Management options

Enteral nutrition and the critically ill

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Summary

Critically ill patients invariably require nutritional intervention. Traditionally, enteral nutrition has not been widely employed in this patient population. This is due in part to the success of present-day parenteral nutrition, and to difficulties encountered with enteral feeding. Recent evidence has demonstrated that enteral is preferable to parenteral nutrition in terms of cost, complications, gut mucosal maintenance, and metabolic and immune function. Enterally administered nutritional support can and should be utilised as the preferred route of nourishment for the critically ill. The appropriate choice of access and formula, as well as a rational strategy for implementation, should improve the likelihood of success. This article describes the unique features of critical illness as they pertain to nutritional support, the benefits of enteral nutrition, and the obstacles to success, and offers suggestions which may improve the ability to provide nutrients adequately via the intestinal tract.

Keywords: malnutrition, nutrition support, critical illness, enteral nutrition

Critical illness is characterized by significant morbidity and mortality. Disease processes evolve quickly and patient status may repeatedly fluctuate. Often, multiple organ systems are affected. Overall, these patients are among the most challenging for the clinician. Physiologically, critically ill patients exhibit the stress response, which is characterised by hypermetabolism and hypercatabolism. Storage nutrients in the form of fat, carbohydrates and protein are liberated and utilised to fuel the engine of metabolism and provide substrates for tissue repair. Successful recovery is often dependent upon the ability to complete the healing process prior to the exhaustion of fuel. Without the proper administration of nutrients to offset lean body wasting, recovery is rare.

Our expanding ability to administer nutritional support has greatly improved the outcome for patients in the intensive care unit. Unlike the typical hospitalised patient, the critically ill are more difficult to nourish adequately. They tend to have greater requirements for protein and calories, yet characteristics such as fluid overload, hyperglycaemia, and pre-existing medical conditions often complicate the ability to provide sufficient nutrients. Ironically, it appears that the patients who require the most nutritional intervention, are often those least likely to be adequately fed.

Traditionally, enteral nutrition has not been widely employed for this patient population. This is due in part to the success and ease of present-day parenteral nutrition, and to difficulties encountered with enteral feedings. Feeding the critically ill can be challenging, no matter which route of administration is chosen, due to problems with fluid restrictions, metabolic derangements and hyperglycaemia. Clinicians attempting to utilise enteral feeding are further frustrated by patient conditions such as gastric dysmotility, diarrhoea, formula intolerance, and occasionally, by intestinal ileus. Appropriate gastrointestinal access can also be a problem and may require surgical placement.

Over the last few years, the value of gastrointestinal feeding has been redefined. Numerous benefits have been identified and myths concerning its inappropriateness for patients in intensive care units debunked. Additionally, with the proper understanding of the unique environment encountered in the intensive care unit, enteral nutrition can be utilised successfully and its benefits attained. This review discusses the rationale behind the impetus to administer enteral nutrition to the critically ill, the difficulties encountered, and some points to optimise the potential for success.

Metabolic characteristics of critical illness

The critically ill form a unique subset of hospitalised patients. Many of these patients have pre-existing medical conditions that significantly influence their outcome, including heart disease, diabetes, chronic obstructive pulmonary disease, renal insufficiency, and cancer. In many cases, the nutritional status of these patients prior to their admission to the intensive care unit is already severely compromised. In addition, many will go on to develop organ dysfunction as a consequence of sepsis or the inflammatory response. Both the pre-existing illnesses and the acquired conditions will significantly alter the ability to nourish.

A common characteristic of critical illness is the stress response. This process is a cytokine- and hormone-mediated mobilisation of endogenous substrates which are utilised for the stabilisation of organ function, maintenance of immunocompetency and recovery from injury.^{1,2} It is known that the stress response is responsible for many of the metabolic consequences of critical illness, namely fluid overload, hypercatabolism, hypermetabolism, and glucose intolerance.

