42

Enteral Nutrition

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Introduction

Historically, enteral feeding can be traced back to ancient Egypt and Greece, where nutrient enemas were used when patients were unable to take oral nutrition. Various combinations of wine, milk, broth, grains, and raw eggs were used with limited success.[1] Rectal delivery of nutrients was continued up until the early 1900s despite lack of supportive benefit. In fact, President James Garfield was given nutrient enemas every 4 hours for 79 days following his attempted assassination until his death.[1]

The first reports of nutrient provision through feeding tubes into the esophagus were in 1598, when an enteral feeding tube was fashioned from eel skin. In 1790 John Hunter initiated the modern era of gastrointestinal (GI) access with his reports of tube feeding the stomach.[1] Up until this time, nutrient mixtures were delivered by gravity force limiting flow rate consistency. The first stomach pump was invented in the 18th century, allowing for consistent enteral nutrient delivery as well as gastric irrigation and emptying.[1] Tubes remained very primitive and uncomfortable until rubber was developed, thus leading to the evolution of the currently available selections. In 1910 Max Einhorn began feeding the duodenum through a rubber tube when gastric access was not feasible, claiming that rectal feeding was unacceptable.[1] The implementation of orojejunal tube feeding in surgical patients implemented by Ravdin and Stengel followed in 1939. In 1950 the use of polyethylene tubes was described with gastric and jejunal tubes 27 inches and 6 feet in length, respectively. With these tubes came the introduction of the feeding pump to deliver the formulation.[1]

Experimentation with the enteral formulations began in the early 1900s with the introduction of the chemically defined or "elemental" diet. The late 1950s through the 1970s marked the space age and the beginning of space diet research. These chemically defined diets were investigated in both animals and healthy humans to produce a low residue diet that would decrease fecal output during space travel. In the late 1960s chemically defined diets were first reported being used in critically ill surgical patients.[1] Since that time, enteral formulations have undergone extensive modification and now exist for nearly every metabolic disease state.

TABLE 42.1

Immune Benefits of Enteral Feeding

Improved mucosal integrity
Enhanced glycemic control
Normalization of GI flora
Preserved GALT
All epithelial surfaces benefit
common mucosal immune hypothesis
Increased secretory IgA

GI: gastrointestinal; GALT: Gut associated lymphoid tissue

Rationale and Benefits

In most major patient care centers enteral nutrition is the preferred route of nutrient delivery. Parenteral nutrition is substituted only if safe access is unavailable or unsuccessful. Extensive review of the numerous benefits of enteral nutrition is beyond the scope of this section and is only briefly addressed. Available reviews provide more extensive background in these areas.[2-5]

One proposed benefit of enteral nutrition is that it is more physiologic than parenteral nutrition. The gut and the liver process enteral nutrients prior to their release into systemic circulation (first pass). When compared to parenteral nutrition, enteral nutrition positively influences nitrogen balance,[6,7] serum protein levels,[5,8,9] and the metabolic response to stress.[2-5,10,11]

Another benefit of enteral nutrition is its affect on the immune system (Table 42.1). The lack of GI stimulation by enteral nutrients may promote gut mucosal atrophy. This may lead to increased intestinal permeability potentially leaving the gut vulnerable to bacterial translocation. Enteral nutrition provides maintenance of the gut-associated lymphoid tissue,[12,13] maintenance of the normal GI flora,[14-16] and a lowering of infectious complications.[13,17-19]

Enteral nutrition is generally less expensive than parenteral nutrition.[20,21] The lower total cost includes factors such as the cost of enteral formulations, cost of equipment used for formula preparation and administration, and cost of personnel specialists. The delivery of enteral nutrition has been shown to be safe in stable as well as in most critical patients.[17-19,22]

Indications/Contraindications

Enteral nutrition is indicated for patients with access to an adequately functional GI tract and whose oral nutrient intake is insufficient to meet estimated needs. Specific conditions for which enteral nutrition is indicated are found in Table 42.2. Although enteral nutrition is the preferred route of nutrient delivery, it is not innocuous and there are some contraindications to its use (Table 42.3). It is not always clear when enteral nutrition will be tolerated. If the individual's needs are not met enterally, parenteral nutrition may be implemented for either full nutrient provision or concurrently with the enteral delivery to provide the balance of nutrients not tolerated.

Enteral Access

Route of administration and type of access for tube feedings are usually determined by the expected length of therapy (Figure 42.1), risk of aspiration (Table 42.4), and local

TABLE 42.2

Enteral Feeding Indications

Hypermetabolism	*Oncologic Disease*
Postoperative major surgery	Chemotherapy
Trauma	Radiotherapy
Sepsis	Neoplasms
Burns	
Organ Transplantation	
Neurologic Disease	*Psychiatric Disease*
Cerebrovascular accident	Anorexia nervosa
Dysphagia	Severe depression
Head trauma	
Demyelinating disease	*Organ System Failure*
Neoplasm	Respiratory failure (ventilator dependence)
Gastrointestinal Disease	Renal failure
Short bowel syndrome (if remaining bowel has sufficient absorptive capacity ~50-100 cm and intact ileocecal valve)	Cardiac failure (cardiac cachexia)
	Hepatic failure
Inflammatory bowel disease	Multiple organ system failure
Enterocutaneous fistula (<800 mL output/day)	Comatose state
Pancreatitis	

TABLE 42.3

Enteral Feeding Contraindications

Bowel obstruction
Persistent intolerance (e.g., emesis, diarrhea)
Hemodynamic instability
Major upper GI bleeds
Ileus
Unable to safely access
Relative Contraindications
Significant bowel wall edema
Nutrient infusion proximal to recent GI anastamosis
High output fistula (>800 mL/day)

expertise. Nasoenteric or oroenteric tubes are generally used when therapy is anticipated to be of short duration (i.e., <4 weeks) or for interim access before the placement of a long-term device. Long-term access requires a percutaneous or surgically placed feeding tube.

Nasoenteric Access

Multiple methods exist for gaining enteral access (Table 42.5), all of which carry various degrees of expertise, risk, and expense. The nasoenteric tube is the most commonly used method of enteral access. It can be inserted into the stomach, duodenum, or jejunum. Since these tubes have low complication rates, are relatively inexpensive, and are easy to place, they are used most often for short-term use. The most common complications are tube malposition and dislodgement.

