Nutrition in the paediatric surgical patient

Simon Eaton

Abstract

Nutritional care of surgical infants and children is of major importance. This is for several reasons: (i) body stores are often smaller and more precarious; (ii) infants and children not only require energy for maintenance, but also for growth; and (iii) as in adults, recovery from surgery is faster in those patients who are adequately nourished. Survival of infants with congenital anomalies dramatically improved following the introduction of parenteral nutrition. However, infection and cholestasis remain problematic for parenterally fed infants and children.

Keywords Cholestasis; enteral nutrition; growth; parenteral nutrition

Background

Nutrition is especially important to surgical infants and children, firstly because of smaller body stores and relatively higher energy expenditure compared with adults, and secondly because of the requirement for growth and adequate neurodevelopment. Energy stores are only sufficient for ~ 2 days at 24–25 weeks gestation, for ~ 20 days at term as glycogen and fat stores increase,[1] and are in excess of 50 days in the adult. Hence, the urgent need for adequate caloric intake in preterm infants after birth. Full-term neonates have higher content of endogenous fat (approximately 600 g) and therefore can tolerate a few days of undernutrition. Nevertheless, adequate nutrition in excess of basic requirements, i.e. enough to support growth, should be instituted as soon as practicable. Although adults following surgery or trauma have increased energy requirements,[2] there is no strong evidence that increased energy should be provided to septic or surgical neonates,[3] or to older children requiring surgery. Target enteral calories are as indicated in Table 1.

The optimum nutritional route is oral enteral feeding. However, artificial enteral feeding or parenteral nutrition (PN) may be required if adequate oral feeds cannot be tolerated. The basic principle underlying choice of feeding routes is that the most physiological route that is safely possible should be used: gastric feeds are preferred over jejunal feeds, enteral feeds are preferred over parenteral feeds etc.

Growth monitoring

Artificial enteral nutrition and PN are both nutritional interventions, and nutritional outcomes should be assessed in order to determine the effectiveness of these interventions.

Simon Eaton BSc PhD is an Associate Professor in Paediatric Surgery at UCL Great Ormond Institute of Child Health, London, UK. Conflicts of interest: Simon Eaton has received speakers' honoraria from Danone and support to attend conferences from Fresenius Kabi. This work has been supported by the NIHR GOSH BRC. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR, or the Department of Health.

Suggested calorific intake in enterally fed infants and children

Age of child	Target caloric intake (kcal/kg/d)	
	Males	**Females**
Premature	110—120	110—120
0 -— 1 mth	113	107
1 — 3 mths	100	97
3 mths — 1 year	80	80
1 — 4y	82	78
5 — 8y	73	70
9 — 13y	64	58
14—18y	53	47

Table 1

Growth of all paediatric surgical patients, especially those receiving artificial nutritional support, should be monitored longitudinally using appropriate charts. Currently in the UK we use the UK-WHO growth charts 0-18 years that are available from the Royal College of Paediatrics and Child Health.

Artificial enteral feeding

Indications

The simplest artificial feeding route encountered by paediatric surgeons is the naso- or orogastric tube given to premature infants for immaturity of swallowing. Other indications for artificial enteral feeds are: delayed gastric emptying, gastroesophageal reflux, impaired intestinal motility, Crohn's disease, neurological or metabolic co-morbidity and intensive care/ventilation.

Route

The administration route can be via naso- or orogastric or jejunal tube, via gastrostomy, or via gastro-jejunostomy or surgical jejunostomy tubes. Gastric feeding is preferable to intestinal feeding because it allows for a more natural and complete digestive process, i.e. allows action of salivary and gastric enzymes and the antibacterial action of stomach acid, in addition to the use of the stomach as a reservoir. Gastric feeding is associated with a larger osmotic and volume tolerance and a lower frequency of diarrhoea and dumping syndrome. Thus, transpyloric feeds are usually restricted to infants or children who are either unable to tolerate naso- or orogastric feeds, at increased risk of aspiration; or who have anatomical contraindications to gastric feeds. Neonates are obligatory nose breathers and therefore orogastric feeding may be preferable over nasogastric feeding in preterm infants to avoid upper airway obstruction. However, nasogastric tubes are easier to secure and may involve a lower risk of displacement. In infants requiring gastric tube feeding for extended periods (e.g. more than 6–8 weeks) it is advisable to insert a gastrostomy, to decrease the negative oral stimulation of repeated insertion of nasal or oral tubes. This can be inserted by an open, laparoscopic or percutaneous endoscopic (PEG) route, or by using interventional radiology. In infants with significant gastro-oesophageal reflux, fundoplication with gastrostomy tube or enterostomy tube placement is indicated, although some authors favour a gastrojejunal tube over

195

fundoplication plus gastrostomy. There is no strong evidence to choose any of these therapeutic approaches over any other.