More than 50 years ago, Cuthbertson and Tilson³ described this phenomenon as a two-phase process. The *ebb* phase occurs immediately after injury and lasts approximately 24–48 hours. During this short-lived period, there is an increase in sympathetic activity and a stimulation of the hypothalamic-pituitary axis.⁴ It is characterised by marked hypometabolism and decreased oxygen consumption.⁴ It is now recognised that these findings

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Metabolic characteristics of critical illness

- hypermetabolism: increased energy expenditure
- hypercatabolism: increased protein breakdown
- hyperglycemia: secondary to insulin resistance, glycogenolysis, and/or gluconeogenesis

are primarily the result of hypovolaemia leading to decreased cardiac output and inadequate oxygen transport to the tissues.⁵ In contrast to the ebb phase, the second, *acute* or *flow*, phase is one of hypermetabolism, catabolism, and increased oxygen consumption.⁶ These mechanisms are thought to be mediated by cytokine release and afferent nerve signals from the injured tissues.⁷ During this period, there is an active liberation of endogenous substrates such as glycogen-derived glucose, skeletal muscle-derived and labile amino acids, and adipose tissue fatty acids.^{1,2,8,9} Increased sympathetic and hypothalamic-pituitary activity stimulate the release of the catecholamines and the counter-regulatory hormones, namely, the glucocorticoids, glucagon, growth hormone, prolactin and aldosterone. These hormones act synergistically to promote lipolysis, muscle protein breakdown, insulin resistance and glycogenolysis.^{2,4,10,11} Since the glycogen stores are limited, this source of glucose is quickly exhausted.¹ The need for readily available glucose will then depend on enhanced muscle protein breakdown to provide amino acids for hepatic gluconeogenesis.^{1,2,4} In response to the rising plasma glucose levels, insulin secretion is increased. However, the hormone's actions on glucose metabolism are greatly restricted by the profound insulin resistance.^{1,4,10}

The net effect of these metabolic pathways is the liberation of peripherally stored substrates to meet the energy requirements of the major organ systems. Each substrate plays an important physiologic role in the stress response. Glucose is an important fuel for the central nervous system, the wound, and the immune system, all of which are metabolically active during stress.^{1,2,6} The fatty acids function to provide energy for cardiac and skeletal muscle, the liver, and many other tissues.^{1,2,6} Although some liberated amino acids are utilised for gluconeogenesis, the majority are required for the synthesis of the acute phase proteins, for thermogenesis, and as precursors for tissue repair.^{1,2,6,9} In states of semistarvation, the ongoing demand for amino acids can lead to marked wasting of lean body tissue. The healthy human body stores approximately 100 g of labile protein nitrogen and is designed to withstand about one week of stress without feeding.¹² In the critically ill, where catabolism is more intense and nutrient stores are likely to be inadequate, storage fuels are more rapidly exhausted. Further protein wasting without supplementation can lead to decreased protein synthesis, organ dysfunction, immunodeficiency, sepsis, and ultimately, death.^{2,4,8}

Recovery, should it occur, is marked by a decrease in metabolic rate, a restoration of appetite, a replenishing of the body energy stores, and a marked rebuilding of the lost lean body mass. The cytokine, catecholamine, and counter-regulatory hormone influences subside, and insulin again becomes the primary nutrient regulatory hormone. There is also a significant uptake of amino acids in the muscle for synthesis and glucose for the production of glycogen and triglycerides.

Benefits of enteral nutrition

- prevents intestinal mucosal atrophy
- supports the intestinal immunologic shield to invasion by pathogens
- attenuates the hypermetabolic response to injury
- less costly than parenteral nutrition
- may improve outcome for critically ill or traumatized patients

Benefits of enteral nutrition for the critically ill

In the intensive care unit, parenteral and enteral nutrition should not be mutually exclusive, but rather, complementary. Both modalities offer unique benefits and have inherent limitations. For many reasons, total parenteral nutrition (TPN) is still considered the gold standard for the nutritional support of the critically ill. It offers the best means of insuring both macro- and micronutrient delivery and is an excellent modality for correcting metabolic derangements. For the fluid-overloaded patient, it can be utilised as a vehicle to deliver intravenous medications without increasing the volume load and can be concentrated significantly, while still providing adequate nutrition. Since most patients in the intensive care unit already have an indwelling central venous catheter and many hospitals provide TPN as stock solutions, prescribing TPN can be quite simple. Occasionally, it also represents the only nutritional option available for patients with severe gastrointestinal dysfunction.