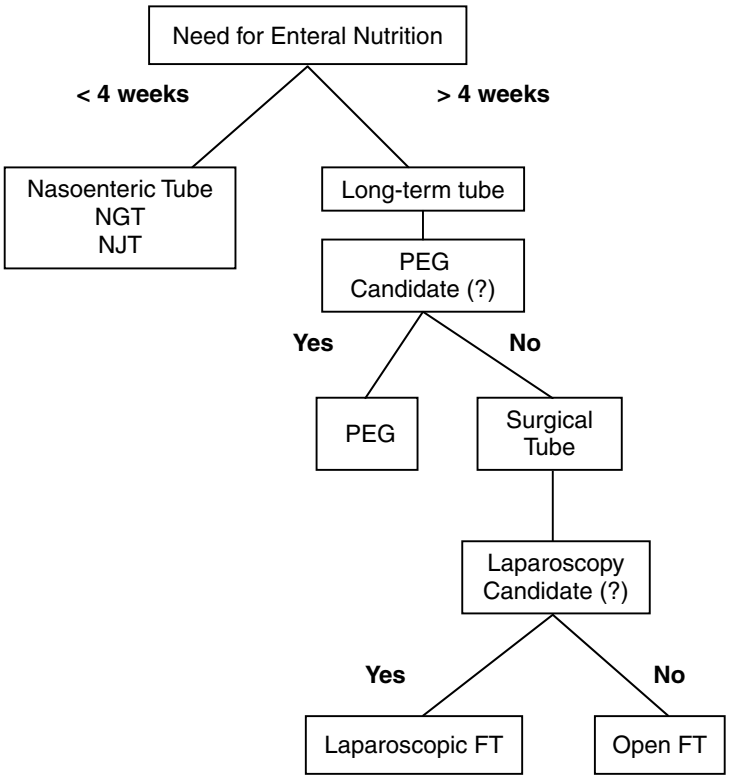

FIGURE 42.1
Enteral access decision tree.

TABLE 42.4

Risk Factors for Aspiration

Altered mental status with inability to protect airway
Swallowing dysfunction
Central (CVA)
Local (vagal disruption, trauma)
History of aspiration
Severe gastroesophageal reflux
Gastric outlet obstruction
Gastroparesis
Patient position restrictions (supine versus semirecumbent)

It is often desirable to place tubes beyond the pylorus in patients with delayed gastric emptying or absent gag reflex to potentially decrease the risk of aspiration. Positioning a nasoenteric tube into the small bowel is much more difficult than positioning into the stomach. Transpyloric tubes can be placed intraoperatively, at bedside, or with endoscopic or fluoroscopic guidance. Intraoperative placement of a nasoenteric tube involves manual manipulation during the surgery; however, this is not common practice, as it requires open laparotomy. Spontaneous placement of a nasoenteric tubes involves advancing the tube into the stomach and allowing it to migrate independently into the small bowel. This technique is not very successful in hospitalized patients, especially the critically ill, due to motility derangements. Several bedside manual methods using special placement techniques, weighted versus non-weighted tubes, pH sensor tubes, prokinetic agents,

TABLE 42.5

Methods of GI Access

Nasoenteric Feeding Tubes	*Percutaneous Feeding Tubes*
Spontaneous Passage	Percutaneous Endoscopic
Bedside — prokinetic agent	Gastric (PEG)
Active Passage	Gastric/Jejunal (PEG/J)
Bedside — assisted	Direct Jejunal (DPEJ)
Endoscopic	*Laparoscopic*
Fluoroscopic	Gastrostomy
Operative	Jejunostomy
	Surgical
	Gastrostomy
	Jejunostomy

magnets, and bioelectrical detection devices have been reported, all with similar success rates (~85%).[23-29]

Do to lack of universal success in manual placement of nasoenteric tubes, fluoroscopic or endoscopic guidance is often sought. If portable equipment is not available, both of these techniques require patient transport to the endoscopy or radiology suite, which may not be feasible for critically ill patients. Fluoroscopic techniques of nasoenteric tube placement involve manipulation of the tube with a long guide wire. Endoscopic techniques include use of a guide wire as well as a "drag" method. In the drag method, a gastrically placed tube is grasped with a snare or biopsy forceps and dragged with the endoscope into the duodenum or farther, and then released. All risks of endoscopy accompany these methods including dental injury, pharyngeal or esophageal injury, gastric bleeding, perforation, and risk of aspiration with the use of intravenous sedation.[30] Both fluoroscopic and endoscopic placement methods are ~85 to 95% successful in obtaining postpyloric feeding tube placement. Although placement of postpyloric feeding tubes using endoscopic, fluoroscopic, and manual techniques may be successful, these tubes are frequently dislodged. Repeated tube insertion increases risk and costs of these access methods. For this reason, patients requiring long-term enteral nutrition should receive more permanent access.

Gastrostomy

Gastrostomy is the most common method for long-term enteral access since it eliminates nasal irritation, psychological stress, and requirement for an infusion pump, as complex formulas may be given as boluses. Gastric tubes, due to their large diameter, can serve many other functions besides feeding, including gastric decompression, gastric pH monitoring, and medication delivery. Insertion can be via laparatomy, laparoscopy, endoscopy, or fluoroscopy.

Permanent gastric placement can be obtained either by surgical procedures (laparotomy or laparoscopically) or by nonoperative procedures. Percutaneous endoscopic gastrostomy (PEG) is the most popular nonoperative procedure for obtaining permanent gastric access. Gauderer et al.[31] first described the procedure in 1980, and despite some modifications, the basic technique used by most endoscopists is similar. Compared to surgically placed tubes, PEGs are less costly, have decreased procedure-related morbidity and mortality, usually do not require general anesthesia, and allow enteral feeding to be initiated

TABLE 42.6

PEG Indications and Contraindications

Indications	*Contraindications*
Long-term access (>4 weeks)	No stomach
Decompression	Unable to scope
Swallowing dysfunction	Hemodynamic instability
Neurologic event precluding swallowing	Coagulation disorders
Tracheoesophageal fistula	Obstruction
	Portal hypertension, esophageal varicies, ascites
	Relative Contraindications
	Peritoneal dialysis
	Prior upper abdominal procedures
	Pregnancy
	Morbid obesity

quickly.[30-36] Indications as well as contraindications for a PEG are described in Table 42.6. Complications of PEGs include dislodgment, bleeding, tube site infection, intra-abdominal leak, site leak, and persistent gastric fistula.[30]

Jejunostomy

The advantage of percutaneous tubes is less apparent when small bowel feeding is required, owing to the high failure rate of PEG tubes with a jejunal extension (PEG/J). While a PEG/J tube is beneficial in the acute care setting when a critically ill patient requiring long-term access is intolerant of gastric feeds, they are not very practical for long-term use. Long-term failure of the jejunal extension is attributed to its small lumen leading to frequent clogging, as well as separation of the inner PEJ tube from the outer gastrostomy tube.[37] For these reasons as well as the expertise required to place the jejunal extension, surgical placement of jejunal tubes is often preferred for long-term jejunal access.

Several choices are available for intraoperative feeding jejunostomy placement. The needle catheter jejunostomy (NCJ) is a quick and easy method that involves inserting a small catheter into the lumen of the jejunum proximal to the ligament of Treitz. The advantage of an NCJ is that nutrients can be administered almost immediately and the catheter can easily be removed when it is no longer needed. Unfortunately, the small lumen of the catheter occludes more readily than larger-bore feeding tubes. Catheters originally designed for other uses have been adapted for jejunal feeding, with the red rubber catheter most frequently used.