Selection of enteral feeds
Breast milk is the ideal feed for infants because it reduces infection and aids gastrointestinal maturation and neurological development. When breast milk is not available, chemically defined formulae can be used, which are designed either for preterm infants, term infants or older children. If malabsorption is present and persists, an appropriate specific formula should be introduced, such as soy-based disaccharide-free feed for disaccharide intolerance; medium chain triglyceride (MCT) formula for fat malabsorption; and elemental (free amino acids) or semi-elemental (protein hydrolysate containing di- and tripeptides) formula for severe malabsorption due to short bowel syndrome or severe mucosal damage as in NEC. For persistent severe malabsorption, a modular diet may be necessary, in which glucose, amino acid and MCT preparations are provided separately. The possibility of cow's milk protein allergy should be considered in the case of intolerance; this can even occur due to transfer of bovine antigens via breast milk, so is not limited to infants fed bovine-based formula.

Administration of enteral feeds
Enteral feeds can be administered as boluses, continuous feeds or as a combination of the two. Bolus feeds are more physiological and stimulate intestinal motility, enterohepatic circulation of bile acids and gallbladder contraction. Bolus feeds mimic or supplement meals and are easier to administer than continuous feeds since a feeding pump is not required. Where bolus feeds are not tolerated, for example in the presence of gastro-oesophageal reflux, continuous feeds should be administered via an infusion pump over 24 hours. Infants and children with jejunal tubes should receive continuous feeds and not bolus feeds, as the stomach is no longer providing a reservoir.

Complications of enteral tube feeding
Complications can be mechanical including leakage, tube blockage, tube displacement or migration and intestinal perforation. Although infection is less of a risk than with PN, the risk of infected enteral feeds should not be ignored, and the gastrostomy/jejunostomy site can become infected. Other complications involve the gastrointestinal tract. These include: gastro-oesophageal reflux with aspiration pneumonia, dumping syndrome and diarrhoea. Jejunostomy tubes inserted at laparotomy can be also associated with intestinal obstruction.

In surgical infants and children, enteral feeding often results in vomiting, interruption of feeding and inadequate calorie intake. In infants with congenital gastrointestinal anomalies, exclusive enteral feeding is commonly precluded for some time after surgery due to large gastric aspirates and intestinal dysmotility, so calorie intake is established initially by PN (see below). Enteral feeding is introduced when intestinal motility and absorption improves. The percentage of calories given enterally is gradually increased at the expense of intravenous calorie intake. This transition time from total PN to total enteral feeding can be quite long. The presence of gastric aspirate often induces clinicians and surgeons not to use the gut for nutrition. However, minimal enteral feeding (trophic feeding) can be implemented early in these patients. These minimal feeds may prevent gut mucosal atrophy, increase intestinal blood flow, improve activity of digestive enzymes and thus 'prime' the gut for subsequent higher volume, nutritive feeds. Although enteral nutritional adequacy ('full enteral feeds') is usually assessed as enteral volume/calorie tolerance, it should be borne in mind that tolerance is not the same as absorption, as infants and children may require a significant period of time for intestinal adaptation to allow complete absorption of administered feeds. Growth monitoring following full enteral nutritional tolerance therefore remains necessary. In infants and children with stomas, frequent urinary electrolyte measurements are particularly important. Low urinary sodium with normal serum sodium suggests active sodium conservation. As sodium is important for growth, sodium supplementation may be appropriate in this scenario.[4]

Parenteral nutrition

Indications
PN should be utilized when enteral feeding is impossible, inadequate or hazardous, but should be given for the shortest period of time possible and the proportion of nutrition given enterally increased as tolerated. Energy reserves are such that stable term infants can tolerate 3−4 days without enteral feeds, and older children 7−10 days, before starting PN, if it is anticipated that enteral nutrition may be resumed within this time. Premature neonates have smaller energy reserves and the time before introducing PN is much shorter.