Unfortunately, TPN is significantly more expensive than enteral nutrition and requires a dedicated central venous catheter or a dedicated lumen of an indwelling multilumen catheter. It also has a higher incidence of serious complications, not exclusively related to the central venous catheter.^{13,14}

The reliance on TPN often coincides with gastrointestinal deprivation which can lead to a breakdown of the gut barrier to bacterial invasion, and possibly, bacterial translocation.^{15,16} To prevent the invasion of microbes into the systemic circulation, the small intestine relies on both a physical barrier and an immunologic shield. The physical barrier is created by the tight junctions between intact epithelial cells, gastric acid, digestive enzymes, mucus production, intestinal motility and a normal bacterial flora. The intestinal immune system, often referred to as the Gut-Associated Lymphoid Tissue

(GALT), consists of cells such as lymphocytes and macrophages situated throughout the intestinal wall and the production of the antibody IgA which is secreted into the lumen and prevents the adherence of microbes to the mucosa, a step necessary for invasion.¹⁷ Conditions in the intensive care unit may predispose patients to translocation. Gastric acid is commonly neutralised to prevent gastritis and ulceration. With the use of sedation and narcotics, there is a tendency toward decreased intestinal motility leading to luminal stasis. Antibiotic usage, commonplace in the intensive care unit, leads to the bacterial overgrowth of pathogens. Without enteral nutrient stimulation, the mucosa atrophies,^{15,18} causing increased permeability.¹⁹ The GALT system also atrophies.²⁰ The combined effect of these changes is a weakening of the gut defence to invasion. The resultant translocation is believed to be associated with and maybe even the underlying cause of the nosocomial sepsis and the multisystem organ dysfunction seen with critical illness.^{16,21} However, the actual relationship between translocation and nosocomial sepsis remains controversial.

While present-day parenteral nutrition can substitute nutritionally for the interruption of oral nutrition, gut mucosal atrophy and immune dysfunction are not prevented.^{15,22,23} A stream of luminal nutrients seems to be essential. It has also become apparent that certain nutrients are indispensable for maintaining the intestinal barrier to microbes. Both the epithelial cells of the mucosa as well as the lymphoid cells of the GALT system require specific nutrients for growth and normal function. These nutrients (glutamine, arginine, nucleotides, and short chain fatty acids) are not found in standard parenteral solutions or even in most enteral formulations.

Enteral nutrition has recently gained new popularity. A large body of literature describes benefits in terms of cost, lower complication rates, and favourable effects on metabolic and immune function. The infusion of nutrients intraluminally has been shown to be important for maintaining gut mucosal integrity, a key factor for organism homeostasis and immunologic competence.^{24,25} The provision of nutrients intraluminally has also been shown to attenuate the stress response. The acute phase reactive protein secretion is blunted and higher levels of synthetic proteins are seen.²⁶ Lowry demonstrated lower counter-regulatory hormone and C-reactive protein levels in subjects fed only enterally after exposure to endotoxin versus those fed parenterally.²⁴ The net effect is a lower rate of catabolism and energy expenditure.

Improvements in outcome have also been reported. Studies including those by Moore and co-workers have demonstrated in trauma patients improved nitrogen balance, lower infection rates, and improved wound healing with early enteral nutrition.^{13,27} In similar work, Kudsk *et al* demonstrated a significantly lower infection rate in critically ill trauma patients who were provided nutrition enterally.¹⁴ A recent meta-analysis of eight published studies by Moore's group also concluded that high risk surgical patients fed enterally had reduced septic complications compared with patients given parenteral nutrition.²⁸ Other benefits seen with enterally administered nutrition include improved gallbladder contraction leading to a reduction in the likelihood of gallstone formation and acalculous cholecystitis, increased pancreatic stimulation with a reduction in sluggish secretion and functional insufficiency, and improved gut healing after surgical anastomosis.²⁹

Most intensive care physicians support the early institution of enteral nutrition for critically ill patients. In general, it is believed that the early initiation of intestinal feeding attenuates the stress response, decreases the loss of lean body tissue from catabolism and improves patient outcome. Unfortunately, these theories have never been conclusively proven to be true. While a number of investigational studies have demonstrated improved metabolic parameters and patient outcome when comparing early enteral nutrition to early parenteral feeding, there are few published reports that evaluate the differences between early and delayed enteral nutrition.