Jejunal access has also been obtained by direct percutanous endoscopic jejunostomy placement (DPEJ).[38] This method is similar to PEG placement except that the endoscope is passed through the duodenum, past the ligament of Treitz, into a loop of jejunum adjacent to the abdominal wall. A regular pull-through PEG tube is used for access. This procedure is technically more difficult than a PEG due to the peristaltic action and narrow lumen of the jejunum, but this procedure has many advantages over a PEG/J such as a decrease in clogging due to the use of a larger diameter tube, and decreased migration or kinking. As this is a fairly new procedure, data on long-term complications are lacking.

Enteral Formulas

The increase in the use of enteral over parenteral nutrition in the past few decades has led to a rapid expansion in the number of commercially available enteral nutrition products. These products do not currently require Food and Drug Administration (FDA) approval for their proposed clinical implications, and fall under the category of food supplements. Nearly every major enteral formula company in the United States today carries a similar line of products. These formulas can be categorized as oral supplements, standard tube feedings, high-protein tube feedings, and disease-specific products. Table 42.7 provides an overview of these categories, specifying the types of macronutrients and physical properties of the select formulas. Since these products do not require the rigorous FDA examination prior to their marketing, it is left up to the experienced nutritionist to decipher the product indications. The optimal selection and administration of a formula requires a thorough knowledge of normal and abnormal digestive and absorptive physiology and formula composition. The physical form and quantity of each nutrient may determine the extent of absorption of and tolerance to the formula (e.g., long-chain versus medium-chain triglyceride). The following discussion provides an overview of select macronutrients found in these products, with related supportive research as available.

Macronutrients

Carbohydrate

Among formulas, the two main differences in carbohydrate composition are form and concentration. The form, ranging from starch to simple glucose, contributes to the characteristics of osmolality, sweetness, and digestibility. In general, the larger carbohydrate molecules (e.g., starch) exert less osmotic pressure, taste less sweet, and require more digestion than shorter ones (e.g., maltodextrin, sucrose, corn syrup solids). In the critical care setting, optimal carbohydrate delivery should be at a level to allow maximal protein sparing while minimizing hyperglycemia. Currently 4 to 6 mg/kg/min appears to meet these criteria during states of hypermetabolism.

Fiber

It has been claimed that fiber is beneficial in the control of a myriad of gastrointestinal disorders, as well as treatment of hyperlipidemia and control of blood glucose. Fiber-containing formulas have 5 to 14 gm of total dietary fiber per liter. The form of fiber used is primarily insoluble fiber (e.g., soy fiber), but some formulas also contain extra-soluble fiber (e.g., guars, pectins). The insoluble fiber is beneficial with regard to colonic function and bowel regulation. The soluble fibers may slow gastric emptying and decrease the rise in postprandial blood glucose levels as well as bind bile acids and dietary cholesterol, thus lowering serum cholesterol levels. The soluble fibers are also substrates for bacterial fermentation in the colon, yielding short-chain fatty acids (SCFA), carbon dioxide, methane, hydrogen, and water. SCFAs are known to be the primary fuel for the colonocyte. It is believed that SCFAs are required to maintain optimal colonocyte function. In patients requiring long-term tube feeding, a fiber-containing formula may help to regulate GI motility. Because of the higher viscosity of these formulas, the use of larger bore tubes (10 Fr or greater) or an infusion pump is suggested.

TABLE 42.7

Overview of Select Enteral Formulas

Formula Category	Protein Sources	% Calories from Protein	Carbohydrate Sources	% Calories from Carbohydrate	Fat Sources	% Calories from Fat	Caloric Density (Calories/mL)	NPC:g N	mL for 100% RDI	% Free Water	mOsm/kg Water	Product Names (Select Number)
Oral supplements	Sodium & calcium caseinates, soy protein isolate	14-24	Corn syrup, sugar, sucrose, maltodextrin	47-64	Corn oil, canola oil, soy oil, sunflower oil, safflower oil	21-39	1.0-2.0	78-154:1	946-2000	73-85	480-870	Ensure, Ensure Plus, Sustacal, Sustacal with Fiber, Resource Plus, NuBasics, Sustacal Plus
Standard tube feedings	Sodium & calcium caseinates, soy protein isolates	13-18	Corn syrup, maltodextrin	45-57	Soy oil, corn oil, canola oil, MCT, safflower oil	29-39	1.0-1.5	116-167:1	830-1890	77-85	270-500	Isocal, IsoSource HN, Nutren 1.0, Nutren 1.5, Osmolite, Osmolite HN, Comply
Standard tube feedings with fiber	Sodium & calcium caseinates, soy protein isolate	14-18	Corn syrup, maltodextrin, corn syrup solids, soy fiber, guar gum, oat fiber, FOS	44-57	Canola oil, soybean oil, corn oil, MCT	29-37	1.0-1.2	110-149:1	933-1500	78-85	300-500	Fibersource, Jevity, Jevity Plus, ProBalance, Ultracal, Nutren 1.0 with Fiber
High protein tube feedings	Sodium and calcium caseinates	22-25	Hydrolyzed cornstarch, maltodextrin, sucrose, fructose, oat fiber, soy fiber	38-52	Canola oil, MCT, soybean oil, safflower oil	23-40	1.0-1.5	75-91:1	1000-2000	78-85	300-490	IsoSource VHN, Replete with Fiber, Promote, Protain XL, TraumaCal

Elemental & semi-elemental	Free amino acids, soy hydrolysates, hydrolyzed whey, hydrolyzed casein, hydrolyzed soy	12-25	Hydrolyzed cornstarch, maltodextrin, sucrose, modified cornstarch	36-82	Soybean oil, safflower oil, canola oil, MCT sunflower oil	3-39	1.0-1.5	67-175:1	1150-2000	76-86	270-650	Vivonex T.E.N., Crucial, Peptamen, Perative, Reablin, AlitraQ, Sandosource Peptide, Subdue, Optimental
Pulmonary	Sodium and calcium caseinates	17-20	Hydrolyzed cornstarch, corn syrup, sucrose, maltodextrin, sugar	27-40	Canola oil, soybean oil, MCT, corn oil, safflower oil, sardine oil, borage oil	40-55	1.5	102-125:1	933-1420	76-79	330-650	Nutrivent, Pulmocare, Respalor, Novasource Pulmonary, Oxepa
Renal	Sodium & calcium caseinates, Whey-L-amino acids	7-15	Corn syrup, sucrose, fructose, maltodextrin, sugar	40-58	Corn oil, safflower oil, canola oil, MCT	35-45	2.0	140-340:1	947-1000	70-71	570-700	Nepro, Magnacal Renal, Novasource Renal RenalCal
Diabetic	Sodium & calcium caseinates, beef, milk protein, soy protein isolate	16-24	Maltodextrin, hydrolyzed cornstarch, fructose, sucrose, guar gum, vegetables, fruits, soy fiber	34-40	Sunflower oil, soybean oil, canola oil, MCT, safflower oil	40-49	1.0-1.06	79-125:1	1000-1890 or N/A	85	355-450	Glucerna, Glytrol, Choice dm, Diabeti-Source, Resource Diabetic
Immune Modulated	Sodium & calcium caseinates, L-Arginine, L-glutamine, BCAA	22-32	Hydrolyzed cornstarch, maltodextrin, soy fiber	38-53	Canola oil, structured lipids: sunflower oil and menhaden fish oil, MCT	20-40	1.0-1.5	52-71:1	1250-2000	78-86	375-550	Impact, Impact 1.5 ImmunAid, Crucial
Hepatic	L-amino acids, Whey	11-15	Sucrose, maltodextrin, modified cornstarch	57-77	Soybean oil, MCT, canola oil, corn oil, lecithin	12-28	1.2-1.5	148-209:1	N/A-1000	76-82	560-690	NutriHep, Hepatic Aid II

