Enteral Nutrition

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The delivery of food via a tube directly into the gastrointestinal tract has been described since pre-Christian times. In ancient Egypt, and later in Greece, feeds were introduced into the rectum, and in the nineteenth century, rudimentary tubes were used to infuse basic foods such as broths, eggs, milk, and even alcohol into the esophagus and stomach.[1] Despite the increasing sophistication in other areas of medical care over the past century, treatment with enteral nutrition had been slow to develop. However, over the past two decades, enteral nutrition therapy has undergone a renaissance.[2–7] Many patients who previously received parenteral nutrition are now successfully managed with enteral nutrition alone or in combination with parenteral nutrition.[2,3,8] This has been made possible by the increasing range of options for gastrointestinal access, improved delivery systems, and advances in enteral nutrition formulas. Home enteral nutrition therapy is now an important adjunct to the management of infants and children with chronic disease or feeding problems.[6]

PRINCIPLES OF ENTERAL NUTRITION

Enteral nutrition therapy has a number of advantages over parenteral nutrition in the management of patients requiring nutritional support. Enteral nutrition aids in the preservation of gastrointestinal function by the provision of enteral nutrients and is easier, safer and less costly to administer. However, despite these relative advantages, the delivery of safe and effective enteral nutrition therapy may still present challenges for families and caregivers in terms of time, technical expertise, and cost.[9]

PRESERVATION OF GASTROINTESTINAL FUNCTION

Enteral nutrition mimics the normal gastrointestinal response following the ingestion of a meal, with the exception of the oral phase. The presence of nutrients within the intestinal lumen provides stimulation of gastrointestinal function and helps to maintain the complex intraluminal environment via a number of key mechanisms.

Intraluminal nutrients stimulate gastrointestinal neuroendocrine function, effecting motility, and digestion through the secretion of digestive enzymes and gastrointestinal hormones. Intestinal functional and structural changes occur through local and systemic interaction of nutrients and neuroendocrine peptides, cytokines, and hormones.[10–12] The list of these mediators is constantly growing and includes gastrin; enteroglucagon; peptide YY; interleukin 3, 11, or 15; epidermal growth factor; growth hormone; insulin-like growth factors I and II; glutathione; fiber; short-chain fatty acids; glutamine; triglycerides; dietary nucleotides; and polyamines.[10–12] Monosaccharides and fatty acids can influence the secretion of enteroglucagon and peptide YY and via these mediators effect mucosal growth and decrease intestinal transit time.[13] Carbohydrate, protein, zinc, magnesium, potassium, or manganese deficiency can modify the effect of growth hormone and insulin-like growth factor II.[14–16]

Intraluminal nutrients assist in the maintenance of gut mucosal mass, including the gut-associated lymphoid tissue (GALT). The GALT consists of the lamina propria, intraepithelial lymphocytes, immunoglobulin A (IgA), Peyer's patches, and mesenteric nodes and is responsible for processing intestinal antigens.[17] During periods of "bowel rest," such as occur with intravenous feeding and starvation, there is a reduction in gut mass and the function of the GALT is suppressed.[17] This has been associated with a reduction in IgA secretion and increased gut permeability resulting in increased bacterial adherence to the intestinal wall, cellular injury, and bacterial penetration with adverse systemic host responses.[17–19] In animal studies, an association between parenteral nutrition and bacterial translocation has been reported; however, these results have not been replicated in humans.[20] Oral or enteral feeding may reduce the potential risk of bacterial translocation, except when disturbances in intestinal permeability are related to an underlying disease process (eg, short-bowel syndrome) or the chemical composition of the enterally provided substance (eg, blue food dye).[17,21–23]

The gastrointestinal tract is a delicate ecosystem with the balance determined and maintained through the interplay between nutrients, bacteria, and the intestinal defense system (luminal, mucosal, and submucosal immune system). Intraluminal nutrients play an important role in the development and function of the gastrointestinal ecosystem through the modulation of the resident bacterial flora. The key contribution of prebiotics to development of normal intestinal flora and the intestinal immune system has been recognized.[24] The interaction between specific bacteria and toll-like receptors located on intestinal enterocytes and lymphoid cell from as early as birth influences the development of physiologic intestinal immune response.[24] The provision of nutrients via the intestine results in improved utilization of digested and absorbed nutrients. Gut and liver work in synchronism utilizing and eliminating nutrients. The actions of digestion and absorption in the gut followed by a first-pass metabolism in the liver contribute to maintenance of physiologic metabolism.

COST

Although enteral nutrition therapy is more costly than standard feeds, compared to parenteral nutrition therapy, enteral nutrition is approximately two- to fourfold cheaper on an inpatient or out-patient basis.[2,3,9,25–27] Based on US Medicare charges, the annual cost of providing enteral nutrition per patient is approximately US$9,605 ± US$9,327 compared with US$55,193 ± US$30,596 for parenteral solutions.[4] In addition, the frequency and cost of hospitalization is higher for patients supported on parenteral nutrition therapy compared with enteral nutrition therapy.[4]

MANAGEABILITY AND SAFETY

Due to advances in technology of enteral feeding tubes and delivery systems, specialization of health professionals, and better education of parents and caregivers, the administration of enteral nutrition has been associated with improved clinical outcome and safety profiles.[28] Enteral nutrition therapy is easier and safer to administer than is parenteral nutrition. Not only are the risks of intravenous access avoided, but there is also a wider margin for error with most metabolic complications. As a result, enteral nutrition therapy is easier to administer in low-intensity hospitals and patient care settings, including the home. However, compared with normal diet, tube feedings require extra time and effort to administer and this additional care need may contribute to increased burden and stress for families and caregivers.[9,29]

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INDICATIONS FOR ENTERAL NUTRITION

Enteral nutrition should be considered for any patient with a functional gastrointestinal tract who requires nutritional support. Enteral feeding may be required if adequate oral nutrient intake cannot be provided in children with growth failure, weight faltering, or weight deficit. This may be defined as a child with a weight or weight for height less than 5th percentile below the mean for sex and age and/or crossing of two growth curves on the weight, height, or weight/height percentile charts, no weight gain or weight loss during the last 2–3 months and/or triceps skin fold measurements less than 5th percentile. Children with severe neurological dysfunction may require prolonged periods devoted to oral feeding. Tube feeding can provide welcome respite for families and caregivers who previously may have spent over 6 hours a day assisting with oral feeding. Patients with severe neurological disabilities associated with oropharyngeal dysfunction may be at risk of chronic aspiration. The risk of aspiration may be reduced by tube feeding. Children with cognitive, pyschiatric, and behavioral disorders that interfere with oral feeding may develop nutritional deficiency. Enteral tube feeding may provide a safe and reliable route for the delivery of essential nutrients. Enteral feeding may be an option for children with increased energy needs that are difficult to achieve via the oral route such as may occur in cystic fibrosis or congenital heart disease. Disorders of the gastrointestinal tract that result in excessive gastrointestinal losses, such as short-bowel syndrome, secretory diarrhea, or dysmotility syndromes, may have improved absorption and reduction in losses with small volume continuous enteral feeds of a specialized formula. Due to their composition these formulas are often unpalatable and require tube administration to obtain adequate volumes of administration. Most patients receiving parenteral nutrition will also receive some enteral nutrition. Enteral nutrition usually provides an important transition stage as the patient progresses from parenteral nutrition to oral diet. Although enteral nutrition has mainly a therapeutic intent, it can also be used to prevent the development of malnutrition, such as can occur during cancer chemotherapy.[30] A more recent concept is that of minimal enteral feeding, in which enteral nutrition is provided at a very slow rate and volume with the aim of presenting nutrients to the intestinal mucosa without attempting to contribute significantly to total-body nutrition.[31] Advances in the understanding of the role of nutrients in the modification of inflammation and specific disease processes have led to the development of disease-specific formulas (eg, for Crohn's disease).[32] Enteral nutrition has been advocated as a primary treatment for conditions associated with a metabolic disturbance, such as the use of gastrostomy tube feeding to infuse a ketogenic diet in children with epilepsy.[33]

Indications for enteral nutrition in pediatric patients are listed in Table 1. With developments in options for gastrointestinal access, delivery systems, and enteral formulas, the list of absolute contraindications for enteral nutrition therapy has been reduced significantly. Contraindications include gastrointestinal ischemia, including necrotizing enterocolitis and toxic megacolon, severe intractable vomiting or diarrhea, diffuse peritonitis, and mechanical intestinal obstruction.[7,34] Extreme care should be taken when administering enteral nutrition in patients in whom the gastrointestinal blood flow could be compromised, such as during treatment with hypothermia, low cardiac output, multiorgan failure, chronic occlusion, compression syndromes, or infusions of specific drugs. However, in addition to a reduction of caloric deficits, enteral nutrition has been shown to protect the splanchnic oxygen balance during intraoperative duodenal feedings in severely burned patients.[35]