The most frequent indications in paediatric surgery are intestinal obstruction due to congenital anomalies, although acquired conditions may require PN for variable lengths of time. Although infants with some neonatal surgical conditions, such as gastroschisis, are all likely to receive PN, there are some other congenital anomalies where the use of PN is more controversial, for example duodenal atresia, in which many surgeons would routinely initiate PN, whereas some surgeons preferentially manage patients without PN by the use of trans-anastomotic tubes. In addition to congenital bowel obstruction, PN may also be used in cases of postoperative ileus, necrotizing enterocolitis, short-bowel syndrome, gastroenterological indications and respiratory comorbidity.

Route
PN must be administered via centrally placed intravenous catheters (including peripherally inserted central catheters [i.e. PICC lines], surgically placed central catheters, or centrally placed umbilical catheters), as peripheral administration gives significant risk of complications from hyperosmolar glucose, which can cause vascular irritation or damage and thrombosis. Central catheter choice depends on what catheters are already in place, and the length of time over which PN is anticipated.[5]

Components of parenteral nutrition
The caloric requirements for PN are provided by carbohydrate and lipid. Protein is required for growth and is not used as a source of calories, since the catabolism of protein to produce energy is an uneconomic metabolic process compared to the oxidation of carbohydrate and fat which produces more energy at a lower metabolic cost. The ideal PN regimen therefore, should provide enough amino acids for protein turnover and tissue growth, and sufficient calories to minimize protein oxidation for energy.

Fluid requirements:[6] hydration and the proportion of extracellular fluid changes rapidly in the neonate. In a surgical infant, these changes will also be occurring simultaneously with surgical intervention and initiation of PN. Fluid overload is a possibility in these infants, and careful consideration of all fluids administered (PN and other prescribed drugs), together with daily monitoring of weight and electrolytes is mandatory, at least until PN has been stabilized. Excessive fluid administration can result in pulmonary oedema or failure of closure of patent ductus arteriosus. Fluid restriction, or a requirement to administer other fluids in significant volume, can result in the delivery of macronutrients being less than current recommendations.

Carbohydrate:[7] up to 17 g/kg/day in neonates, up to 14 g/kg/d in infants <10 kg, up to 9 g/kg/day in children up 30 kg, 6 g/kg/31−45 kg and 4.5 g/kg/d in children >45 kg; avoid hyperglycaemia. Glucose is a main energy source for body cells and should be the primary energy substrate in PN, covering 60−70% of non-protein calories.

Lipid emulsion:[8] up to 4 g/kg/day in infants, 3 g/kg/d in children.

Although pure soybean lipid emulsions can be used short-term, composite lipid emulsions with or without fish oils should be used for PN lasting more than a few days.

Lipids provide an energy-dense, isotonic alternative to glucose as an energy source, which also prevents essential fatty acid deficiency and facilitate provision of fat-soluble vitamins. Combined infusion of glucose and lipids confers metabolic advantages over glucose, because it lowers the metabolic rate and carbon dioxide production and increases the efficiency of energy utilization.

Amino acids:[9] up to 3.5 g/kg/day in preterm infants, 1.5−3 g/kg/d in term infants and 1.0−2.0 g/kg/day in older children. In contrast to healthy adults who exist in a state of neutral nitrogen balance, infants and children need to be in positive nitrogen balance in order to achieve satisfactory growth and development. Preterm infants who are receiving glucose alone lose protein quickly (at the rate of 1−2% body protein per day), so should start to receive at least 1−1.5 g/kg/d amino acids parenterally if not receiving EN.

Electrolytes:[10] sodium 2−4 mmol/kg/d infants, 1−3 in children; potassium 1−3 mmol/kg/d, chloride 2−5 mmol/kg/d. Other electrolytes (calcium, phosphate, magnesium[11]) are very dependent on the clinical condition of the patient and technical pharmacy aspects of stability and compounding.

Vitamins:[12] both lipid-soluble (vitamins A, D, E, K) and water-soluble vitamins (vitamin B complex, C, pantothenate, niacin biotin and folic acid) are added to paediatric PN, usually as commercially available mixtures.

