPN is not beneficial, there were several limitations of the study. The majority of patients were not malnourished at ICU admission, the severely malnourished were excluded, the patient population was that requiring primary cardiac surgery, and approximately half the patients were extubated by day 2, suggesting that those patients who may have benefited from supplemental nutrition were not included in the study. There is a completed pilot study by Wischmeyer et al. that is examining the efficacy of supplemental parental nutrition in under and overweight patients (personal communication). Patients must be candidates for EN but not receiving their nutritional goal on enteral feeds alone. Results of this study are currently being analyzed. However, until the time supplemental TPN is shown to have proven benefit, it is not recommended in the surgical patient when EN can be used.

Complications of Nutritional Support

Refeeding

The refeeding syndrome can occur in any nutritionally deplete individual regardless of the manner in which he or she is being fed. The syndrome is most frequently seen in patients who are alcoholics, have eating disorders, suffer from hyperemesis gravidarum, or who have experienced excessive, rapid weight loss following bariatric surgery. Symptoms are not limited to cardiac arrhythmias, organ failure, and death. The crux of the syndrome is that fat metabolism, which predominated in the unstressed, starved state, now with refeeding, switches to a primarily carbohydrate-based metabolism. The carbohydrate-based metabolism is responsible for a rapid uptake of electrolytes causing intra- and extracellular levels to drop quickly creating disturbances and related effects. Prevention is by recognizing inherent risks and repleting electrolytes before the syndrome can ensue. An additional strategy is to start feeds at one-third to one-half of goal and increase gradually. Electrolytes should be serially checked in high-risk patients.

Non-occlusive Mesenteric Ischemia

There is no decisive data regarding feeding the gut for patients on pressor therapy. Based on primarily retrospective data, it appears that if vasopressors may be safe, though there is no high quality evidence to date. In examining different pressor agents and doses, a norepinephrine dose less than 12.5 mcg/min, utilization of phenylephrine, and the exclusion of dopamine and vasopressin were associated with enteral nutrition tolerance in a large retrospective study [92]. In a small prospective observational study of cardiac surgery patients with circulatory failure (2 or more vasopressor agents utilized and/or mechanical circulatory support), investigators sought to assess the feasibility of providing nutrition via the enteral route [93]. Enteral nutrition was successfully instituted though only 40 % of patients achieved adequate delivery. Complications were identified in 62 % of patients, 46 % of whom developed constipation. There were no reported cases of mesenteric ischemia.

The major concern in feeding patients on vasopressors is the risk of bowel ischemia. A non-occlusive pattern would involve the entire length of the bowel, and, if it were from feeds, would be expected to begin at the site wherever feeds came in contact with the bowel mucosa. For example, if the stomach is the point of nutritional entry, then any non-occlusive bowel necrosis would be expected to involve the stomach, even despite its robust blood supply. Patchy areas may result if the period of ischemia were short. However, the data appear to be lacking for definitive recommendations in such situations. The mortality for fulminant non-occlusive bowel necrosis approaches 50 % [94].

Nutritional Support in Specific Surgical Patients

Pancreatitis

Pancreatitis, though not strictly a surgical disease, demands special attention. There is some debate in the literature of whether post-ligament of Treitz feeding prevents continued inflammation. Placement of endoscopic or push nasojejunal tubes has allowed the patient with pancreatitis to be fed enterally. There are several well-documented populations where outcomes have shown a positive benefit to enteral feeds as compared to nutrition provided by TPN [95, 96]. Enteral feeds are thought to decrease the expression of endotoxin, TNF-a, IL-6 as well as APACHE II scores, pancreatic sepsis and overall mortality in patients with severe acute pancreatitis [97]. Of special interest, is that early EN seems to moderate the excessive immune response without leading to subsequent immunosuppression [98]. Despite previous concern that small bowel enteral feeds would still have some, even if minimal, effect on pancreatic stimulation, this has proven to be unfounded [99]. The time to start of feeds continues to be an area of research and debate. A recent Dutch randomized controlled trial by Bakker et al. did not show any superiority of early nasoenteric tube feeding as compared with an oral diet after 72 h in reducing the rate of infection or death in patients with severe pancreatitis at high risk for complication [100]. Furthermore, the role of glutamine was recently investigated and oral glutamine administered early to patients with pancreatitis was not shown to have any significant effect on gut permeability, degree of inflammation,

infectious complications, or length of ICU or hospital stay. Mortality was also noted to be unaffected [101]. The role of very early nutritional and additive supplementation in pancreatitis continues to be unclear, though the initiation of feeds does not seem to cause any harm to these patients.