Graham *et al* compared early jejunal feeding (within 36 hours of injury) to late gastric feeding in head-injured patients and demonstrated a reduced incidence of bacterial infections and hospital stay in the early fed group.³⁰ In contrast, Eyer and co-workers evaluated the effects of early enteral feeding (within 24 hours of injury) versus initiating feeding after 72 hours and found that early feeding did not blunt the stress response or alter patient outcome.³¹

Despite the lack of strong clinical evidence to support early gut feeding, most intensive care physicians would support initiating enteral nutrition within the first 48 hours of injury or surgery in a stable patient since it is usually well-tolerated and may improve outcome.³²

Obstacles to providing enteral nutrition for the critically ill

- fluid restriction
- acid–base disorders
- electrolyte abnormalities
- access difficulties
- formula intolerance

Obstacles to providing enteral nutrition for the critically ill

Unfortunately, administering enteral nutrition to the critically ill can be difficult. It is far easier to prescribe TPN through an indwelling catheter than to contend with the problems associated with enteral feeding. Most of these difficulties fall into two main categories, access problems and feeding intolerance. In addition, enteral feeds are often withheld for diagnostic testing or surgery so that patients commonly receive only a fraction of the required feeding. It is therefore no surprise that TPN-fed patients tend to receive more nutrients per kilogram of body weight than those given enteral nutrition.¹⁴ While these concerns frustrate clinicians, many have reported good success in providing enteral nutrition in the intensive care unit.

ACCESS PROBLEMS

Many critically ill patients will manifest some degree of gastric dysmotility. Potential aetiologies include sepsis, use of hyperosmolar feeds, electrolyte abnormalities, gastritis, peptic ulcer disease, chemotherapy, medications and comorbidity such as diabetes mellitus.³³ Interleukin-1, a cytokine released during stressed states, has been shown to delay gastric emptying when administered to rats fed a liquid diet.³⁴ Feeding into the stomach in these circumstances was believed to increase the risk of regurgitation and aspiration. However, recent studies have failed to support this notion.^{35,36} Patients with normal gastric emptying but diminished ability to protect their airway, such as those with neurologic disorders may also require postpyloric tube placement.

The appropriate gastrointestinal access can also be challenging to obtain and maintain. Placement may require endoscopic, fluoroscopic or even surgical assistance. Often, after successful access has been obtained, feeding tubes occlude, dislodge or are inadvertently removed. Tubes commonly occlude, particularly if used to deliver crushed medications or when not carefully flushed clear with water after the feeding has been discontinued.

FEEDING INTOLERANCE

Intolerance may present as vomiting, abdominal pain and/or bloating, paralytic ileus or diarrhoea. While postpyloric tube placement alleviates gastric emptying problems, the other manifestations of intolerance are more difficult. Diarrhoea, a common problem for the critically ill, has a number of potential causes. These include medications, such as antibiotics, antacids, and sorbitol, infections such as *Clostridium difficile* and other enteric pathogens, formula osmolality and fat content, infusion rate, malabsorption, lactose intolerance, and hypoalbuminaemia.^{37–41} Many studies have evaluated the contribution of some or all of these factors. There seems to be no true consensus concerning the relative importance of each. In all likelihood the cause of diarrhoea is usually multifactorial. If the aetiology can be elucidated, the offending agent can then be addressed. For instance, *C. difficile* colitis can be treated, sorbitol-based medications may be eliminated, and the choice of enteral formula being used can be reconsidered.

The contribution of formula osmolality in feeding intolerance and diarrhoea is also controversial. Traditionally, hyperosmolar feed has been thought to be difficult to tolerate. However, Borlase *et al* have demonstrated that even formulas with an osmolality of 630 mOsm, about twice that of serum, can be successfully administered.³⁷ Presently most commercial products are isosmolar or slightly hyperosmolar relative to serum.

Formula options

Currently there is no consensus in the literature regarding the optimal choice of enteral product for the critically ill. As patients and illnesses vary widely, it could be said that the formula of choice is that which the patient best tolerates. Fortunately, there are many commercial enteral products available which offer a vast array of options to fit the needs of most critically ill patients. These products differ in protein source and form, fat content and source, caloric density, and osmolality. Carbohydrate source is less important since most commercial formulas are lactose free and most other types of carbohydrate are generally well tolerated.