Fructooligosaccharides

Fructooligosaccharides (FOS) are undigestible sugars that occur naturally in food (e.g. onions, blueberries). These sugars consist of a sucrose molecule linked to one, two, or three additional fructose units. Gastric acid or digestive enzymes do not degrade FOS. These oligosaccharides appear to remain intact in the small intestine and pass into the colon unaltered, where they are fermented by colonic microorganisms (e.g., *bifidobacteria*) to lactate and SCFAs. It is suggested that the proliferation of *bifidobacteria* species and the presence of FOS with the consequent production of the fermented byproducts acetate and lactate, produce an environment undesirable for some pathogenic bacteria such as *Clostridium difficile* by lowering the colonic pH.[14,16] A few enteral formulations now contain FOS, but proposed benefits remain to be elucidated.

Fat

The major sources of fat in standard lactose-free formulas are corn, soy, safflower, and canola oils, lecithin, and medium-chain triacylglycerol (MCT). In addition to their importance as a concentrated caloric source (9 kcalories/gm), fat is required for essential fatty acids, and serves as a carrier for the fat-soluble vitamins. Fat also enhances the flavor and palatability of a formula without increasing osmolality. Long-chain triacylglycerol (LCT) is a rich source of essential fatty acids, linoleic and linolenic acid. The estimated daily requirement for essential fatty acids is 3 to 4% of total kcalories. However, due to LCT route of absorption via the lymphatic system, their limited utilization during hypermetabolism, and their immunosuppressive effects when given in large quantities, many formulas now combine LCT with MCT.

MCTs are 6 to 12 carbons long and are prepared from palm kernel or coconut oil. MCT is advantageous because it is more rapidly hydrolyzed and water soluble than LCT, requires little or no pancreatic lipase or bile salts for absorption, and can be transported directly into portal venous circulation where it crosses the mitochondrial membrane and can be oxidized independent of carnitine.[39] MCTs are generally well tolerated by the enteral route but can be associated with some GI symptoms such as nausea, vomiting, and diarrhea. As they produce ketones, they should not be used in patients who are prone to high ketone levels.[39] Since they do not contain essential fatty acids (EFA), most enteral formulas that contain MCT also provide some LCT in order to meet the requirement for EFA.

Recent metabolic research has led to the incorporation of omega-3 fatty acids (linolenic) into enteral formulas. Numerous reports using various *in vivo* and *in vitro* models suggest that the slight structural difference between omega-3 and omega-6 fatty acids strongly favors anti-inflammatory, antithrombotic, antiarrhythmic, hypolipidemic, and antiatherosclerotic effects.[40-42]

Structured lipids are a chemical mixture of LCTs and MCTs incorporated onto the same glycerol molecule. They differ from the more simple random physical mixtures of LCT and MCT. Structured lipids may offer the advantages of both types of fats. Structured lipids have been shown to decrease infection and improve survival by producing less inflammatory and immunosuppressive eicosanoids as compared with conventional triacylglycerols.[39] Enteral formulas, particularly the immune-modulated category, are beginning to include structured lipids as a source of fat.

Protein

Protein contained in enteral formulas may be in the form of intact protein (e.g., lactalbumin, casein, caseinates), partially hydrolyzed protein (e.g., oligopeptides, di- or tripep-

tides), or crystalline L-amino acids. Intact protein and protein hydrolysates (≥4 amino acid residues) require further luminal digestion by pancreatic or brush border enzymes into peptides (di- or tripeptides) and free amino acids, which are then absorbed primarily in the proximal small bowel. The peptide transport mechanisms are felt to be responsible for absorption of the majority of nitrogen, with the single amino acid carriers playing a minority role in protein absorption. Intact proteins do not add appreciably to the osmolality of the formula, unlike hydrolyzed or crystalline amino acids. The higher the percentage of hydrolyzed protein or free amino acids, the greater the solution osmolality will be. A knowledge of the source and form of protein is essential when prescribing diets for patients with defects in either protein digestion (e.g., pancreatic insufficiency) or absorption (e.g., short bowel syndrome).

Stress and other forms of injury may alter protein metabolism. In times of decreased absorptive surface area, ischemic injury, or malabsorption, provision of enteral formulas containing hydrolyzed protein or free amino acids has been suggested. At present, no clear consistent clinical data support the use of solutions in which protein is in the form of free amino acids or hydrolysates. This may be due to the fact that the small bowel has a very adaptive absorptive mucosa, even when a large percentage of the small bowel mucosa is nonfunctional or resected. Although patients with maldigestion and/or malabsorption may benefit from a peptide-based enteral formula, the higher cost of these formulas and lack of clinical supportive data discourage routine use in patients with normal GI physiology.

Glutamine

Glutamine is the most abundant amino acid in the body and in normal situations is considered a non-essential amino acid. It can be synthesized in many tissues of the body, predominantly skeletal muscle, and is the primary fuel for rapidly dividing tissues such as the small bowel. Glutamine serves many purposes including maintenance of acid-base status as a precursor of urinary ammonia, as a primary fuel source for enterocytes, as a fuel source for lymphocytes and macrophages, and as a precursor for nucleotide synthesis.[43,44] Glutamine is also a precursor for glutathione, an important antioxidant that may be protective in a variety of circumstances. During catabolic illness, glutamine uptake by the small intestine and immunologically active cells may exceed glutamine synthesis and release from skeletal muscle, making glutamine conditionally essential.[43]

Limited human data exist regarding the use of enteral glutamine supplementation. In animal models, supplemental glutamine has been shown to enhance intestinal adaptation after massive small bowel resection, to attenuate intestinal and pancreatic atrophy, and to prevent hepatic steatosis associated with parenteral and elemental enteral feeding.[43] Glutamine appears to maintain GI tract mucosal thickness, maintain DNA and protein content, reduce bacteremia and mortality after chemotherapy, and reduce bacteremia and mortality following sepsis or endotoxemia.[43,44]

In humans with surgical stress, glutamine-supplemented parenteral nutrition appears to maintain nitrogen balance and the intracellular glutamine pool in skeletal muscle.[43] In critically ill patients, glutamine supplementation may attenuate villous atrophy and increased intestinal mucosal permeability associated with parenteral nutrition.[45] Parenteral nutrition supplemented with glutamine has also resulted in fewer infections, improved nitrogen balance, and significantly shorter mean hospital length of stay in bone marrow transplantation patients.[46] Glutamine supplementation may also play a role in protecting the GI tract against chemotherapy-induced toxicity. Oral glutamine supplementation reduced the severity and decreased the duration of stomatitis that occurred during chemotherapy.[47]

While a large volume of animal data supports the beneficial effects of glutamine in a variety of experimental models, the benefit of enteral glutamine supplementation in critically ill human patients is less clear. Well-designed clinical trials with clearly defined endpoints and adequate statistical power are needed to assess whether the animal effects translate into a reduction in hospital stay and mortality rate in humans.