Table 1 Indications for Pediatric Enteral Nutrition

1. Inability to ingest adequate nutrition orally
   - i. Disorders of sucking and swallowing
     - Prematurity
     - Neurological and neuromuscular disorders (eg, cerebral palsy, dysphagia)
   - ii. Congenital abnormalities of the upper gastrointestinal tract or airways
     - Tracheoesophageal fistula
   - iii. Tumors
     - Oral cancer
     - Head and neck cancer
   - iv. Trauma
   - v. Critical illness
     - Mechanical ventilation
   - vi. Severe gastroesophageal reflux
   - vii. Drug related
     - Chemotherapy
   - vii. Severe food aversion
   - viii. Severe depression
2. Disorders of digestion or absorption
   - i. Cystic fibrosis
   - ii. Short-bowel syndrome
   - iii. Inflammatory bowel disease
   - iv. Congenital abnormalities of the gastrointestinal tract
     - Microvillus inclusion disease
     - Tufting enteropathy
   - v. Enteritis
   - vi. Intractable diarrhea of infancy
   - vii. Auto-immune enteropathy
   - viii. Immunodeficiency
     - AIDS
     - Severe combined immunodeficiency
   - ix. Postgastrointestinal surgery
   - x. Graft-versus-host disease
   - xi. Solid organ transplantation
   - xii. Intestinal fistulae
   - xiii. Chronic liver disease
     - Biliary atresia
     - Alagille's Syndrome
3. Disorders of gastrointestinal motility
   - i. Chronic pseudo-obstruction
   - ii. Ileocolonic Hirschsprung's disease
4. Increased nutritional requirements
   - i. Cystic fibrosis
   - ii. Chronic renal disease
   - iii. Congenital heart disease
   - iv. Chronic pulmonary disease
     - Bronchopulmonary dysplasia
   - v. Burn injury
5. Psychiatric and behavioral disorders that interfere with oral intake
   - i. Anorexia nervosa
   - ii. Severe behavioral disorders
     - Autism
6. Metabolic diseases
   - i. Inborn errors of metabolism
   - ii. Diabetes mellitus
7. Acute or acute/chronic pancreatitis
8. Administration of disease treatment
   - Ketogenic diet in epilepsy
   - Administration of pharmaceutical agents
   - Bowel washouts in severe chronic constipation

ROUTES OF ADMINISTRATION

During enteral nutrition therapy, nutrients are directly delivered via a tube into the stomach, duodenum, or jejunum. The tube is inserted either through the nose or mouth for short-term enteral nutrition (<3 months) or through a surgically or endoscopically created stoma for long-term enteral nutrition (>3 months). The choice of the location and the route of administration will depend on the patient's underlying medical or surgical condition, including gastrointestinal anatomy and function, the indication and duration of enteral nutrition therapy, and psychosocial factors. Additional factors, such as local technical expertise, tube availability, and cost, will also influence the route and type of device selected. As a general principle, tubes that deliver nutrients into the stomach are the preferred choice. Gastric tubes are easier to insert and allow a physiological digestive process with bolus or continuous feeding regimens. However, in the presence of gastric outlet dysfunction, severe gastroesophageal reflux, or gastric paralysis, trans-pyloric access may be indicated.

NONINVASIVE GASTROINTESTINAL ACCESS FOR ENTERAL NUTRITION

Nonsurgical or nonendoscopic placement of a feeding tube through the mouth or nose is the most common method of establishing gastrointestinal access in infants and children due to the ease of placement and cost. The nasal access is usually preferred, except in preterm infants or in patients with nasopharyngeal abnormalities or obstruction as may occur following trauma or with congenital malformations. The tube is generally of small diameter (5 to 12 Fr) and is well suited to nutritional support of short or intermediate duration or intermittent nutritional therapy. However, the small luminal diameter renders these tubes susceptible to blockage particularly when medications or nutrient supplements are infused. To minimize the risk of blockage it is recommended that the tube is flushed after each feed and each infusion of medication.

There is a wide range of enteric feeding tubes available for use in children. Early nasogastric

tubes were composed of polyethylene or polyvinyl chloride. Because of their inherent stiffness, they required regular replacement to reduce the risk of skin necrosis, gastric ulceration, and perforation. Current feeding tubes are made from flexible silicone, polyurethane, or elastomer and may require a stylet to assist placement. Despite the increased flexibility, they have a longer life span and may incorporate specialized features. These features may include (1) aids for tube placement and to prevent dislodgment, including water-activated hydrophilic lubricant at the distal end and in the lumen, plastic-coated stylets to minimize the risk of tube perforation, marked reference points on the tubing to allow proper tube selection and positioning, and a rounded, non-weighted bullet-shaped tip to favor insertion; (2) a combination of distal-end and side exit ports to prevent blockage; and (3) a double port at the proximal end to allow for feeding and side injections. Accurate tube positioning is enhanced by radiopaque material within the tube wall. Tube sizes differ in length and port diameter, with the longer tubes suited for jejunal feeding (Table 2). Weighted tips are designed to allow gravity to assist with small-bowel placement and to prevent retrograde displacement. However, with the possible exception of patients requiring mechanical ventilation, the weighted tubes have not been shown to have significant benefit over non-weighted tubes.[36,37]

Nasogastric Tube Placement

With training, nasogastric tubes can be safely inserted by allied health staff, caregivers, family members, and even the patients themselves. Prior to insertion, the desired length of the tube is estimated by measuring the distance from the tip of the nose to the ear and down to the xiphoid. This provides an estimate of the distance between the external nares and the gastroesophageal junction. An additional measurement from the xiphoid to the right or left lateral costal margin provides an estimate of the distance from the gastroesophageal junction to the pylorus. A small amount of lubricant is applied to the nostril and along the length of the tube. The tube is then advanced through the nares past the nasopharynx into the stomach. Voluntary swallowing and head flexion by the patient can aid the passage of the tube. Once the tube is in place, the stylet is removed and the proximal end of the tube is secured close to or behind the ear. The tube location must be verified before commencing the infusion. Fluoroscopy or endoscopy can be used to assist difficult gastric tube placement as well as magnetic assisted devices.

Table 2 A Guide to Pediatric Enteric Tubes

	Tube French size	Tube length (cm)
Premature to neonate	4–5	38–41
Infants to young children	5–8	41–91
Older children to adolescents	8–14	91–114

Nasoduodenal or Nasojejunal Tube Placement

Advances in tube technology and the techniques for placement of nasoenteric tubes in children have provided an opportunity for postpyloric feeding in children in whom gastric feeding is difficult or contraindicated. As a result there has been renewed enthusiasm for the use of postpyloric feeding in the critically ill. Compared to intragastric feeding, postpyloric feeding in the critically ill is associated with a higher caloric intake and avoids complications associated with parenteral nutrition therapy. In a recent study in critically ill children requiring mechanical ventilation the risk of aspiration was similar in patients receiving either postpyloric or intragastric feeding.[38]

Nasoenteric tubes can be placed blindly or under fluoroscopic or endoscopic guidance.[39] The blind tube placement technique relies on the spontaneous passage of an enteric feeding tube from the stomach into the small intestine. This can occur after a period of hours or days and be facilitated by positioning the patient on the right side using the "corkscrew" technique, whereby a wire stylet is twisted when the tube is in the stomach. The use of air insufflation, a pH sensing tube, or the administration of prokinetic drugs, such as metoclopramide, may assist in tube placement.[40,41] The role of erythromycin for postpyloric intubation is controversial, with both positive and negative results observed in children.[42,43] In a randomized controlled trial comparing enteric-tube placement in critically ill children using either the standard technique, the standard technique with gastric insufflation, or the standard technique with erythromycin, a high rate of successful placement was reached at the first attempt (88%) in all groups, with approximately 95% of the tubes placed at the second attempt.[44] The operator's experience rather than the specific technique was correlated with successful placement.

Postpyloric tube placement can also be performed under fluoroscopic guidance.[45] In this method, the passage of the tube is monitored with the aid of the radiopaque markings on the tube. Endoscopic placement of a nasoenteric tube has a number of advantages over the other methods of placement. The tube is placed under direct vision with a guidewire within the tube lumen using a drag and pull technique.[39,46] Endoscopic placement avoids exposure to ionizing radiation and can be performed in high-dependency patient care areas such as the intensive care unit.[39] The application of magnets to guide tubes into small bowel seems promising although pediatric experience using this technique is limited.

INVASIVE ACCESS FOR ENTERAL NUTRITION

Placement of a feeding tube using surgical, radiological, or endoscopic techniques is recommended for long-term enteral nutrition therapy or gastric decompression. There are a number of important advantages of these tubes over nasogastric or nasoenteric tubes in children. The gastrostomy or jejunostomy tube can be maintained in position for a longer period because it is fixed against the anterior abdominal wall. The tube does not interfere with breathing and avoids potential complications of chronic nasal discharge, sinusitis, and developmental abnormalities of the nose. In addition, for some children, repeated insertion of a nasogastric tube is associated with psychological trauma and feeding aversion. Once the decision has been made to place a gastrostomy or jejunostomy tube for long-term home enteral nutrition therapy, education of the patient, parents, and caregivers should be initiated.

Gastrostomy Tube

Gastrostomy tubes are usually large-bore tubes (14–24 Fr) to deliver high feeding volumes and medications with minimal risk of occlusion. The original gastrostomy tubes were made from latex with a balloon retention device, such as the Foley catheter. Today, most gastrostomy tubes are made of a biocompatible material such as silicone or polyurethane, and are anchored in place with either parallel bumpers, or a mushroom or balloon at the gastric site with a retention disk at the skin. This allows the tube to be secured to the stomach wall without sutures. The gastrostomy tube can extend through the stoma with at least two access ports for simultaneous administration of feed and medical medications. A wide range of gastrostomy tubes is available for use in children. These tubes can be summarized into the following types: (i) standard gastrostomy tube with a cupped internal bolster, a sliding external bolster, and a separate cap to be placed at the distal end of the tube with a large feeding infusion port and a small side port for medications; (ii) a balloon gastrostomy replacement tube with a balloon-type internal bolster, a sliding external bolster, and a one-piece external tube ending with three openings for feedings, medications and inflation; (iii) low-profile gastrostomy devices with a balloon-type or a stylet distensible internal bolster and a shaft of a predetermined length to suit the fistula length. The external bolster contains the access infusion port. When in use a connector is used to allow infusion of formula or medications. Most low-profile devices have an antireflux valve to prevent the release of gastric contents when the tube is accessed. This device is easily disguised under clothing and the feeding tube is connected only at the time of infusion; as a result it is particularly popular with older children and adolescents (Figure 1).