Formulas are usually categorised based on their contents and applications (table). These include polymeric (encompasses standard, high calorie/high nitrogen, fibre enriched, and disease specific), peptide, special nutrient, and modular. For the critically ill, the choice is usually between a low fat, peptide formula or a standard formula that contains intact protein and provides approximately 30% of its calories as fat. Some studies have demonstrated improved tolerance with peptide (+/- low fat) formulas.^{38,42} Meredith and co-

Table 1 Selected enteral formulas for critically ill patients

Formula	Calcium/ml	Protein (g/l)	Fat (%)	mOsm	Vol to meet US RDA
<i>Peptide formulas:</i> partially hydrolysed protein source, may be better absorbed and utilized during critical illness, the early postoperative period and with malabsorptive conditions					
Vital HN ¹	1.0	42	10	500	1500
Reabilan HN ²	1.3	58	35	490	1875
Peptamen VHP ³	1.0	62.4	35	430	1000
<i>Standard formulas:</i> intact protein with moderate fat content					
Osmolyte HN ¹	1.06	44.4	30	300	1321
Promote ¹	1.0	62.5	21	300	1250
Sustacal ⁴	1.01	61	21	650	1060
<i>High nitrogen/high calorie formulas:</i> hyperosmolar, high fat content formulas useful for fluid restricted and/or hypermetabolic patients					
Ensure Plus HN ¹	1.5	63	30	650	947
Two Cal HN ¹	2.0	84	41	690	947
Isocal HCN ⁴	2.0	75	46	690	1000
<i>Fibre-enriched formulas:</i> polymeric formulas with intact protein and added fibre					
Jevity ¹	1.06	44	30	310	1321
Compleat ⁵	1.07	43	31	300	1500
Ultracal ⁴	1.06	44	37	310	1250
<i>Special nutrient formulas:</i> standard or peptide formulas containing specific nutrients thought to be conditionally essential during critical illness					
Alitraq ¹	1.0	53	14	575	1500
Impact ⁵	1.0	56	25	375	1500
Perative ¹	1.3	67	25	385	1155
<i>Disease-specific formulas:</i> modified substrate form and contents to meet restrictions of specific organ dysfunction					
Nepro ¹	2.0	70	43	635	947
Hepatic Aid II ⁶	1.2	44	28	560	*
Pulmocare ¹	1.5	62.5	92	520	1420

This list represents only a sample of the many commercial products on the market. A number of equally useful formulas are also available. 1=Ross, 2=Elan Pharma, 3=Clintec, 4=Mead Johnson, 5=Sandoz, and 6=Kendall McGaw. RDA: recommended daily allowance; *, not nutritionally complete

Manifestations of formula intolerance

- abdominal bloating and distention
- abdominal pain
- diarrhoea
- high gastric residuals
- paralytic ileus

workers demonstrated better hepatic protein responses in trauma patients when fed a peptide formulation.⁴² In contrast, other studies have had similar success with standard whole protein, high fat formulas.⁴³⁻⁴⁵ While small peptides have been shown to be better absorbed and utilised by the intestinal mucosa, it would seem that whole protein can be well tolerated in most cases in which the intestine is normal. The type and quantity of fat may also play a role in determining success. Long chain triglycerides (LCTs) such as those commonly seen in most commercially available enteral products require significant digestive effort as well as sufficient mixing with bile and pancreatic lipase to get absorbed into the intestinal lymphatics. Formulations high in LCTs may cause diarrhoea.⁴⁶ On the other hand, medium chain triglycerides (MCTs) require less digestive effort and can be absorbed directly into the portal circulation.⁴⁷ Therefore, formulas containing small amounts of LCTs and/or utilising MCTs as the predominant fat source, may be better choices for patients with gastrointestinal dysfunction.

For patients requiring extended enteral feeding, it is wise to choose an economical formula suitable for extended use. Excellent choices include standard, fibre-enriched formulas and high calorie/high nitrogen products. For patients with potential malabsorption secondary to the prolonged interruption of enteral nutrition, it may be best to introduce these formulas after tolerance has been established to peptide or standard isotonic feeds. The high calorie/high nitrogen formulas do require significant digestion but may be useful for the fluid-restricted patient. In these cases, postpyloric administration is strongly encouraged since the high fat content and hyperosmolality may contribute to gastric retention. Fibre-containing products may also be introduced in the intensive care unit setting; tolerance needs to be established gradually to avoid the associated side-effects including gas production and abdominal bloating. Two recommended strategies are offered. Either begin with a formula containing a lower fibre content or use a mix of a high-fibre formulation and a standard fibre-free feed. When tolerance is established, the patient can be placed on a high-fibre formula.