Arginine

Arginine is classified as a nonessential amino acid in normal situations, since the body synthesizes adequate arginine for normal maintenance of tissue metabolism. During injuries such as trauma or stress an increase in urinary nitrogen, excreted largely as urea, represents the end-products of increased tissue catabolism and reprioritized protein synthesis. As the activity of the urea cycle increases, so does the demand for arginine.

Studies indicate that supplemental dietary arginine is beneficial for accelerated wound healing, enhanced immune response, and positive nitrogen balance.[43] The exact mechanism for these benefits is unknown but may in part result from arginine's role as a potent stimulant of growth hormone, glucagon, prolactin, and insulin release.[43] Arginine is also a precursor for nitric oxide, a highly reactive molecule synthesized from arginine by the action of nitric oxide synthase resulting in the formation of nitric oxide and citrulline.[48] Nitric oxide is a ubiquitous molecule with important roles in the maintenance of vascular tone, coagulation, the immune system, and the GI tract, and has been implicated as a factor in disease states as diverse as sepsis, hypertension, and cirrhosis.[48]

In animal models, arginine supplementation has been associated with improved wound healing, with increased wound tensile strength and collagen deposition.[49] Arginine-supplemented rats also had improved thymic function as assessed by thymic weight, the total number of thymic lymphocytes in each thymus, and the mitogenic reactivity of thymic lymphocytes to phytohemagglutinin and concanavalin A.[48] Animal arginine supplementation resulted in improved survival in burns, and with intraperitoneal bacterial challenge.

Multiple human clinical trials have been conducted comparing the use of various enteral formulations that contain arginine as well as other supplemental nutrients (e.g., glutamine, omega-3 fatty acids, nucleotides) to a standard nonsupplemented formula. Results of these trials have found the supplemented formula groups to have various improved outcomes such as decreased number and severity of septic complications,[50-53] decreased antibiotic use,[50] and decreased hospital and intensive care unit stay.[50, 54]

While supplemental arginine has been shown to improve survival in various animal models and to improve a number of *in vitro* measures of immune function, the benefit of arginine supplementation alone in critically ill humans is uncertain.

Other Nutrients

Vitamins and Minerals

Most nutritionally complete commercial formulas contain adequate vitamins and minerals when a sufficient volume of formula to meet energy and macronutrient needs is provided. Some disease-specific formulas are nutritionally incomplete in relation to vitamin and mineral content (e.g., hepatic formulas). Liquid vitamin and mineral supplements may be indicated for patients receiving nutritionally incomplete or diluted formulas for prolonged periods of time. Fat-soluble vitamin supplementation such as vitamin K may be indicated for patients with fat malabsorption or for patients with vitamin K deficiency; most commercial formulas include vitamin K.

Water

A large percentage of all enteral formulas is free water. The quantity of water in enteral formulas is often described as water content or moisture content. The quantity of water is usually reported in milliliters of water either per 1000 mL of formula or per liter of formula. Most enteral formulas contain water in the general range of 690 to 860 mL per 1000 mL of enteral formula. This must be considered when making fluid recommendations.

Physical Properties

Osmolality

Osmolality is the function of size and quantity of ionic and molecular particles (protein, carbohydrate, electrolytes, and minerals) within a given volume. The unit of measure for osmolality is mOsm/kg of water versus the unit of measure for osmolarity, which is mOsm/L. Osmolality is considered the preferred term to use in reference to enteral formulas.

Osmolality is important because of its role in maintaining the balance between intracellular and extracellular fluids. Several factors affect the osmolality of enteral formulas. The primary factor is nutrient hydrolysis. The smaller the chain length of carbohydrates and proteins, the greater will be the formulation's osmolality. Hence, formulas containing increased amounts of simple sugars or free amino acids and/or di- and tripeptides will have a greater osmolality than those containing starch and longer-chain intact proteins. Lipids contribute minimally to the osmolality of an enteral formula with the exception of MCT, owing to their water solubility. Because of dissociation properties and small size, minerals and electrolytes also increase the osmolality.

GI tolerance (e.g., gastric retention, abdominal distention, diarrhea, nausea, and vomiting) is influenced by the osmolality of enteral formulas. Generally, the greater the osmolality, the greater the likelihood of GI intolerance. Administering hypertonic formulas at a slow, continuous rate initially (10 to 20 cc/h) with a gradual titration to the final volume while monitoring for GI complications can reduce the incidence of GI intolerance and allow these formulas to be administered at full strength. What may be more important than the osmolality of the enteral formula is the osmotic contribution from liquid medications either infused with the enteral formula or bolused through the feeding tube. The average osmolality range of commercially prepared liquid medications is reported to be 450 to 10,950 mOsm/kg. The osmolality of enteral formulas ranges from 270 to 700 mOsm/kg.

Hydrogen Ion Concentration (pH)

Gastric motility is reportedly slowed with solutions with a pH lower than 3.5. The pH level of most commercial formulas is >3.5. Feeding tube occlusion can be caused in part by the pH of the enteral formula. Most intact protein formulas coagulate when acidified to a pH of less than 5.0.

kcalorie-Nutrient Density

The kcalorie density of enteral formulas is generally 1.0, 1.5, or 2.0 kcalories per milliliter. This is important as it not only determines how many kcalories, but also the other macro- and micronutrients that the patient receives. As a formula becomes more nutrient dense, it contains less free water.

Caloric density often affects the patient's tolerance for tube feeding. Delayed gastric emptying frequently occurs in patients who are given concentrated formulas. High fat

formulas contribute to this, being potent inhibitors of gastric emptying. Since the patient's nutrient needs are met by a decreased volume of this class of formula, free water supplementation should be given to ensure that fluid requirements are met, and to prevent dehydration and constipation. Generally, these products are best tolerated as voluntary oral supplements and not as tube feeding.