Chylothorax/Chyloperitoneum

Although an uncommon phenomenon, chylothorax and even chyloperitoneum do require special attention. While overall this complication is more likely seen as a result of malignancy or operative management of malignancy, they are also seen in the trauma population, after central line placements, with lumbar spine fractures, and iatrogenic. Recommendations include attempting nonoperative management with dietary modification and TPN, chest tube drainage to quantify the volume, followed by surgical ligation if the output continues of 1500 ml/24-h periods or for more than 2 weeks [102]. When the volume of this problem is uncontrollable, TPN or enteral feeds with medium-chain fatty acids seem to be most effective in decreasing the output. Typically elemental formulas are recommended to expedite adequate seal of the lymphatic chain. When conservative treatment fails, there may be a role for percutaneous thoracic duct embolization or percutaneous destruction of lymphatic vessels which are reportedly successful in 70–80 % of cases in controlling the lymphatic leak [103]. These therapies are more popular in Europe but present an alternative route for management. Substantial loss of protein and albumin occurs during the leak and this can lead to significant malnutrition and immunologic derangement if allowed to continue [104, 105].

Enterocutaneous Fistulas

Enterocutaneous fistulas drain bowel content to the atmosphere and are the bane of surgical complication. They are thought to be caused by anastomotic failure and breakdown, intra-abdominal abscesses, foreign body erosion (for example, drains), malignancy, or inflammatory processes, and there is some data that they can be due to prolonged wound vac usage [106, 107]. They additionally can occur without identifiable cause. The biggest problems are damage and excoriation to the skin, loss of electrolytes and fluid with dehydration risk, and challenges in providing effective and usable nutritional support [108]. Spontaneous closure is more likely if the output is low, the surrounding bowel is healthy, and the fistula resulted as a postoperative complication [109]. There is no definitive data in the literature regarding medications or supplements that will decrease fistula output and promote ultimate closure; glutamine, use of TPN with avoidance of enteral nutrition, and specific dressings have all been credited with enabling closure [110–114]. Spontaneous closure does not occur often, and if does not occur, indicates need for planned, delayed, surgical closure [115–117]. Mortality is directly correlated with output volume and additional related complications [109]. High-output fistula is defined as volume loss greater than 500 ml per 24-h period. This fluid contains significant electrolytes, mimicking the makeup of the specific fluid in that part of the gastrointestinal system. These electrolytes must be accounted for and appropriately replaced to prevent dehydration and complications related to specific electrolyte loss [118, 119]. Significant albumin wasting is associated with increased morbidity and mortality [120, 121].

Short Bowel Syndrome

Short bowel is more associated with the clinical outcomes of having insufficient length to perform effective digestion, than defined by the actual length, since there is evidence that the bowel has some ability to adapt function over time [122, 123]. Providing long- and short-chain fatty acids, immunomodulators, and trophic feeds or elemental formulas may play a role in gut adaptation [124–126]. It should be noted that the adaptation of the bowel includes adaptation of each of the enterocytes, overall function, motility, secretion, and absorption [127, 128]. Short bowel implies inadequate length to enable all the necessary components of digestion without the ability to maintain nutritional support. It is a spectrum, with some patients still able to maintain some degree of enteral support. Less than 100 cm of missing length of small bowel is extremely well tolerated; total remaining lengths of less than 100 cm are poorly tolerated and typically require complete replacement of nutrition by the parenteral route [129]. Those with true short bowel are TPN dependent, which of course introduces the risks of line sepsis, intra-abdominal sepsis from gut overgrowth, and bowel disuse. There is also increased cost of the TPN itself and of hospitalization necessary for placement of lines and treatment of infections. The most likely cause of short bowel is from resection, the majority of these cases resulting from resections in childhood [130, 131]. Treatment focuses on nutrition. Pharmacologic treatment includes transit slowing medications (loperamide, diphenoxylate-atropine, cholestyramine, narcotics, pancreatic enzymes), drugs that reduce gastrointestinal secretions (acid-reducing medications, octreotide, clonidine), drugs that provide trophic effect and growth factors (glutamine, teduglutide) as well as drugs to treat small intestinal bacterial overgrowth [132]. Surgical management includes preserving any remaining length, reversing small segments to enhance absorption and motility, and intestinal transplants [133–139]. No surgical intervention has been shown to have overwhelming benefit.

Conclusion

The delivery of early, appropriate nutritional support is a critical component of the comprehensive care of the surgical patient. An understanding of the various options for EN, the indications for enteral versus parenteral nutrition, and the complications of the various modalities of nutrition delivery are fundamental for delivering optimal care.

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