The ideal position for gastrostomy tube placement is on the greater curvature of the stomach with the stoma sited on the anterior abdominal wall just below the costal margin with consideration of the axis of bending and clothing. However, if proximity to the small intestine is a priority, such as in the use of the gastric stoma for placement of a jejunostomy tube, placement close

Figure 1 A child with PEG ready for a physical education session.

to the antrum may be preferred. The external bolster should be routinely checked for secure position to avoid tube migration and to prevent injury to the abdominal wall due to compression between the internal and external bolsters. The stomal site requires regular cleaning to avoid chemical injuries from the gastric secretions and infection. Stomal granulomas and skin irritation or infection need prompt attention with referral to a stomal therapist if necessary, to prevent further complications. Eventually gastrostomy tubes will degrade over months to years due to physical or chemical trauma from yeast contamination or medications. The most vulnerable gastrostomy devices are balloon-type skin level devices which have a high rate of inner balloon rupture limiting their longevity to an average of 5 months.[47] However, these devices have been associated with few major complications.

The introduction of the percutaneous endoscopic gastrostomy (PEG) technique has revolutionized the placement of enteric feeding tubes in children.[48] This relatively simple and fast procedure can be performed during esophagogastroduodenoscopy in an endoscopy suite with the use of conscious sedation and local anesthesia or general anesthesia with a success rate of up to 96% even in small infants (<3.5 kg).[49] Although several techniques (Ponsky pull, Sachs-Vine push, Russel introducer) have been developed, all have in common the basic principle that the endoscope locates the site of tube placement from within the stomach while transillumination of the light from the endoscope through the abdominal wall identifies the site of skin incision. Using the Ponsky pull technique, the anterior abdominal wall is indented at the point of maximal transillumination of the endoscope light. This should be seen as a sharp indentation on the gastric wall by the endoscopist. A poorly defined indentation could indicate an overlying viscus (eg, transverse colon) and either an alternative site should be sought or the procedure converted to a surgical gastrostomy. After sterile preparation of the abdominal wall, local anesthetic is instilled and a small incision is made. The endoscopist distends the stomach with air and prepares the snare. A cannula is inserted perpendicular to the abdominal wall through the incision and punctures the gastric wall. The stylet is then removed; a thick suture is introduced along the cannula and is snared by the endoscopist. The endoscope and the suture are retrieved. The gastrostomy tube is tied to the suture and is slowly pulled back, by tension at the abdominal wall, through the mouth, along the esophagus, and into position on the gastric wall under direct vision by the endoscopist. Once the position has been verified by endoscopy, the external bolster can be opposed to the abdominal wall and the tube cut to the desired length. Postinsertion edema at the stoma site is common, and care should be taken not to pull the bolster too tight. To minimize infection at the stoma site, perioperative antibiotic prophylaxis is advocated.[50] The tube can be used within 6 to 24 hours in most patients.

Immediate complications of PEG placement include abdominal wall skin infection, necrosis of the skin or mucosa caused by a tight bolster, perforation of a viscus, hepatogastric-, gastrocolic- or colocutaneous fistula, and pharyngeal or esophageal trauma associated with the passage of the internal fixation device. Pneumoperitoneum is common following PEG placement and does not necessarily indicate a complication of insertion. Long-term complications include gastroesophageal reflux, granulation tissue formation, recurrent stoma-site infection, stoma enlargement, and dislodgment of the tube distally into the small bowel or proximally along the fistula track (ie, buried bumper syndrome).[51] Occlusions are rare events and can be easily treated with warm water instilled by a syringe, pancreatic enzymes and bicarbonate solution, or specific tube cleaning brushes. Contraindications to the PEG technique include gastric varices, severe esophageal stricture, or abnormalities that might restrict the ability to oppose the stomach against the anterior abdominal wall, such as ascites, previous gastrointestinal surgery, or abnormalities in gastrointestinal rotation or position. Extreme care must be taken in patients with musculoskeletal deformities, hyperinflation of the lungs, organomegaly, immunodeficiency disease, cyanotic heart disease, or prior gastrointestinal surgery, including ventriculoperitoneal shunts.

Table 3 Bedside Evaluation Tests for Tube Placement in Children[61–66]

Aspirate	Color and appearance	pH value	Pepsin (μg/mL)	Trypsin (μg/mL)	Bilirubin (mg/dL)
Gastric secretions	Yellow-gray or white-tan; cloudy	< 6	> 100 (> 20)*	< 30 (< 50)*	< 5
Intestinal secretions	Green	> 6	< 100 (< 20)*	> 30 (> 50)*	> 5
Respiratory secretions	Yellow-gray; mucoid	6–8	< 100 (< 20)*	< 30 (< 50)*	< 5

*Trypsin and pepsin ranges specific for children.

Radiological placement of gastrostomy tubes has been shown to be safe and cost-effective in pediatric patients.[52,53] During this procedure, the stomach is distended with air instilled via a nasogastric tube. The stomach is then directly punctured under fluoroscopic control. A guidewire is inserted, followed by an introducer, a dilator, and then finally the feeding tube. The major disadvantage of this procedure is the exposure to ionizing radiation during the procedure.

With the development of the PEG technique surgical placement of a gastrostomy tube has become generally restricted to patients who have a contraindication for PEG placement, had a failed PEG placement, or require another surgical procedure in conjunction with tube placement, such as a fundoplication. Placement of the feeding tube can be performed using an open surgical approach or by laparoscopy. In the classic open gastrostomy technique (Stamm and Witzel techniques), the tube is inserted into the stomach along a serosa-lined tract, whereas in the revised version (Janeway technique), a small portion of the stomach is used to make a mucosa-lined tube attached to the skin as a modified fistula. The procedure for laparoscopic gastrostomy tube placement requires the creation of pneumoperitoneum and insertion of an umbilical catheter. The anterior stomach wall is fastened to the abdominal wall with temporary sutures. An opening (by a needle and a J-wire) is made in the stomach and progressively enlarged using dilators to finally allow the insertion of the feeding tube.[54] In experienced hands, laparoscopic gastrostomy is faster than the open procedure and is associated with reduced length of hospital stay, and patient discomfort.[55] Laparoscopy is also a useful aid during PEG placement by monitoring tube placement by direct vision.[56]

Jejunostomy Tube

The percutaneous endoscopic technique can be used to place an enteric tube using either a direct approach (direct percutaneous endoscopic jejunostomy, DPEJ) or by the creation of a PEG and placement of a specialized double-lumen tube (percutaneous endoscopic gastrostomy jejunostomy, PEJ).[57] The method for the DPEJ is similar to the PEG technique but with the endoscope placed in the jejunum. Transillumination of the bowel through the anterior abdominal wall and a sharp indentation easily seen within the small bowel by the endoscopist is necessary prior to the direct puncture of the duodenum or jejunum.[57] The suture is advanced along the cannula and is grasped by forceps rather than a snare. The PEGJ can be inserted as part of an initial PEG procedure or through an existing gastrostomy stoma. The specialized tube has a gastric lumen and port and another longer lumen and port for the small bowel. This enables gastric decompression during postpyloric feeding. Once the tube is inserted into the stomach, the intestinal lumen of the tube can then be advanced into the duodenum or jejunum under direct endoscopic vision using a guidewire and grasping forceps.

Among the range of surgical techniques described for jejunostomy tube placement, the needle-catheter jejunostomy is the most common. Using this technique, a large-bore needle is tunneled through the seromuscular layers of the jejunum distal to the ligament of Treitz. The jejunum is then anchored to the anterior abdominal wall and the tube is secured to the skin. The tunneling procedure limits reflux of formula and heals quickly upon tube removal. In the presence of intestinal adhesions, severe intestinal disease, or high risk of infection or bleeding, straight insertion of a tube into the jejunum (Stamm technique) or direct jejunal stoma (Maydl technique) might be preferred. Laparoscopic placement of a jejunostomy tube requires two additional cannulae to bring the proximal jejunum into proximity of the abdominal wall and secure it there.[58] Fluoroscopic J-tube positioning is also possible using similar techniques as described above for the insertion of an intragastric tube.

J-tubes are generally smaller in diameter (9–12 Fr) than gastrostomy tubes and tend to have a shorter lifespan (3–6 months). Dislodgement of gastro-jejunal tubes is not uncommon and may be a limiting factor for the use of this type of tube in patients requiring long-term home enteral nutrition.[59]

MONITORING TUBE POSITION AND CONDITION

Before infusing any fluid through a gastric or enteric feeding tube, the position of the tube should be confirmed. Complications owing to incorrect placement or tube dislodgment can potentially be fatal. Plain or contrast radiography is a universally accepted method for assessing tube position. However, repeat radiological studies are impractical and potentially unsafe in patients requiring long-term enteral nutrition therapy. As a result, bedside methods have been developed to screen for correct tube position. These include clinical observation, auscultation, and analysis of tube aspirate. If a nasogastric tube is incorrectly placed in the airways, cough, choking, and pulmonary distress can occur. However, these features can be minimal or absent if the tube is small or if tracheal reflexes are absent (eg, in coma or intubated patients). Although commonly used, auscultation does not reliably distinguish between either gastric and pulmonary placement or gastric and small-bowel placement.[60] Aspirates from either the feeding tube or the lungs can be assessed for color, appearance, pH level, and enzyme measurements (Table 3).[61–66] Aspirate pH value can be measured with a qualitative colorimetric test strip or a quantitative pH meter. Bilirubin can also be measured with spectrophotometer readings, using urine bilirubin test strips, or on the colorimetric visual bilirubin scale. Detection of gastric pepsin in tracheal aspirates relies on a Western blot immunoassay using a rooster polyclonal antibody against human pepsin.[38] Trypsin also can be used to screen for intestinal juice present in the aspirate. Both trypsin and pepsin are secreted at a physiologically lower level in infants and young children compared to adults but continue to be effective tools in the discrimination of tube placement.[64] However, measurement of gastric aspirate may be modified by fasting, intermittent or continuous feeding, and use of gastric acid suppressants. Due to the difficulties in verifying tube position, new approaches are being developed to confirm correct tube position. The measurement of myoelectric slow-wave frequencies has been proposed because these differ in the stomach (3 cycles/min) and the duodenum (11 to 12 cycles/min) and are not influenced by other gastrointestinal contractions.[67] The "bubbling under water" method relies on the exit of bubbles from the external end of the tube when placed under water if the tube has been misplaced in the lungs. However, misplacement of the tube into the bronchioles or pleura will not produce bubbles. Other options under development include electromagnetic navigation devices, self-propelling tubes, fiberoptic tube tips, and ultrasonography-guided tube placement.[68–71]

In the case of PEG or other invasive tube placement it is recommended to routinely examine the stoma tract and the access device to prevent the occurrence of major complications. For instance, incorrect positioning of the external bolster, cracks or ruptures, discoloration or irregular beading of the tube, stoma leaking are examples of initial problems of access devises to be looked for and promptly managed. In patients in whom dislodgment or migration of the gastrostomy or jejunostomy tube is suspected, endoscopy or liquid contrast radiology may assist in defining the tube position.