Iron and trace elements:[13] zinc, copper, manganese, selenium, fluoride and iodide are usually added to paediatric PN of longer than a few days duration. Again, they are available as commercially mixtures.

Water:[10] this is necessary to decrease osmolality of the solution. The volume depends on the fluid status of the patient.

The caloric requirement of a PN-fed patient is approximately 10−20% lower than that of enterally-fed patients, because stool and other losses are minimal (Table 2). PN is usually given 24 hours a day, but with a 4-hour suspension of lipid infusion to allow triglyceride clearance.

Complications of PN[14]

Mechanical catheter complications: include thrombosis and incorrect position/displacement. Extravasation of PN solution is a common complication of peripheral PN. Unfortunately, even a low osmolarity solution is detrimental for peripheral veins, leading to inflammation and extravasation of the solution, which can cause tissue necrosis and infection. Intravenous lines may become blocked by thrombus formation, calcium precipitates or lipid deposition.

Metabolic complications: include hyper- or hypoglycaemia, hypertriglyceridaemia, electrolyte alterations.

Fluid overload: can result in oedema but can also lead to failure of closure of patent ductus arteriosus.

Infection: the majority of surgical infants on PN have at least one suspected episode of sepsis, and around 30% of surgical neonates have at least one positive blood culture. Although catheter-borne infections, which can be reduced by rigorous precautions, such chlorhexidine antisepsis, are important, microbial translocation from the intestine is also a significant source of infection in surgical infants on PN.

Cholestasis: the most common hepatobiliary complication of PN is cholestasis (PN-associated cholestasis or PNAC). The clinical significance of this cholestasis itself is unknown, but if untreated, intestinal failure associated liver disease (IFALD) may occur, which can result in the need for liver transplantation, or can result in death. Various clinical factors contribute to the development of PNAC, including prematurity, low birth weight, duration of PN, immature entero-hepatic circulation, intestinal microflora, septicaemia, failure to implement enteral nutrition, short-bowel syndrome due to resection, and the number of laparotomies (reviewed[15]). Infants with gastroschisis or jejunal atresia seem to be at particular risk. PNAC and IFALD are

Suggested calorific intake in parenterally fed infants and children[7]

Age of child	Target caloric intake (kcal/kg/day)		
	Recovery phase	Stable phase ICU	Acute phase ICU
Premature	90−120		45−55
0−1 year	75−85	60−55	45−50
1−7 years	65−75	55−60	40−55
7−12 years	55−65	40−55	30−40
12−18 years	30−55	25−40	20−30

Table 2

197

diagnoses of exclusion without any specific marker, so a diagnostic work-up for other potential causes of elevated bilirubin, e.g. inborn errors of metabolism, biliary atresia, choledochal cyst, is necessary. The aetiology of PNAC/IFALD remains unclear; possible causes include the toxicity of components of PN, lack of enteral feeding, continuous non-pulsatile delivery of nutrients and host factors, infection and sepsis.[15] In particular, the lipid component of PN has been implicated and many units now use alternative lipid management strategies such as: (i) decreasing the amount of lipid administered; (ii) use of a lipid emulsion of 10% fish oil; (iii) use of lipid emulsions containing a mixture of long- (LCT) and medium-chain (MCT) triglycerides; or (iv) use of mixed lipid emulsions such as mixtures of soybean, medium-chain, olive and fish triglycerides. In addition to lipid management, careful overall management of these patients under a multi-disciplinary team seems to be beneficial. Bowel lengthening procedures, such as the STEP procedure or longitudinal intestinal lengthening, may help transition to enteral nutrition, whereas in those with advanced liver disease and no prospect of enteral autonomy, transplantation may need to be considered.

Careful monitoring of patients on PN is mandatory. This is particularly important during the period over which PN is introduced. Full blood count, electrolytes, blood gases, urea/creatinine, glucose, calcium/phosphate, albumin, liver function tests (especially bilirubin) and cholesterol/triglycerides should all be monitored, together with vitamins and trace elements for long-term PN patients. PN should not be a 'one size fits all' prescription, but individual tailoring is recommended, dependent on clinical status, monitoring results and introduction of enteral nutrition.