In the past few years, disease-specific products have been introduced. These are predominantly polymeric formulas in which the macronutrients, free water, and electrolytes have been modified to accommodate the specific requirements of certain disease states such as liver failure, renal insufficiency, intestinal malabsorption, diabetes and respiratory failure. Renal failure formulas are useful as they do not significantly increase the need for haemodialysis and are relatively concentrated. Hepatic failure formulas contain di- and tripeptides as well as higher concentrations of branched chain amino acids and lower concentrations of aromatic amino acids. Both of these types of formula are relatively low in protein. Unfortunately there is little evidence demonstrating improved outcome with the use of these formulations. In addition, for the critically ill where higher nitrogen requirements exist, standard, nondisease-specific products may be necessary.

Respiratory compromise may be complicated by excess carbohydrate administration. Formulas with less carbohydrate and greater fat content may be useful for decreasing carbon dioxide production thus decreasing the work of breathing. However, for most patients, the avoidance of overfeeding with a standard product may be sufficient.

Modular enteral products can be used to augment specific macronutrient requirements at a sensible cost. Specific products exist to provide additional protein, carbohydrate and fat to enteral formulas. The use of modular products greatly expands the flexibility of standard feeds to better support a wide variety of patients.

The newest enteral products have been supplemented with nutrients such as glutamine, arginine, nucleotides, fish oil and MCTs for their theoretical benefits. These nutrients have been shown to be required in greater quantities during critical illness. Preliminary studies have demonstrated a decreased incidence of sepsis and improved outcome for the critically ill with the use of these formulations.^{48,49} Two studies have reported reduced septic complications and hospital length of stay in critically ill cancer patients after major surgery when given a formula which contained arginine, fish oil and nucleotides.^{49,50} A recent multicentre trial in critically ill patients by Bower and colleagues also demonstrated a decreased incidence of acquired infections and shortened hospital stay with the same enteral formula.⁵¹ In the near future, these formulations may become the standard of care in intensive care units.

Keys to success

With so many obstacles to overcome, it is paramount to have a good game plan to maximise the potential for successful utilisation of enteral nutrition in the intensive care unit. While certain principles may hold true in most cases, following a rigid protocol is undesirable. A good game plan should allow for flexibility and creativity. Not all critically ill patients will respond in a similar fashion. What might succeed for the majority may fail miserably for the minority. Equally important is persistence. A setback should be viewed only as a temporary situation requiring reassessment of the patient's status and possible reconsideration of the present formula, rate, or access.

Upon entry into the intensive care unit, all patients should be evaluated for placement of enteral access. The choice of access must be based upon

Figure 1 Algorithm for determining appropriate enteral access for the critically ill

Figure 2 Nasoenteric feeding tube

Enteral feeding in the critically ill: keys to success

- carefully select the most appropriate enteral access (usually postpyloric)
- initiate enteral feeding early (usually within 48 h)
- start formula at a low rate (10–25 ml/h)
- increase formula rate based on the patient's condition and the duration of time that oral nutrition has been interrupted

the particular needs of the patient (figure 1). In most cases, postpyloric placement is desirable. The majority of patients requiring a laparotomy should have a jejunostomy tube placed at that time. While needle catheter jejunostomies have been successful for elemental feeds,^{27,52} placement of large bore rubber catheters such as Red Robinson and T-tubes may be more reliable.^{43,53} These are usually 'witzled' or 'stammed' in place and secured to the abdominal wall. For patients who are not scheduled for abdominal surgery, consideration should be given to determine whether the feeding tube will be temporary, of long duration but possibly not permanent, or needed permanently. Those patients who are deemed likely to depend upon tube feeding for an extended period of time should be considered for percutaneous or operative jejunal access. A limited laparotomy can be performed solely for placement of the jejunostomy tube. This can be done quickly, through a small incision and is relatively well tolerated. Currently, techniques even exist for laparoscopic jejunal tube placement. If surgery is contraindicated, or for those patients who are considered likely to recover in the near future to the point of nutritional self-sufficiency, a thin, flexible naso-jejunal tube can be placed (figure 2). A number of techniques have been described to improve passage into the duodenum. If migration through the pylorus is unsuccessful, endoscopic or fluoroscopic guidance may be necessary. In some cases, the use of metoclopramide or erythromycin which stimulates gastric motility can aid in the successful migration of the tube postpylorically.^{54,55} If access to the jejunum proves impossible in a patient who is not a candidate for surgical placement, every attempt must be made to establish gastric feeding. This may require a low fat (LCT) formula, a slow increase in infusion rate and even the use of motility-stimulating agents such as metoclopramide, cisapride or erythromycin.