DELIVERY OF ENTERAL NUTRITION

The method of delivery of enteral nutrition will depend on the route of administration (gastric, duodenal, or jejunal), characteristics of the feeding tube (small- versus large-bore catheter), the

desired feeding pattern (bolus, intermittent, cyclic, or continuous), and the cost and availability of equipment (by gravity or syringe, or by infusion pump). Bolus tube infusion usually mimics the normal meal pattern based on age. Intermittent or cyclic feedings are delivered at a specified rate over 1 or more hours, with 4 to 8 hours per day of gut rest allowing freedom from tubes and pumps for a period of the day. Continuous feeding delivers a constant-rate infusion, usually by an infusion pump, over the entire day or a prolonged period. The infusion pump allows accurate nutrient delivery with less intestinal pressure. Some pumps are specifically designed for outdoor activities and may be carried in a small backpack improving mobility for children on long-term enteral nutrition therapy.

Gastric feeding is preferred because it is considered more physiologic, allows bolus feeds through large-bore tubes, and is generally cheaper and easier to administer.[7] Many patients intolerant of bolus feeds can be successfully fed intragastrically using an intermittent or continuous feeding regimen. Jejunal feeding is an option for patients with disorders of gastric or esophageal anatomy or function and in the nutritional management of the critically ill.[2,3,39] Jejunal feedings are usually delivered as an intermittent or continuous infusion because rapid-rate infusion of nutrients is often limited by abdominal discomfort, diarrhea, or dumping syndrome.

There are specific physiologic and metabolic considerations associated with continuous-rate feeding. During continuous intragastric feeding, gastric emptying increases parallel to the rate of infusion if the infusion rate is maintained at less than 3 kcal/minute.[72] However, increased caloric density, fat, and osmolarity of the formula can delay gastric emptying. The protein composition is also an important factor influencing the gastric emptying rate. Whey-based enteral formulas enter the duodenum faster than casein-based enteral formulas.[73] In animals, the absorptive capacity of the proximal small intestine is unchanged during continuous enteral nutrition. However, the protein and DNA content of the distal small intestine and the enzymatic and functional capacity of the distal small intestine and colon are reduced.[72] The relative lack of nutrients reaching the distal gut during jejunal feeding with an elemental or hydrolyzed formula could explain this observation. This might provide an opportunity for the treatment of gastrointestinal inflammatory diseases, such as Crohn's disease, by providing nutrients to the proximal intestine but reducing antigenic stimulation to the distal gut.[74] In addition, energy expenditure owing to the thermic effect of feeding is lower in patients receiving continuous enteral nutrition than in patients receiving the same quantity of nutrients delivered by bolus.[75] The continuous delivery of nutrients into the small intestine affects glycemic control by modifying the typical fluctuating pattern of insulin and glucagon production.[76,77] The reduction in steatosis during continuous enteral nutrition compared with parenteral nutrition with the same carbohydrate intake is also consistent with this observation. Energy intake goals are more successfully achieved in critically ill patients receiving postpyloric feeds compared with those receiving intragastric feeds.[78]

Enteral feedings should be monitored in terms of intestinal tolerance: nausea, vomiting, diarrhea, abdominal distension, and bowel movements. Tube-feeding residuals are an indirect parameter to determine enteral feeding tolerance. Residuals should be checked more frequently during the first days from starting enteral feedings, when changing infusions or during symptoms. Residuals are checked by aspirating through a tube with a syringe gastric or intestinal fluids. Changes in infusions are recommended when residual volume exceeds twice the hourly infusion volume in continuous feedings or 50% of the infusion volume in bolus. Tubes should be irrigated after any infusion (feeding or medication). Irrigation volume to be instilled into tubes after every meal or medication can vary between 3 and 5 mL in infants and between 20 and 30 mL in children and adults.

ENTERAL FORMULAS

In the early days of enteral nutrition, a mixture of blenderized diets and milk products was administered through a large bore feeding tube. This approach was associated with nutritional imbalance, micronutrient deficiencies, feeding intolerance, and tube blockage. Today, there is a wide range of enteral nutrition products suitable for use in infants and children (see Chapter "Enteral Products" in Appendix III). Most formulas are designed to provide complete macro- and micronutrient requirements as the sole nutritional intake. Specific needs of different ages and stages of development are reflected in the composition of preterm infant, full-term infant, and pediatric enteral nutrition formulations. Modular formulas are specialized combinations of nutrients that provide a nutritional supplement or fulfill a specific nutrient requirement. A recent area of development has been the introduction of disease-specific enteral formulas. These formulas aim to modify the metabolic or gastrointestinal response to feeds by limiting some nutrients, supplementing others, or both (eg, branched-chain amino acid formula for hepatic failure and immune-enhancing formula for critical illness).[79–82]

The majority of patients with normal gastrointestinal tracts will tolerate the gastric administration of a polymeric formula. Polymeric formulas are based on intact protein or polypeptides usually derived from cow's milk or soybeans. The nitrogen to nonnitrogen calorie ratio approximates 1 to 150. Carbohydrates are sourced from different starches, including corn and tapioca. Maltodextrin and hydrolyzed cornstarch, glucose-derived saccharides, and corn syrup are commonly used. Formulas can have different lactose contents. Fats are usually present as polyunsaturated fatty acids from corn, safflower, sunflower, or soybean oil or from animal fat. The content of medium-chain tryglicerides is increased in formulas developed for the treatment of patients with malabsorption. Some enteral nutrition formulas contain soluble fiber. The soluble fiber is added primarily to normalize gastrointestinal transit, but after it is converted to short-chain fatty acids it provides an additional source of calories and can exert trophic effects on the colonic mucosa. In all complete enteral formulas, electrolytes, vitamins, and trace elements are added to provide the Dietary Reference Intakes of micronutrients and minerals at the target volume. However, in a study of stable young children receiving long-term enteral feeding and without excessive gastrointestintal losses, the serum vitamin B_{12} and copper levels exceeded the reference range despite receiving a median energy intake of only 75% of their estimated requirement.[83] At standard dilution, the caloric content of infant formula is usually 0.67 kcal/mL, and of standard enteral formula, 1 kcal/mL. Concentrated enteral nutrition formulas are also available (1.5 and 2 kcal/mL). The osmolality of enteral formulas can range widely depending on the nutrient composition and caloric density (~ 200 to 750 mOsm/L).

In patients with underlying gastrointestinal disease or those requiring jejunal feeding, an oligomeric formula might be indicated. The protein in oligomeric formulas has been hydrolyzed to peptides or a combination of peptides and amino acids. The carbohydrate complexity varies among formulas, although many oligomeric formulas are lactose free. A proportion of medium-chain triglycerides is usually provided to improve fat absorption. Elemental formulas contain completely digested macronutrients, such as monosaccharides, medium-chain triglycerides, and amino acids, with an essential to nonessential amino acid ratio reflecting high biologic protein values. Lactose and gluten are absent and residues are low. The unpalatability and high osmotic load of simple sugars and amino acids generally limit the use of elemental formulas to tube feeding when feeding intolerance occurs with other types of formula (eg, severe malabsorption or short-bowel syndrome).