Non-Protein Calorie to Gram of Nitrogen Ration (npc:gm N)

In general, the average healthy adult requires a non-protein calorie to gram of nitrogen (npc:gm N) ratio of 150-250:1. In a catabolic state, the body catabolizes lean body mass as a nitrogen and energy source. To minimize this process, it is recommended to provide a npc:gm N of 100-150:1. This protein content of enteral formulas becomes extremely important in patients who require wound healing due to trauma, burns, metabolic stress, infection, and increased wound healing requirements.

Renal Solute Load

Renal solute load refers to the constituents in the formula that must be excreted by the kidneys. Major contributors to renal solute load in enteral formulas are protein, sodium, potassium, and chloride. There is an obligatory water loss for each unit of solute. Therefore, as a formula becomes more concentrated or its renal solute load increases, the patient will require more water.[39] Pediatric and geriatric patients, as well as those with diarrhea, emesis, fistulas, or fevers, should be monitored closely for hydration status.

Disease-Specific Formulations

Most patients can tolerate enteral nutrition safely with a standard enteral formula and do not require specialty enteral formulations. Specialty enteral formulas have an increased cost that often may not be reimbursable; however, factors such as severe hypercatabolism, renal or hepatic failure, pulmonary insufficiency, or malnutrition may alter nutrient metabolism and may thereby warrant an enteral formulation tailored to the specific disease process. Determining the location of enteral nutrient delivery, mode of delivery, and the patient's overall current clinical condition as well as past medical history is necessary for appropriate cost-effective formula selection.

Renal Formulas

The clinical status of patients with renal failure is diverse; therefore, prescribed nutrient intake may vary greatly among patients and should depend on individual nutritional status, catabolic rate, residual glomerular filtration rate, and intensity of dialysis or hemofiltration therapy. Formulas for renal insufficiency do not clearly distinguish the difference between patients with acute failure and those with chronic renal failure.

Renal enteral formulas were first developed as oral supplements; therefore, they tend to be hyperosmolar secondary to their large simple sugar content for flavor enhancement. This hypertonicity often causes GI complications if these formulas are tube fed. The simple sugar content can also be problematic, causing impaired glycemic control in patients who are hypermetabolic, insulin resistant, or diabetic. The goal of feeding patients with renal failure is to provide optimal nutrients without compromising their medical condition through the accumulation of nitrogenous compounds, electrolytes, and fluid. Hence, renal formulas are all calorically rich, providing 2 kcalories per milliliter and containing low-to-moderate amounts of protein, electrolytes, and various minerals. Essential amino acid

(EAA) formulas were developed to decrease urea toxicity. However, previously presumed nonessential amino acids (NEAAs) are probably conditionally essential (e.g., arginine, glutamine, histidine) during metabolic stress. Recent guidelines recommend the use of EAAs and NEAAs for enhanced protein synthesis, correction of low plasma NEAA values, provision of nonspecific nitrogen via NEAAs, and enhanced protein synthesis.[55,56] Nutrients should be provided as needed. The development of fluid and electrolyte disorders or accumulation of metabolic waste products should not be minimized solely by nutrient restriction, but also by adjusting the intensity of dialysis treatment as tolerated.[57] Many patients with stable levels of creatinine, blood urea nitrogen, and electrolytes with or without dialysis can be fed with standard complete enteral formulas.

Pulmonary Formulas

Respiratory insufficiency and ventilator dependence can have a major impact on the feeding of critically ill patients. Often these patients do not receive their full nutritional needs due to the increased work of breathing, carbon dioxide retention, and fluid and electrolyte restrictions. This reduced nutrient intake results in loss of lean body tissue (e.g., intercostals, diaphragm) and malnutrition that in turn leads to fatigue and further difficulty with weaning from the ventilator.

Lipid oxidation is known to produce less carbon dioxide than oxidation of either glucose or protein. This has been the basis for the development of high fat (~45 to 55% of kcalories) and calorically concentrated (1.5 kcalories per milliliter) enteral formulas. Originally these products consisted of 100% long-chain triacylglycerol, which can suppress the immune system as well as cause malabsorption. Pulmonary formulas now contain a variety of lipids including MCT, omega-6 and omega-3 fatty acids, and more recently, γ-linolenic acid (GLA).

Animal research has shown that omega-3 fatty acids produce reduced amounts of proinflammatory eicosanoids relative to animals fed omega-6 fatty acids.[58] In another study, animals fed diets enriched by GLA, as borage oil, were found to have higher inflammatory exudate cellular levels of GLA and dihomogamma-linolenic acid (DGLA) with reduced levels of prostaglandin E_2 (PGE_2) and leukotrienes,[59] suggesting that GLA modulates inflammatory status in a manner similar to that of omega-3 fatty acids. In another animal study, authors concluded that dietary fish oil and fish and borage oil as compared with corn oil may ameliorate endotoxin-induced acute lung injury by suppressing the levels of proinflammatory eicosanoids in bronchoalveolar lavage fluid, and reduce pulmonary neutrophil accumulation.[60] More clinical trials are necessary to determine these claims and patient indications.

Aside from the previously mentioned studies with pulmonary patients, previous research evaluating the use of pulmonary enteral formulas has not demonstrated a clear benefit in providing a high-fat, reduced-carbohydrate nutrient prescription for the patient with compromised pulmonary function.[61] The excessive carbohydrate associated with overfeeding can result in a significant rise in pCO_2 and respiratory quotient that influences respiratory function. Close attention should be made to the avoidance of overfeeding by providing energy intakes from 1.2 to 1.5 times the predicted resting energy expenditure or by measuring energy expenditure via indirect calorimetry.[61]

There are potential detrimental effects in using a high-fat, low-carbohydrate enteral formula. It is well known that high-fat diets can impair gastric emptying.[62] Delayed gastric emptying can result in increased gastric residual volumes and increased risk of aspiration. Carbohydrate is the primary energy source during vigorous muscle exercise, as required during ventilator weaning. During vigorous exercise, depleted muscle glycogen stores may limit muscle endurance and strength. Nutritional support for the

pulmonary compromised patient requires a balanced energy mix so that prompt replenishment of respiratory muscle glycogen can occur.[61] Pulmonary formulas, with their low carbohydrate levels, are a potential disadvantage to fully support muscle glycogen repletion during ventilator weaning.

Literature and clinical practice demonstrate that by not calorically overfeeding pulmonary compromised patients, especially if they are septic, nutritional goals may be met with a standard enteral product (~30% kcalories as fat).[56,61]

Diabetic Formulas

Nutrition is an integral component in the management of diabetes mellitus (DM). Whether during critical illness or long-term support, it can be extremely challenging. Over the past several years, enteral formulas have been developed emphasizing glycemic control for patients with DM. These formulations contain high fat- low-carbohydrate nutrient ratios, with actual ingredients varying among the manufacturers (see Table 42.7). The carbohydrate sources include fructose and fiber to assist in glycemic management. Some fat sources have been modified to contain a higher ratio of monounsaturated fatty acids than saturated fatty acids to better meet the 1994 guidelines of the American Diabetes Association.