Once reliable access has been established, enteral feeding should be initiated in a timely fashion. There is good evidence to suggest that the intestinal mucosa begins to atrophy within 24 hours of nutrient deprivation. The longer the delay, the greater the risk of translocation and formula intolerance. While it would be optimal to commence feeding within hours of admission to the critical care unit, this may not always be practical or desirable. Patients should never receive tube feeding if they are haemodynamically unstable or require inotropic support to maintain blood pressure. Should intestinal hypoperfusion exist, enteral feeding could lead to pneumatosis intestinalis or bowel infarction.³² In addition, the first few days of intensive care unit admission are often interrupted by tests, procedures, and other activities. The initiation of enteral nutrition can always be delayed until the dust settles. As briefly discussed above, the selection of the enteral formula which will maximise the potential for success has not been definitively agreed upon in the literature. The choice is generally between a peptide formula with low fat or supplemented with MCT versus one with intact protein which provides approximately 30% of its calories as LCT. One acceptable strategy is to begin all patients on the peptide formula and after good tolerance is established, consider switching to the intact protein mix. A second option would be to attempt to use the intact protein formula first and substitute the peptide formulation if intolerance presents.

Formula infusion rate can also influence ultimate success. While there are many recommendations in the literature concerning how rapidly to increase the rate of infusion, one simple principle should be mentioned. The initial rate should be low, such as 10–25 ml/h and the rate of increase should be inversely proportional to the duration of nutrient deprivation and the degree of intestinal dysfunction. In nearly all cases, formulas do not need to be diluted unless there is also a need to administer supplemental free water. Most formulas are iso- or mildly hypertonic relative to serum. As previously stated, even formulas as hyperosmolar as 630 mOsm/l have been shown to be well tolerated when properly administered.³⁷

One final issue to discuss is how to treat intolerance when it develops. In most cases, intolerance can be overcome. It should not necessarily lead to abandonment of enteral nutrition. If the problem is due to high gastric residuals a few simple maneuvers can be attempted. First, reassess tube position with an X-ray. It may be coiled in the oesophagus or, if it was initially positioned postpyloric, it may have pulled back into the stomach. Consider switching the formula to one that is lower in fat. One might also try improving gastric emptying with medications such as erythromycin, cisapride, or metoclopramide. In most cases, stopping the infusion for a few hours then resuming at a lower rate and increasing slowly will be effective. If intolerance manifests as diarrhoea, one must first rule out treatable aetiologies such as those related to infections or medications. Stool output can be decreased by adding fibre to the feed or slowing transit time with

Summary points

- nutrition support is an important adjunct to critical care
- early enteral nutrition is the preferred route for feeding critically ill patients with functional gastrointestinal tracts but may be difficult to provide
- postpyloric tube placement may be necessary due to poor gastric emptying
- with careful formula selection, enteral feeding can usually succeed, even in critically ill patients

opiates or codeine. If patients develop abdominal pain, bloating, or dilated bowel on X-ray it is probably best to hold tube feeding until the condition resolves. With all cases of intolerance, one must reassess the patient's clinical condition. In many circumstances, the sudden development of intolerance after a period of successful enteral feeding is probably due to a change in clinical status such as a new episode of sepsis, etc.

Conclusion

There is little debate that enteral nutrition is difficult to institute in most critically ill patients. Many obstacles have to be overcome. Yet the many benefits offered to the patient who is fed enterally more than compensates for the difficulties in delivery. Can enteral nutrition be successfully utilized in the critically ill? From the published reports in the literature and personal experience, the conclusion is an unconditional yes. All that is necessary is an understanding of the potential problems to be encountered and the many treatment options in the nutritional armamentarium to combat these issues.

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