Advances in the understanding of the role of specific nutrients and their effects on metabolism have led to modification of enteral nutrition formulas for treatment of specific diseases. The aim of the disease-specific formulas is to provide therapeutic benefits in addition to the maintenance of general nutritional status. Glutamine-enriched formula has been advocated for the prevention and treatment of intestinal mucosa injury associated with chemotherapy and critical illness.[79,81] Formulas supplemented with arginine, glutamine, ribonucleic acid nucleotides, medium-chain triglycerides, and/or omega-3 fatty acids have been also been advocated as enhancing the immune system of critically ill patients.[79–82] However, whether the benefit of glutamine or immune-enhancing formulas in malnourished and critically ill children outweighs the additional cost of these formulas remains controversial.[84,85] Omega-3 fatty acids have been supplemented in infant formula to enhance neurologic development.[86] Evidence for improved clinical and economic outcomes of many of the disease-specific formulas, compared with

Table 4 Complications Associated with Enteral Tube Feeding

Complication	Possible cause	Prevention and treatment
	Most common tube feeding complications	
Tube occlusion	Failure to flush tube regularly, inappropriate feed or medications placed down tube, inadequately dissolved feed, high-energy feed	Flush tube regularly with water; use prescribed feeds and Medications only
Tube dislodgment	Inadequate securing of tube, inadequate monitoring of tube position	Check tube placement every 8 hours during continuous feeds and before every bolus or intermittent feed
Accidental tube removal	Inadequate securing mechanism, deterioration of tube (balloon rupture)	Review method of securing tube; consider specific dressings or clothing to prevent access to tube; regularly review tube function and integrity; ensure availability of replacement or "emergency" tube to prevent stoma closing prior to reinsertion
Diarrhea	Gastroenteritis, medications (antibiotics, sorbitol-containing drugs), rapid administration or bolus feeds, malabsorption, formula intolerance (lactose, hyperosmolar)	Review for possible causative factors including medications, formula, and gastrointestinal absorptive function; consider changing rate of delivery or formula as indicated (reduced osmolality, fiber enriched, lactose free)
Bloating, abdominal cramps	Gastrointestinal dysmotility, bowel obstruction, intolerance to formula (lactose intolerance), bacterial overgrowth	Reduce or cease feeds according to severity until cause is defined; consider investigations to address possible causes
Constipation	Inadequate fluid intake or large fluid losses, gastrointestinal dysmotility, medications, immobilization, underlying medical condition	Correct fluid losses and provide adequate ongoing fluid requirements; review medications; consider stool softeners or fiber supplementation
Dumping syndrome	Rapid infusion of high-volume or hypertonic feeds into duodenum or jejunum, postgastric surgery or vagotomy, gastrostomy sited in distal gastric antrum	Administer continuous feeds, reduced volume or osmolality of formula; use uncooked cornstarch
Nosocomial infection (bacteremia, pneumonia)	Enteral feed or equipment contamination, aspiration, increased risk with gastric acid suppression in paralyzed or ventilated Patients	Change sets every 24 hours; limit feed hang time; use sterile system and sterile water to reconstitute feeds; culture feeds and equipment if contamination suspected; review for "silent" reflux or aspiration or tube dislodgment
Metabolic complications (increased or decreased glucose, phosphate, potassium or magnesium)	Complication of the primary disease (renal failure) or treatment (amphotericin), refeeding syndrome	Correct any significant electrolyte abnormalities prior to initiating enteral nutrition; undertake regular biochemical monitoring particularly in malnourished patients; gradually increase feed volume and concentration if at risk of refeeding syndrome
Malabsorption	Underlying gastrointestinal disease (cystic fibrosis), inappropriate formula selection or rate of administration	Assess absorptive status and alter formula and rate of delivery as appropriate
Perforation	Tube malposition, wrong type of tube, disorders of mucosal Integrity	Ensure appropriate tube selection and placement technique; regularly check tube position; surgical treatment might be required
	Gastric tube feeding (general)	
Vomiting, nausea	Gastroenteritis, intolerance to formula, rate of infusion too rapid, medications, bacterial contamination of feed, delayed gastric Emptying	Review feeding regime and medications; culture feeds and equipment if contamination is suspected; consider intermittent or continuous infusion, postpyloric administration, or prokinetic agents
Gastroesophageal reflux	Underlying abnormality of esophagus or stomach associated with reflux or medical illness (eg, neurologic, pulmonary diseases), mechanical aspects related to tube	Assess for underlying reflux; consider antireflux therapies or Postpyloric feeding
Large-volume gastric as pirates	Delayed gastric emptying related to underlying medical condition (eg, neurologic disease, critical illness, diabetes, intestinal pseudo-obstruction) or medications	Review medications; consider continuous or postpyloric feeds or prokinetic agents
Pulmonary aspiration	Incorrect tube placement, tube dislodgment, gastroesophageal reflux, gastric stasis, or vomiting (neurologic disorders, coma)	Cease feeds and check tube position; consider postpyloric feeding
Gastrointestinal bleeding	Tube-related irritation, ulceration, or perforation; vitamin K deficiency	Review tube position and gastrointestinal status; assess for alternative tube placement sites; try gastric acid suppression; supplement with vitamin K when indicated
Cellulitis	Postplacement contamination of wound, inadequate cleaning of stoma site, bolster too tight, chronic leakage through stoma	Perioperative antibiotics during tube placement, regular skin care, antibiotic therapy as appropriate; check tube and stoma site for areas of mechanical irritation and poor fit
Stoma leakage	Site infection; incorrect tube size, type, or position; perished tube; gastric stasis (eg, diabetes, pseudo-obstruction); medications	Examine site; assess tube integrity, suitability, and position; check balloon volume; replace with larger or different type of tube if appropriate; treat stoma site infection
	Nasogastric tube feeding	
Nasal airway obstruction	Inappropriate tube size, nasopharynx disorders	Insert smaller-bore tube
Chronic nasal discharge or ulceration Epistaxis Sinusitis or otitis media	Inappropriate tube size or composition, immunodeficiency, disorder of mucosal or skin integrity	Re-evaluate tube size and type; regularly change tube; assess for alternative sites for tube placement
Feeding aversion	Repeated tube replacement in infants and young children, development implications of tube feeding	Consider alternatives to nasogastric route in infants requiring long-term enteral nutrition; involve speech therapist early
Esophageal perforation	Incorrect tube placement, ulceration related to tube position, underlying disorders of mucosal integrity (eg, epidermolysis bullosa)	Use appropriate tube placement technique; regularly check tube position
Pulmonary intubation	Incorrect tube placement or tube dislodgment	Use appropriate tube placement technique; take particular care in children with neurologic disorders or disturbances of conscious state

(continued)

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Table 4 Complications Associated with Enteral Tube Feeding *(continued)*

Complication	Possible cause	Prevention and treatment
	Gastrostomy tube feeding	
Granulation tissue formation	Chronic inflammation at stoma site, leakage, tube moving too freely along tract	Check tube size, type, and position; specialized dressings, corticosteroid cream, or cautery might be required
Site swelling or tenderness	Site infection, migration of tube along tract, tube shaft too short or bolster too tight	Examine site; tube removal and replacement might be required
Fasciitis	Incorrect tube or bolster position	Remove tube; obtain surgical opinion; administer intravenous antibiotics
Buried bumper syndrome	Retaining device and bolster secured too tight	Remove tube; administer antibiotics
Gastritis, gastric ulceration, or perforation	Trauma caused by tube, often of wall opposite insertion site, wrong tube type (composition or design)	Consider change in tube design to minimize trauma and gastric acid suppression
	Duodenal or jejunal tube feeding	
Reflux into stomach	Tube placed in proximal small intestine, dysmotility	Consider more distal placement
Bowel obstruction	Tube too large, tube malposition, disorder of gastrointestinal anatomy	Review tube size and position
Volvulus	Tube providing an abnormal fixation point	Remove tube; obtain surgical review

standard formulas, is still required to justify recommendations of their routine use.[79,81]

COMPLICATIONS OF ENTERAL NUTRITION THERAPY

Despite the potential benefits of enteral nutrition, complications can occur (Table 4).[87–91] Fortunately, life-threatening events are rare. However, problems related to the tube, the method of delivery, or the composition of the formula can seriously interfere with achieving nutritional goals. To minimize complications, prior consideration should be given to the patient's medical and physical condition, including any metabolic or electrolyte abnormalities, previous diet, dietary tolerance, and the time that has elapsed since the last significant oral or enteral nutrition.

GASTROINTESTINAL COMPLICATIONS

Intestinal discomfort, bloating, cramping, diarrhea, nausea, and vomiting can occur during enteral feeding. Whenever symptoms occur, the position and integrity of the tube should be confirmed. In some cases, these symptoms relate to the high osmolality of the formula or the rate of infusion and alterations to the formula composition or infusion rate will be sufficient to improve feeding tolerance. The assessment of a sample of stool from a patient with diarrhea can assist in directing further investigations as required (bacterial culture, guaiac test, absorptive status by pH, microscopy, and reducing substances). The use of concomitant medications should be reviewed for possible drug-nutrient interactions or gastrointestinal side effects. Bacterial contamination of the feed or the tubing can result in diarrhea or vomiting. Therefore the method of formula preparation and storage and the technique of hanging and administering should be reviewed.[87] Samples of the feed, the tubing, and the feeding reservoirs are necessary to confirm this diagnosis.

Patients with a primary gastrointestinal disorder could be at risk of bacterial overgrowth owing to disturbances in gut motility. Bacterial culture of an intestinal biopsy or a lactulose breath hydrogen test will assist in establishing the presence of bacterial overgrowth of the small intestine. Constipation associated with enteral nutrition is uncommon and when it occurs is usually associated with insufficient fluid or fiber intake, intestinal dysmotility or obstruction, or medications. Gastroesophageal reflux can be exacerbated by tube feeding. The nasogastric tube can split open the lower esophageal sphincter, and irritation owing to tube trauma can contribute to lower sphincter incompetence. Placement of a gastrostomy tube plicates the stomach against the anterior abdominal wall, distorting the normal gastric anatomy, with a potential impact on gastric function. Owing to the frequency of this complication in high-risk patients, such as those with cerebral palsy or cystic fibrosis, assessment for gastroesophageal reflux with a 24-hour pH probe and nuclear gastric emptying study may be considered prior to gastrostomy tube placement.

If the gastrostomy or jejunostomy tube is no longer required, the tube can be removed after consideration of the original method of insertion. For tubes inserted using a PEG, DPEJ, or PEGJ technique, the tube can usually be withdrawn and the stoma edges opposed using a dressing or suture. In most cases, this will be sufficient to allow closure of the stoma and tract within days to weeks. This process can be assisted with the use of gastric acid suppression aimed at minimizing gastric secretion. If the gastro- or jejunocutaneous fistula does not close spontaneously, formal surgical closure might be required. Patients in whom the gastrostomy or jejunostomy tube has been inserted using the traditional surgical approach usually require surgical closure of the fistula.