A few individual outcome studies have been conducted to determine any benefit of providing these formulations to gain optimal glycemic control.[63,64] Overall, the recommendation is to begin by administering a standard, fiber-containing enteral formula with moderate carbohydrate and fat content. Blood glucose levels will vary based on the patient's diabetes history, metabolic stress level, and nutrient delivery method. Blood glucose levels should be monitored closely with appropriate insulin management, especially if feeding regimens are altered or interrupted. If metabolically stable diabetic patients do not exhibit desired glycemic control with a standard formula, then a diabetic enteral formula may be beneficial.

Hepatic Failure Formulas

The specialized formulas for patients with cirrhosis and hepatic failure are designed to correct the abnormal amino acid profile associated with hepatic encephalopathy. In certain instances of hepatic failure, amino acid metabolism is altered, resulting in increased plasma aromatic amino acids (AAA) with a significant change in the branched-chain amino acid (BCAA)-to-AAA ratio. This change results in altered blood-brain barrier transport, with resultant hepatic encephalopathy. Specialized enteral formulas for hepatic enchephalopathy have been designed to reduce the availability of AAAs and decrease their passage through the blood-brain barrier. Therefore, these formulas contain low quantities of AAAs and methionine and high quantities of BCAAs.

In metabolically stressed, malnourished cirrhotic patients with encephalopathy, the effectiveness of the BCAA-enriched formulas may lie in correcting malnutrition by increasing nitrogen intake without aggravating the encephalopathy. However, some life-threatening derangement in liver failure, such as portal hypertension and esophageal varices, are unaffected by nutritional repletion. Therefore, these formulas should be provided only in malnourished patients with liver failure and concomitant encephalopathy who have failed to respond to conventional medical therapy, and in whom a potentially dangerous higher level of nitrogen intake is required to induce anabolism.[56] Due to the incidence of associated fluid and electrolyte abnormalities, these formulas are calorically concentrated and contain minimal amounts of electrolytes, with some formulations failing to provide 100% of the U.S. recommended daily intake. Therefore, patients receiving these formulations should be monitored closely to ensure that no further associated nutrient deficiencies occur.

Immune-Modulated Formulas

Over the past several decades, predominantly animal models have shown that certain individual nutrients demonstrate immune benefits. These nutrients include arginine, glutamine, omega-3 fatty acids, and nucleotides. Because of this, several enteral formula manufacturers have developed immune-modulated enteral formulas to potentially improve clinical outcomes in high-risk or critically ill patients. These products all vary in the amounts of these nutrients they contain. More recently, several human studies have been conducted to determine if critically ill or other immune-compromised individuals experience positive outcomes as a result of receiving these formulations. Results of these studies vary; they have been scrutinized for several variables, including lack of feeding comparisons, lack of homogeneous study population comparisons, and the manner in which the data were analyzed. Outcomes from the studies also vary, with some showing no benefits regarding the immune formulas and others showing reduced rates of infection, antibiotic use, incidence of intra-abdominal abscesses, and reduced intensive care unit and hospital length of stay.[40]

Overall, the literature suggests that these immune-modulated formulas may be beneficial for some patients. In patients who had undergone complicated GI surgery, sustained severe trauma, or had complicated ICU stays, immune formulas were linked with decreased incidence of infections and hospital length of stay, but were not shown to reduce mortality in severely injured and immune-compromised patients.[65] More research is necessary to determine the optimal patient populations and duration of therapy for which these formulas may be appropriate.

Methods of Administration

The method for enteral tube feeding is limited to the type and site of enteral feeding access. The formula delivery method selected for the patient also depends on the patient's hemodynamic stability, gastric emptying rate, GI tolerance to tube feeding, type of formula selected, nutrient needs, patient mobility, and ease of administration. The main methods of tube feeding are by continuous, intermittent, or bolus delivery. Each institution should have an established protocol for the initiation and advancement of enteral feedings.

Bolus Feeding

Bolus feedings involve the delivery of larger amounts of formula over short periods of time, usually five minutes or less. The bolus method should only be used with gastric delivery. The stomach can act as a reservoir to handle relatively large volumes of formula (e.g., 400 mL) over a short time as opposed to the small intestine. The feedings are usually administered via a gastrostomy tube, owing to the large lumen, but they can also be given through a small-bore nasogastric tube. Usually a syringe or bulb is used to push 200 to 500 mL of formula into the feeding tube several times a day. A patient should demonstrate adequate gastric emptying and the ability to protect his/her airway (i.e., an intact gag reflex) prior to initiating bolus feedings, especially in the critical care setting, to decrease the risk of aspiration. The ability to absorb nutrients using this type of feeding depends on the access site and the functional capability of the gut.

Bolus feedings are considered the most physiologic method of administration since the gut can rest between feedings and allow for normal hormonal fluctuations. They are the

easiest to administer since a pump is not required. Bolus feedings also allow for increased patient mobility, since they are delivered intermittently and do not require a pump. For these reasons, this method of feeding is most desirable for stable patients who are going home or to an extended care facility with tube feedings.

Intermittent Feedings

This method of feeding requires the formula to be infused over a 20- to 30-minute period. A feeding container and gravity drip is usually used for this method. Intermittent feedings are less likely to cause GI side effects than bolus feeding, since the formula is administered over a longer interval. Depending on the volume delivered, this method may be used for gastric as well as small bowel formula delivery.

Continuous Feedings

Continuous formula delivery is usually the enteral delivery method best tolerated. Continuous feedings are delivered slowly over 12 to 24 hours, typically with an infusion pump. In order to avoid accidental bolus delivery, continuous infusion is preferred over gravity, as a constant infusion rate can be sustained. Postpyloric feedings require continuous infusion. The small bowel does not act as a reservoir for large volumes of fluid within a short time, and GI complications usually arise if feedings are delivered in this manner.

Initiation and progression of continuous feedings should be individualized and based upon the patient's clinical condition and feeding tolerance. Typically, feedings may be initiated at 10 to 50 mL/hour, with the lower range for the critically ill. Progression of tube feedings may range from 10 to 25 mL/hour every 4 to 24 hours, depending on the patient's tolerance, until the desired goal rate is achieved. As a patient is beginning to transition to oral intake, the tube feedings may be cycled to allow for appetite stimulation, or to allow for bowel rest and time away from the pump. The feedings may be administered at night and held during the day to allow for patient mobility and an opportunity to eat.

Enteral Feeding Complications

Although enteral nutrition is the preferred route of nutrient provision in those individuals unable to consume adequate nutrients orally, it is not without complications. Compared to parenteral nutrition, enteral nutrition complications are less serious. Most of the complications with enteral nutrition are minor; however there are a few that may be serious. Most complications can be prevented, or at least made less severe. Appropriate patient assessment for needs and risks, proper feeding route and formula selection, in addition to appropriate monitoring of the enteral nutrition feeding regimen can increase the success of enteral feeding. The most common complications can be categorized as mechanical, metabolic, and gastrointestinal. Table 42.8 lists some of the common complications; their possible causes, and suggested corrective measures.