Conclusion and future directions

Nutrition of surgical infants and children is complicated by the requirement for growth. Inadequate or unbalanced nutrition may lead to future problems for these children, and we are now able to start to improve nutrition delivery in order to optimize outcomes as well as survival. Despite advances in nutritional care, such as the multi-disciplinary approach, complications such as sepsis and cholestasis remain relatively frequent. Future research should be aimed towards the prevention and treatment of these complications. ◆

REFERENCES

1 Denne SC, Poindexter BB, Leitch CA, Ernst JA, Lemons PK, Lemons JA. Nutrition and metabolism in the high-risk neonate. In: Martin RJ, Fanarof AA, Walsh MC, eds. Fanaroff and Martin's neonatal-perinatal medicine. 8th. Philadeplhia, PA: Mosby-Elsevier, 2006; 661–93.

2 Hill AG, Hill GL. Metabolic response to severe injury. *Br J Surg* 1998; **85:** 884–90.

3 Koletzko B, Goulet O, Hunt J, Krohn K, Shamir R. Guidelines on paediatric parenteral nutrition of the European Society of paediatric gastroenterology, hepatology and nutrition (ESPGHAN) and the European Society for clinical nutrition and metabolism (ESPEN), supported by the European Society of paediatric research (ESPR). *J Pediatr GastroenterolNutr* 2005; **41**(suppl 2)**:** S1–87.

4 Mansour F, Petersen D, De Coppi P, Eaton S. Effect of sodium deficiency on growth of surgical infants: a retrospective observational study. *Pediatr Surg Int* 2014; **30:** 1279–84.

5 Kolacek S, Puntis JWL, Hojsak I, EEECwgopp nutrition. ESPGHAN/ESPEN/ESPR guidelines on pediatric parenteral nutrition: venous access. *Clin Nutr* 2018; **37:** 2379–91.

6 Ncepod. A mixed bag: an enquiry into the care of hospital patients receiving parenteral nutrition. In: Stewart JAD, Mason DG, Smith N, Protopapa K, Mason M, eds. London: national confidential enquiry into patient outcome and death, 2010.

7 Mesotten D, Joosten K, van Kempen A, Verbruggen S, EEECwgopp nutrition. ESPGHAN/ESPEN/ESPR guidelines on pediatric parenteral nutrition: Carbohydrates. *Clin Nutr* 2018; **37:** 2337–43.

8 Lapillonne A, Fidler Mis N, Goulet O, et al. ESPGHAN/ESPEN/ESPR/CSPEN guidelines on pediatric parenteral nutrition: Lipids. *Clin Nutr* 2018; **37:** 2324–36.

9 van Goudoever JB, Carnielli V, Darmaun D, Sainz de Pipaon M, EEECwgopp nutrition. ESPGHAN/ESPEN/ESPR guidelines on pediatric parenteral nutrition: Amino acids. *Clin Nutr* 2018; **37:** 2315–23.

10 Jochum F, Moltu SJ, Senterre T, et al. ESPGHAN/ESPEN/ESPR guidelines on pediatric parenteral nutrition: Fluid and electrolytes. *Clin Nutr* 2018; **37:** 2344–53.

11 Mihatsch W, Fewtrell M, Goulet O, et al. ESPGHAN/ESPEN/ESPR/CSPEN guidelines on pediatric parenteral nutrition: Calcium, phosphorus and magnesium. *Clin Nutr* 2018; **37:** 2360–5.

12 Bronsky J, Campoy C, Braegger C, EEECwgopp nutrition. ESPGHAN/ESPEN/ESPR/CSPEN guidelines on pediatric parenteral nutrition: Vitamins. *Clin Nutr* 2017; **37:** 2366–78.

13 Domellof M, Szitanyi P, Simchowitz V, Franz A, Mimouni F, EEECwgopp nutrition. ESPGHAN/ESPEN/ESPR guidelines on pediatric parenteral nutrition: Iron and trace minerals. *Clin Nutr* 2018; **37:** 2354–9.

14 Hartman C, Shamir R, Simchowitz V, et al. ESPGHAN/ESPEN/ESPR guidelines on pediatric parenteral nutrition: Complications. *Clin Nutr* 2018; **37:** 2418–29.

15 Carter BA, Shulman RJ. Mechanisms of disease: update on the molecular etiology and fundamentals of parenteral nutrition associated cholestasis. *Nat Clin Pract Gastroenterol Hepatol* 2007; **4:** 277–87.