PULMONARY COMPLICATIONS

Aspiration of gastric contents—and pneumonia owing to aspiration—incorrect tube placement, or tube dislodgment into the airway can be fatal. Children at high risk include those with chronic neurologic disease, depressed conscious state, intestinal dysmotility, severe gastroesophageal reflux and patients requiring mechanical ventilation. In addition to checking tube position, tracheal aspirates can be measured for glucose content or fat-laden macrophages. The addition of blue dye to the formula to assess tube position and aspiration patients should be avoided because it has been associated with fatalities.[26]

INFECTIOUS COMPLICATIONS

Irritation due to the mechanical trauma of tube or exposure to gastric or intestinal secretions renders the skin and mucosa susceptible to infection. A well-fitting, well-maintained device specifically designed for enteral nutrition use will limit this complication. Contamination of the formula and the delivery set can occur as a result of preparation or administration of the feed.[87] Bacterial contamination resulted to occur in 35 to 50% of enteral bags at the end of infusions in a pediatric hospital.[92] Feeding devices also harbor biofilm growth of various bacteria and fungi in the outer and inner surfaces with potential risk of local and systemic infections.[93] Longterm nasogastric and nasoenteric tubes are associated with chronic nasal discharge, otitis media, and sinusitis.

MECHANICAL COMPLICATIONS

Mechanical complications related to enteral tube feeding are common. However, these can generally be treated and do not involve the same risks as a central venous catheter in critically ill children.[88–91] Tube occlusion can occur as a result of problems related to the tube (length, caliber, characteristics), the infusion (formula, drugs), pumps and clamps, the method and rate of delivery, and the level of tube care (method, frequency). Infusions that contain a highly viscous formula or crushed or powdered drugs are often associated with tube occlusion. If flushing the tube with warm water is unsuccessful in clearing the blockage, a number of other

options are available. The instillation of pancreatic enzyme supplements is sometimes successful in clearing a blockage. The use of meat tenderizer is not recommended because of its high sodium content and the risk of tube breakage during infusion. Cytology brushes, guidewires, neonatal tubes inserted into larger tubes, and specially designed catheters have been developed to treat tube occlusion. A biliary catheter for endoscopic retrograde cholangiopancreatography was described for the treatment of an occlusion in a nasojejunal tube.[94] The key to the prevention of tube occlusion is careful monitoring and repeated flushing of the tube. Mixing of drugs and formula should be avoided.

Irritation can occur anywhere along the interface between the tube and the skin or mucosa, resulting in inflammation, ulceration, or even perforation of any structure present along the tube lining (eg, laceration or bleeding of nasal or oral cavity, perforation of trachea or pulmonary parenchyma, perforation or laceration of the gut). Granulation tissue formation at the stoma site can occur as a result of chronic irritation. The migration of tubes with an internal balloon or retention device distally along the gastrointestinal tract can cause bowel obstruction. Migration of a gastrostomy tube along the fistula tract can cause pain and inflammation around the site or the buried bumper syndrome.[51] Complications such as dislodgment, ampullary obstruction, and jaundice can occur with jejunal tubes.

METABOLIC COMPLICATIONS

Metabolic complications associated with enteral nutrition are uncommon. Patients with chronic malnutrition or cardiac, hepatic, or renal impairment require careful monitoring of fluid and electrolyte status to prevent imbalance.[95] Patients commencing enteral nutrition who have had a period of prolonged fasting, inadequate nutritional intake, or significant weight loss (>10% body weight) should be monitored for the metabolic features of the refeeding syndrome. Daily monitoring of fluid status, serum sodium, potassium, phosphate, and magnesium levels is required until stability has been achieved with feeding advancement. Serum glucose levels, which are usually stable during continuous enteral nutrition therapy, can increase with overfeeding or as part of a stress response during critical illness. Hypoglycemia during intermittent feeding can be prevented with a progressive slowing of the rate of infusion in the period prior to disconnection. Dumping syndrome is reported in patients during enteral feeding in response to the presence of nutrients within the proximal intestine. This is can be associated with a rapid infusion of a formula with a high nutrient density and is treated with modifications to the formula (such as adding uncooked cornstarch) or rate of delivery.

HOME ENTERAL NUTRITION

Over the past two decades, advances in tube design, methods of delivery, and formulas have made enteral nutrition therapy safer, cheaper, and easier to administer for a wide range of disorders in childhood.[5,96,97] With attention to appropriate patient selection, education, and providing adequate technical support, enteral nutrition therapy can be safely and effectively provided in the home.[97] In 1992, data from Medicare and insurance companies estimated that there were about 152,000 patients of all ages receiving home enteral nutrition in the United States.[5] The rapid growth of home enteral nutrition therapy observed in the United States in 1987 had reached a plateau in 1992.[5] In contrast, in Britain, home enteral nutrition therapy has been increasing rapidly—at a rate of up to 20% per year—and is about 10 times more common than home parenteral nutrition therapy.[97] Of patients receiving home enteral nutrition therapy in Britain, 40% are children, compared with 5 to 20% in three US cohorts.[98]

Best-practice guidelines for the administration of safe and effective home enteral nutrition therapy have been developed by national nutrition support organizations.[7,97] These guidelines take into consideration patient selection, assessment, monitoring, and the development of the enteral nutrition plan, including methods of implementation, documentation, and protocols for termination of therapy. Training patients, their families, or caregivers in safe and effective home enteral nutrition therapy is ideally performed by a multidisciplinary team, including the gastroenterologist or surgeon, dietitian, stomal therapist, or nurse specialist.[97] An essential component of a successful home enteral nutrition service is availability of health professionals to provide support to the patient or caregivers at home to address concerns and direct appropriate intervention when necessary. A mechanism should be available to manage after-hours tube malfunction. To facilitate this process, all patients should carry a card with the type and size of tube and the date of last insertion clearly listed. Written documentation of routine tube care, as well as the recommended steps to take in the event of tube malfunction, is invaluable to the patient and caregiver at home.

Regular review by the multidisciplinary home enteral nutrition team is cost-effective and improves quality of care.[99] At regular outpatient appointments, all aspects of the administration of enteral nutrition can be assessed. The tube is examined for size, function, and integrity and can be changed if necessary. The stoma site is examined. The method of delivery is reviewed, along with protocols for formula preparation. The intake of formula is assessed with reference to the nutritional goals. The patient is examined for growth, weight gain, and nutritional status. Laboratory markers of nutritional status can be obtained as indicated.

Table 5 List of Common Medications That May Contain Sorbitol

Acetaminophen (Tylenol pediatric elixir, suspension, and maximum strength liquid)
Acyclovir (Zovirax suspension)
Al/MgOH (Maalox suspension and extra strength plus)
Al/MgOH/simethicone (Mylanta cherry cream-, milk cream-, double strength-liquid)
AlOH (Alternagel, Gaviscon liquid) and AlOH gel
Aminocaproic acid (amicar syrup)
Calcium carbonate ($CaCO_3$ oral suspension), calcium glucobionate (neo-calglucon)
Carbamazepine (tegretol susp)
Chloral hydrate (syrup)
Cimetidine (Tagamed liquid)
Clemastine (Tavist syrup)
Dexamethasone (oral solution)
Diazepam (oral solution)
Digoxin (elixir)
Diphenhydramine (Benadryl elixir)
Felbamate (Felbatol susp)
Ferrous sulfate (drops, syrup)
Furosemide (solutions)
Hydroxyzine (Vistaril susp)
Ibuprofen (Pedia-Profen susp)
Lithium citrate
Metaproterenol and metaproterenol sulfate (syrup)
Metoclopramide (syrup, oral solution)
Milk of magnesia
Multivitamins (Iberet 250, 500 liquid)
Naproxen (Naprosyn oral suspension)
Nitrofurantoin (Furadantin suspension)
Oxybutynin (Ditropan syrup)
PE/Tripolidine (Children's actifed liquid)
PE/PPA/chlorpheniramine/phenyltoloxamine (Naldecon Ped drops, syrup)
Potassium chloride (Rum-K)
Prednisolone (Pediapred)
Propanolol (oral solution)
Ranitidine (Zantac syrup)
Sodium polystyrene sulfonate (suspension)
Sucralfate (Carafate suspension)
Tetracyline (sumycin)
Theophylline (oral solution, Slo-phyllin syrup, Theoclear syrup)
Trimethoprim/Sulfamethoxazole (Septra suspension, sulfa/trimethoprim, bactrim susp)
Valproic acid (Depakene syrup)
Vitamin E (Aquasol E drops)

INTERACTIONS BETWEEN DRUGS AND ENTERAL FEEDING

Administration of medications through an enteral feeding tube, either in combination with formula or alone, is problematic because only a few drugs have been tested and approved for tube delivery. Characteristics of the composition of the tube can influence the binding of drug to the tube wall (eg, carbamazepine reacts with polyvinyl chloride feeding tubes).[7] Interactions between a drug and a nutrient can result in an undesired side effect of the drug or feeding intolerance. Drug-nutrient interactions can result in changes in medication bioavailability, distribution, metabolism, or excretion.[100] Common drug-nutrient interactions are described with substances containing calcium, zinc, and iron, or when acidic and neutral liquid medications are combined with casein or soy protein. Impaired absorption of phenytoin is well documented in the literature and occurs as a result of pharmacokinetic incompatibility.[7] Long-chain fatty acids can enhance the absorption of lipid-soluble drugs but hasten the degradation of other medications (eg, carbamazepine) (Table 5).

Liquid preparations of medications are preferred by children and for enteral administration.