Monitoring

It is very important to continuously monitor patients for signs of formula intolerance, hydration and electrolyte status, and nutritional status. Physical indicators that should be

TABLE 42.8

Common Complications Associated with Enteral Feeding[66,67]

Complication	Possible Causes	Suggested Corrective Measures
Mechanical		
Obstructed feeding tube	Formula viscosity excessive for feeding tube	Use less viscous formula or larger bore tube
	Obstruction from crushed medications administered through tube	Flush tube before and after feeding Give medications as elixir or assure medications are crushed thoroughly Flush tube before and after delivering each medication
	Coagulation of formula protein in tube when in contact with acidic medium (medication, flushing solution)	Flush feeding tube only with warm water Avoid flushing with sodas, coffee, juices or any other acidic medium
Metabolic		
Hyperglycemia	Metabolic stress, sepsis, trauma Diabetes	Treat origin of stress and provide insulin as needed Avoid excessive carbohydrate delivery Give appropriate insulin dose
Elevated or depressed serum electrolytes	Excessive or inadequate electrolytes in the formula	Change formula
	Refeeding syndrome	Monitor electrolytes closely (e.g., potassium, magnesium, phosphorus) and replace as indicated Initiate carbohydrate gradually, not increasing amount provided until electrolytes and blood glucose levels stabilized
Dehydration	Osmotic diarrhea caused by rapid infusion of hypertonic formula	Infuse formula slowly Change to isotonic formula or dilute with water
	Excessive protein, electrolytes, or both	Reduce protein, electrolytes or increase fluid provision
	Inadequate free water provision	Assure patient receives adequate free water, especially if provided calorically dense formula
Overhydration	Excessive fluid intake	Assess fluid intake; monitor daily fluid intake and output
	Rapid refeeding in malnourished patient Increased extracellular mass catabolism causing loss of body cell mass with subsequent potassium loss	Monitor serum electrolytes, body weight daily; weight change >0.2 kg/d reflects decrease or increase of extracellular fluid
	Cardiac, hepatic, or renal insufficiency	Use calorically dense formula to decrease free water if needed Diuretic therapy
Gradual weight loss	Inadequate calories	Assure patient is receiving prescribed amount of calories Assure to monitor patient over time as nutrient requirements may change due to metabolic alterations
Excessive weight gain	Excess calories	Decrease calories provided, change formula or decrease volume per day
Visceral protein depletion	Inadequate protein or calories	Increase protein and/or calorie provision

TABLE 42.8 *(Continued)*

Common Complications Associated with Enteral Feeding[66,67]

Complication	Possible Causes	Suggested Corrective Measures
Essential fatty acid (EFA) deficiency	Inadequate EFA intake Prolonged use of low fat formula	Include at least 4% of kcal needs as EFA
Gastrointestinal		
Nausea and vomiting	Improper tube location	Reposition or replace feeding tube
	Excessive formula volume or rate infusion	Decrease rate of infusion or volume infused
	Very cold formula	Administer formula at room temperature
	High osmolality formula infused	Change to isotonic formula or dilute with water prior to infusing
	High fat formula infused	Change to lower fat formula
	Smell of enteral formulas	Add flavorings to formula; use polymeric as have less offensive odor
Diarrhea	Too rapid infusion	Decrease rate of infusion
	Lactose intolerance	Use lactose-free formula
	Bolus feedings into small bowel	Only provide continuous or slow gravity feedings into small bowel
	High osmolality formula infused	Change to isotonic formula or dilute with water prior to infusing
	Hyperosmolar medication delivery	Change medications or dilute with water to make isotonic prior to delivery
	Altered GI anatomy or short gut	Change to hydrolyzed or free amino acid formula with MCT oil
Vomiting and diarrhea	Contamination	Check sanitation of formula and equipment; assure proper handling techniques
Abdominal distention, bloating, cramping, gas	Rapid bolus or intermittent infusion of cold formula	Administer formula at room temperature
	Rapid infusion via syringe	Infuse continuously at low rate and gradually increase to goal
	Nutrient malabsorption	Use hydrolyzed formula, MCT containing, lactose free
	Rapid administration of MCT	Administer MCT gradually as tolerated
Constipation	Lack of fiber	Use fiber containing formula or add stool softener
	Inadequate free water	Increase free water intake
	Fecal impaction, GI obstruction	Rectal exam, digital disimpaction
	Inadequate physical activity	Increase ambulation if able
Aspiration or gastric retention	Altered gastric motility, diabetic gastroparesis, altered gag reflex, altered mental status	Assure post-pyloric nutrient delivery with continuous infusion Add prokinetic agent if changed feeding position does not help
	Head of bed <30 degrees	Elevate head of bed to >30 degrees if possible
	Displaced feeding tube	Verify feeding tube placement and replace as needed
	Ileus or hemodynamic instability	If small bowel feedings not tolerated then hold feedings and initiate TPN for prolonged intolerance
	Medications that may slow gastric motility (e.g., opiates, anticholinergics)	Evaluate medications and change if feasible
	Gastric or vagotomy surgery	

TABLE 42.9

Example Monitoring Protocol for Enteral Feeding

Parameters	During Initiation and Advancement of Feedings Until Stable at Goal Rate	Stable at Goal Rate	Long-term Enteral Support — Stable
Body weight	Daily	1-2 times per week	Monthly
Fluid intake/output	Daily	Daily	Daily
Bowel function			
Glucose	Daily unless abnormal then every 1 to 8 hours until stable	2-3 times per week; unless diabetic, then daily	Every 6 months; unless diabetic, then daily
Electrolytes	Daily	2-3 times per week	Every 3-6 months
Blood urea nitrogen			
Creatinine			
Magnesium			
Phosphorus			
Calcium			
Liver function tests	1-2 times per week	1-2 times per month	Every 3-6 months
Triglyceride			
Visceral proteins (prealbumin, transferrin)	1-2 times per week	Weekly	Every 3-6 months
Gastric residuals (for gastric feeds only)	Every 4-6 hours	If < 200 mL, then discontinue	N/A unless gastroparesis, then every 4-6 hours

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monitored include incidence of vomiting, stool frequency, diarrhea, abdominal cramps, bloating, signs of edema or dehydration, and weight changes. In addition, several laboratory parameters should be monitored daily with the initiation of enteral feeding and tapered as the patient stabilizes and demonstrates tolerance (Table 42.9).

Summary

Enteral feeding is the preferred method of providing nutrition in those who cannot consume adequate nutrients orally. Enteral feeding has many advantages over parenteral nutrition, including preservation of the structure and function of the GI tract, more efficient nutrient utilization, fewer infections and metabolic complications, greater ease of administration, and lower cost. In order for enteral nutrition to be successful, patient assessment for the optimal access site, appropriate formula selection, nutrient requirements, monitoring, and trouble-shooting complications are required.

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