However, liquid drug preparations tend to have a high osmolality (1,000 and 11,000 mOsm/kg) and may cause feeding intolerance when infused into the jejunum. The osmotic properties of these liquids are usually attributable to the presence of sorbitol, propylene glycol, or polyethylene glycol. Although included among the inactive ingredients on drug labels, the amount is not usually quantified. Osmotic diarrhea and delayed gastric emptying can occur if the cumulative dose is more than 20 g (range, 7.5 to 30 g).

CONCLUSION

Advances in tube placement techniques, tube design, delivery systems, and enteral formulas have enabled the safe and effective provision of enteral nutrition therapy for a broad range of disease indications in children. Successful enteral nutrition therapy has limited the use of parenteral nutrition therapy, resulting in important benefits in terms of safety, manageability, and cost. The management of nutritional problems in patients with chronic diseases, such as neurologic disorders, Crohn's and cystic fibrosis, can be enhanced by home enteral nutrition therapy. The development of disease-specific formulas provides new therapeutic options. Further improvements in technology enabling light, simple, feeding pumps and modifications to tube design will continue to assist in the administration of enteral nutrition to children.

REFERENCES

1. Harkness L. The history of enteral nutrition therapy: From raw eggs and nasal tubes to purified amino acids and early post-operative jejunal delivery. J Am Diet Assoc 2002;102:399–404.
2. Braga M, Gianotti L, Gentilini O, et al. Early postoperative enteral nutrition improves gut oxygenation and reduces costs compared with total parenteral nutrition. Crit Care Med 2001;29:242–8.
3. de Lucas C, Moreno M, Lopez-Herce J, et al. Transpyloric enteral nutrition reduces the complication rate and cost in the critically ill child. J Pediatr Gastroenterol Nutr 2000;30:175–80.
4. Reddy P, Malone M. Cost and outcome analysis of home par-enteral and enteral nutrition. JPEN J Parenter Enteral Nutr 1998;22:302–10.
5. North American Home Parenteral and Enteral Nutrition Patient Registry. Annual report with outcome profiles 1985–1992. Albany, NY: Oley Foundation; 1994.
6. Howard L, Ament M, Fleming CR, et al. Current use and clinical outcome of home parenteral and enteral nutrition therapies in the United States. Gastroenterology 1995;109:355–65.
7. Board of Directors and Clinical Guidelines Task Force, American Society for Parenteral and Enteral Nutrition. Guidelines for the use of parenteral and enteral nutrition in adults and pediatric patients. JPEN J Parenter Enteral Nutr 2002;26:18SA–9SA.
8. Braunschweig CL, Levy P, Sheean PM, et al. Enteral compared with parenteral nutrition: A meta-analysis. Am J Clin Nutr 2001;74:534–42.
9. Heyman MB, Harmatz P, Acree M, et al. Economic and psychologic costs for maternal caregivers of gastrostomy-dependent children. J Pediatr 2004;145:511–6.
10. Wong WM, Wright NA. Epidermal growth factor, epidermal growth factor receptors, intestinal growth, and adaptation. JPEN J Parenter Enteral Nutr 1999;23:S83–8.
11. Chen K, Nezu R, Wasa M, et al. Insulin-like growth factor-1 modulation of the intestinal epithelial cell restitution. JPEN J Parenter Enteral Nutr 1999;23:S89–92.
12. Ziegler TR, Estivariz CF, Jonas CR, et al. Interactions between nutrients and peptide growth factors in intestinal growth, repair, and function. JPEN J Parenter Enteral Nutr 1999;23:S174–83.
13. Aponte GW, Fink AS, Meyer JH, et al. Regional distribution and release of peptide YY with fatty acids of different chain lengths. Am J Physiol 1985;249:745–50.
14. Thissen JP, Ketelslegers JM, Underwood LE. Nutritional regulation of the insulin-like growth factors. Endocr Rev 1994;15:80–101.
15. Ninh NX, Thissen JP, Collette L, et al. Zinc supplementation increases growth and circulating insulin-like growth factor I (IGF-1) in growth-retarded Vietnamese children. Am J Clin Nutr 1996;63:514–9.
16. Dorup I. Magnesium and potassium deficiency. Its diagnosis, occurrence and treatment in diuretic therapy and its consequences for growth, protein synthesis and growth factors. Acta Physiol Scand 1994;618:S1–55.
17. Li J, Kudsk KA, Gocinski B, et al. Effects of parenteral and enteral nutrition on gut-associated lymphoid tissue. J Trauma 1995;39:44–51.
18. Alverdy JC, Aoys E, Moss GS. Total parenteral nutrition pro-motes bacterial translocation from the gut. Surgery 1988;104:185–90.
19. Kudsk KA, Li J, Renegar KB. Loss of upper respiratory tract immunity with parenteral feeding. Ann Surg 1996;223:629–38.
20. Sedman PC, MacFie J, Palmer MD, et al. Preoperative total par-enteral nutrition is not associated with mucosal atrophy or bacteria translocation in humans. Br J Surg 1995;82:1663–7.
21. Alverdy JC, Aoys E, Moss GS. Effect of commercially available chemically defined liquid diets on the intestinal microflora and bacterial translocation from the gut. JPEN J Parenter Enteral Nutr 1990;14:1–6.
22. Weber TR. Enteral feeding increases sepsis in infants with short bowel syndrome. J Pediatr Surg 1995;30:1086–9.
23. Maloney JP, Bhargava R, Ryan TA, et al. FD&C Blue No. 1 food dye is a mitochondrial poison that can be absorbed from enteral tube feedings in sepsis: A report of 3 deaths linked to systemic absorption. JPEN J Parenter Enteral Nutr 2001;25:S25.
24. Forchielli ML, Walker WA. The role of gut-associated lymphoid tissues and mucosal defence. Br J Nutrition. 2005;93: S41–8.
25. Adams S, Dellinger P, Wertz MJ, et al. Enteral versus parenteral nutrition support following laparotomy for trauma: A randomized prospective trial. J Trauma 1986;26:882–91.
26. Bower RH, Talamini MA, Sax HC, et al. Postoperative enteral vs parenteral nutrition. Arch Surg 1986;121: 1040–5.
27. Deswarte-Wallace J, Firouzbakhsh S, Finklestein JZ. Using research to change practice: Enteral feedings for pediatric oncology patients. J Pediatr Oncol Nurs 2001;18:217–23.
28. Anonymous. Making health care safer: A critical analysis of patient safety practices. Evid Rep Technol Assess 2001;43:1–668.
29. Enrione EB, Thomlison B, Rubin A. Medical and psychological experiences of family caregivers with children fed enterally at home. JPEN J Parenter Enteral Nutr 2005;29:413–9.
30. Hastings Y, White M, Young J. Enteral nutrition and bone marrow transplantation. J Ped Onc Nurs 2006;23:103–10.
31. Adan D, La Gamma EF, Browne LE. Nutritional management and the multisystem organ failure/systemic inflammatory response syndrome in critically ill preterm neonates. Crit Care Clin 1995;11:751–84.
32. Beattie RM. Enteral nutrition as primary therapy in childhood Crohn's disease: Control of intestinal inflammation and anabolic response. JPEN J Parenter Enteral Nutr 2005;29:S151–9.
33. Hosaid SA, La Vega-Talbott M, Solomon GE. Ketogenic diet in pediatric epilepsy patients with gastrostomy feeling. Pediatr Neurol 2005;32:81–3.
34. Lord L, Harrington M. Enteral nutrition implementation and management. ASPEN Nutrition Support Practice Manual, 2nd edition; 2005. p. 76–89.
35. Andel D, Kamolz LP, Donner A, et al. Impact of intraoperative duodenal feeling on the oxygen bilance of the splanchnic region in severely burned patients. Burns 2005;31:302–5.
36. Zaloga GP. Bedside method for placing small bowel feeding tubes in critically ill patients: A prospective study. Chest 1991;100:1643–6.
37. Lord LM, Weser-Maimone A, Pulhamus M, et al. Comparison of weighted versus unweighted enteral feeding tubes for efficacy of transpyloric intubation. JPEN J Parenter Enteral Nutr 1993;17:271–3.
38. Meert KL, Daphtary KM, Metheny NA. Gastric vs small-bowel feeding in critically ill children receiving mechanical ventilation. Chest 2004;126:872–8.
39. Chellis MJ, Sanders SV, Dean JM, et al. Bedside transpyloric tube placement in the pediatric intensive care unit. JPEN J Parenter Enteral Nutr 1996;20:88–90.
40. da Silva PSL, Paulo CST, de Oliveira, et al. Bedside transpyloric tube placement in the pediatric intensive care unit: A modified insufflation air technique. Intensive Care Med 2002;28:943–6.
41. Kittinger JW, Sandler RS, Heizer WD. Efficacy of metoclopramide as an adjunct to duodenal placement of small-bore feeding tubes: A randomized, placebo-controlled, double-blind study. JPEN J Parenter Enteral Nutr 1987;11:33–7.
42. Di Lorenzo C, Lachman R, Hyman PE. Intravenous erythromycin for post-pyloric intubation. J Pediatr Gastroenterol Nutr 1990;11:45–7.
43. Gharpure V, Meert KL, Sarnaik AP. Efficacy of erythromycin for postpyloric placement of feeding tubes in critically ill children: A randomized, double-blind, placebo controlled study. JPEN J Parenter Enteral Nutr 2001;25:160–5.
44. Phipps LM, Weber MD, Ginder BR, et al. A randomized controlled trial comparing three different techniques of nasojejunal feeding tube placement in critically ill children. J Par Enter Nutr 2005;29:420–4.
45. Guitierrez ED, Balfe DM. Fluoroscopically guided nasoenteric feeding tube placement: Results of a 1-year study. Radiology 1991;178:759–62.
46. Dranoff JA, Angood PJ, Topazian M, et al. Transnasal endoscopy for enteral feeding tube placement in critically ill patients. Am J Gastroenterol 1999;94:2902–4.
47. Michaud L, Guimber D, Blain-Stregloff AS, et al. Longevity of balloon-stabilized skin-level gastrostomy device. J Ped Gastroent Nutr 2004;38:426–9.
48. Gaudener MWL, Ponsky JL, Izant RJ, Jr. Gastrostomy without laparotomy: A percutaneous endoscopic technique. J Pediatr Surg 1980;15:872–5.
49. Wilson L, Oliva-Hemker M. Percutaneous endoscopic gastrostomy in small medically complex infants. Endoscopy 2001;33:433–6.
50. Jonas SK, Neimark S, Panwalker AP. Effect of antibiotic prophylaxis in percutaneous endoscopic gastrostomy. Am J Gastroenterol 1985;80:438–41.
51. Klein S, Heare BR, Soloway RD. The "buried bumper syndrome": A complication of percutaneous endoscopic gastrostomy. Am J Gastroenterol 1990;85:448–51.
52. Chait PG, Weinberg J, Connolly BL, et al. Retrograde percutaneous gastrostomy and gastrojejunostomy in 505 children: A 4 1/2 year experience. Radiology 1996;201:691–5.
53. Barron MA, Duncan DS, Green GJ, et al. Efficacy and safety of radiologically placed gastrostomy tubes in paediatric haematology/oncology patients. Med Pediatr Oncol 2000;34:177–82.
54. Duh QY, Way LW. Laparoscopic gastrostomy using T-fasteners as retractors and anchors. Surg Endosc 1993;7:60–3.
55. Rosser JC, Rodas EB, Blancaflor J, et al. A simplified technique for laparoscopic jejunostomy and gastrostomy tube placement. Am J Surg 1999;177:61–5.
56. Lotan G, Broide E, Efrati Y, et al. Laparoscopicaly monitored percutaneous endoscopic gastrostomy (PEG) in children. Surg Endosc 2004;18:1280–2.
57. Shike M, Latkany L, Gerdes H, et al. Direct percutaneous endoscopic jejunostomies for enteral feeding. Gastrointest Endosc 1996;44:536–40.
58. Duh QY, Way LW. Laparoscopic jejunostomy using T-fasteners as retractors and anchors. Arch Surg 1993; 128:105–8.
59. Fortunato JE, Darbari A, Mitchell SE, et al. The limitations of gastro-jejunal (G-J) feeding tubes in children: A 9-year pediatric hospital database analysis. Am J Gastroenterol 2005;100:186–9.
60. Metheny N, Dettenmeir P, Hampton K, et al. Detection of inadvertent respiratory placement of small-bore feeding tubes: A report of 10 cases. Heart Lung 1990;19:631–8.
61. Westhus N. Methods to test feeding tube placement in children. Am J Mat Child Nurs 2004;29:282–89.
62. Metheny NA, Clouse R, Clark J, et al. pH testing of feeding-tube aspirates to determine placement. Nutr Clin Pract 1994;9:185–90.
63. Metheny NA, Reed L, Berglund B, et al. Visual characteristics of aspirates from feeding tubes as a method of predicting tube location. Nurs Res 1994;43:282–7.
64. Gharpure V, Meert KL, Sarnaik AP, et al. Indicators of postpyloric feeding tube placement in children. Crit Care Med 2000;28:2962–6.
65. Metheny NA, Smith L, Stewart BJ. Development of a reliable and valid bedside test for bilirubin and its utility for improving prediction of feeding tube location. Nurs Res 2000;49:302–9.
66. Ellett MLC, Croffie JMB, Cohen MD, et al. Gastric tube placement in young children. Clin Nurs Res 2005;14:238–52.
67. Cannon RA, Isaac RM, Puryear H. Confirmation of enteral feeding tube placement using duodenal slowwave frequency analysis. Gastroenterology 1989;96:A73.

68. Greenberg M, Bejar R, Asser S. Confirmation of transpyloric feeding tube placement by ultrasonography. J Pediatr 1993;122:413–5.
69. Grathwohl KW, Gibbons RV, Dillard TA, et al. Bedside videoscopic placement of feeding tubes: Development of fiberoptics through the tube. Crit Care Med 1997;25:629–34.
70. Gabriel SA, Ackermann RJ, Castresana MR. A new technique for placement of nasoenteral feeding tubes using external magnetic guidance. Crit Care Med 1997;25:641–5.
71. Ozdemir B, Frost M, Hayes J, et al. Placement of nasoenteral feeding tubes using magnetic guidance: Retesting a new technique. J Am Coll Nutr 2000;19:446–51.
72. Goulet O. Paeditric enteral feeding. Int Semin Paediatr Gastroenterol Nutr 1998;7:1–8.
73. Khoshoo V, Brown S. Gastric emptying of two whey-based formulas of different energy density and its clinical implication in children with volume intolerance. Eur J Clin Nutr 2002;56:656–8.
74. Morin CL, Ling V, Bourass D. Small intestinal colonic changes induced by a chemically defined diet. Dig Dis Sci 1980;25:123–8.
75. Nacht CA, Schutz O, Vernet L, et al. Continuous versus single bolus enteral nutrition: Comparison of energy metabolism. Am J Physiol 1986;251:E524–9.
76. Heymsfield S, Casper K, Grossman GD. Bioenergetic and metabolic response to continuous versus intermittent feeding. Metabolism 1987;36:570–5.
77. Pinchofsky-Devin GD, Kaminsky MV. Visceral protein increase associated with interrupt versus continuous enteral hyperalimentation. JPEN J Parenter Enteral Nutr 1985;9:474–6.
78. Graham TW, Zadrozny DB, Harrington T. The benefits of early jejunal hyperalimentation in the head-injured patient. Neurosurgery 1989;25:729–35.
79. Heyland DK, Novak F, Drover JW, et al. Should immunonutrition become routine in critically ill patients? A systematic review of the evidence. JAMA 2001;286:944–53.
80. Kenler AS, Swails WS, Driscoll DF, et al. Early enteral feeding in postsurgical cancer patients: Fish oil structured lipid-based polymeric formula versus a standard polymeric formula. Ann Surg 1996;223:316–33.
81. Heys S, Walker L, Smith L, et al. Enteral nutritional supplementation with key nutrients in patients with critical illness and cancer. A meta-analysis of randomized controlled clinical trials. Ann Surg 1999;229:467–77.
82. Schloerb P. Immune-enhancing diets: Products, components, and their rationales. JPEN J Parenter Enteral Nutr 2001;25:S3–7.
83. Johnson TE, Janes SJ, MacDonald A, et al. An observational study to evaluate micronutrient status during enteral feeding. Arch Dis Child 2002;86:411–5.
84. Briassoulis G, Filippou O, Hatzi E, et al. Early enteral administration of immunonutrition in critically ill children: Results of a blinded randomized controlled trial. Nutrition 2005;21:799–807.
85. Lima AAM, Brito LFB, Ribeiro HB, et al. Intestinal barrier function and weight gain in malnourished children taking glutamine supplemented enteral formula. J Ped Gastroent Nutr 2005;40:28–35.
86. Uauy-Dagach R, Mena R. Nutritional role of omega-3 fatty acids during the neonatal period. Clin Perinatol 1995;22:157–75.
87. Bott L, Husson MO, Guimber D, et al. Contamination of gastrostomy feeding systems in children in a home-based enteral nutrition program. J Pediatr Gastroenterol Nutr 2001;33:266–70.
88. Ramage IJ, Harvey E, Geary DF, et al. Complications of gastrostomy feeding in children receiving peritoneal dialysis. Pediatr Nephrol 1999;13:249–52.
89. Israel DM, Hassal E. Prolonged use of gastrostomy for enteral hyperalimentation in children with Crohn's disease. Am J Gastroenterol 1995;90:1084–8.
90. Weiss B, Fradkin A, Ben-Akun M, et al. Upper gastrointestinal bleeding due to gastric ulcers in children with gastrostomy tubes. J Clin Gastroenterol 1999;29:48–50.
91. Bastow MD. Complications of enteral nutrition. Gut 1986; 27:51–5.
92. Roy S, Rigal M, Doit C, et al. Bacterial contamination of enteral nutrition in a pediatric hospital. J Hosp Infect 2005;59:311–6.
93. Dautle MP, Wilkinson TR, Gauderer MWL. Isolation and identification of biofilm microorganisms from silicone gastrostomy devices. J Pediatr Surg 2003;38:216–20.
94. Golioto M, Lytle J, Jowell P. Re-establishing patency of a small bore feeding tube with complete occlusion-a novel use for an ERCP catheter. Nutr Clin Pract 2001; 16:284–5.
95. Pardoe EM. Tube feeding syndrome revisited. Nutr Clin Pract 2001;16:144–6.
96. Daveluy W, Guimber D, Mention K, et al. Home enteral nutrition in children: An 11-year experience with 416 patients. Clin Nutr 2005;24:48–54.
97. Elia M, Cottee S, Holden C, et al. Enteral and parenteral nutrition in the community. The British Association for Parenteral and Enteral Nutrition. Berkshire, UK: The Association; 1994.
98. Elia M. Home enteral nutrition: General aspects and a comparison between the United States and Britain. Nutrition 1994;10:115–23.
99. Scott F, Beech R, Smedley F, et al. Prospective, randomized, controlled, single-blind trial of the costs and consequences of systemic nutrition team follow-up over 12 months after percutaneous endoscopic gastrostomy. Nutrition 2005;21:1071–7.
100. Thomson FC, Naysmith MR, Lindsay A. Managing drug therapy in patients receiving enteral and parenteral nutrition. Hosp Pharm 2000;7:155–64.