

# Parenteral Nutrition in paediatrics

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## Abstract

The provision of nutrients intravenously or Parenteral Nutrition (PN), has been one of the biggest therapeutic progress over the last 50 years. PN has increased survival rates in children with gastro-intestinal and extra-intestinal diseases not tolerating oral or enteral nutrition. Moreover PN decreases malnutrition and therefore increases survival. PN related complications have been minimized thanks to advances in equipment, training and expertise, however, this treatment still has substantial risks. PN in paediatrics should be delivered by expert multidisciplinary nutrition team to ensure appropriate use, delivery, monitoring and weaning.

In this review we will take the reader through the ESPGHAN/ESPEN guidelines, at present the main reference for paediatric PN.

**Keywords** child; electrolytes; energy intake; fat emulsion; glucose; intravenous; minerals; nutritional requirement; parenteral nutrition; vitamins

## Indications

Acknowledging PN risks, every effort should be put in place to promote enteral feeding, PN is usually indicated when adequate nutrient supply cannot be provided enterally to prevent/correct malnutrition or to sustain appropriate growth.

The timing of PN depends on the clinical indications and on the patient's age and size. In the small premature baby just one day starvation could have serious consequences hence the need to start PN shortly after birth where it is clear enteral feeds won't be tolerated. In most cases though, there is little clinical or nutritional benefit in prescribing PN where the requirement is likely to be less than five to seven days. Up to this point the risks of PN outweigh nutritional benefit.

Table 1 summarizes some of the most common indication for PN in paediatrics across the different subspecialties.

## Venous access

### Peripheral access

The use of peripheral PN is strongly discouraged. There are associated risks of phlebitis and extravasation injury when hypertonic

solutions are administered peripherally. A peripheral access can be used only for fluids with an osmolarity <850 mOsm and Glucose concentration <12.5%, therefore the nutritional provision is compromised. A peripheral access with a suboptimal PN could be considered while waiting for a central line to be inserted.

### Central access

Initiation of PN requires the placement of a Central Venous Catheter, ideally via a dedicated line. The catheter should preferably be placed in the superior (or the inferior) vena cava and the catheter tip should sit as close as possible to the right ventricle (small infants at 0.5 cm and older/larger infants at 1 cm outside the cardiac outline at X-ray; in older children above the carina). Central lines are long-term devices to administer PN and if properly cared for can last months if not years.

If post PICU or surgery the patient may have a subclavian or jugular line in place. These are short term lines, only recommended to remain in place for 7 days (or up to 10 days at the clinicians' discretion). These can be triple or double lumens allowing one line to be dedicated to PN.

### PICC (Peripherally Inserted Central Catheter)

This line uses a subcutaneous vein as the entry site and is advanced to the central vein using aseptic technique. This type of line can be placed on the ward with or without sedation using local anaesthetic by an expert clinician and could last up to 2–3 years depending on the manufactures recommendations and functionality.

### Tunnelled central lines

Broviac or Hickman Central Venous Catheters are commonly placed if the patient is expected to be on long-term PN. These can be single or double lumens and are placed surgically. In order to minimize the infection risk and avoid accidental line removals these catheters are subcutaneously tunnelled and have a Dacron cuff implanted 1–2 cm from the cutaneous exit to anchor it to the subcutaneous tissue.

## Composition

PN composition in paediatric patients is formulated according to age, weight, clinical condition and requirements such as nutritional status, fluids and electrolyte imbalances and specific metabolic conditions.

PN is composed by caloric substrates (lipids and carbohydrates), aminoacids, ions, vitamins, micronutrients and water.

Tailoring the PN composition is the first step to a safe and successful artificial nutrition. PN should be prescribed by qualified personnel who understand its risks and potential complications. The prescription should ideally be carried out in discussion with a paediatric trained dietitian, pharmacist and paediatrician.

Generally speaking paediatric patients requires more energy comparatively to adults. The energy requirement is formulated according to size and age, clinical condition and level of activity. Table 2 shows the total parenteral energy needs for age group (Table 3).

### Carbohydrates

Carbohydrates are the main source of energy in nutrition and should provide for 40–60% of the total energy requirement. In

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## PN indications

### Extra-digestive indications:

**Neonatology** ⇒ premature baby <1.2–1.5 kg

**Metabolic disease** ⇒ end-stage liver disease, CF, congenital disorder of metabolism

**Haematology & oncology** ⇒ solid tumors, BMT, leukaemia

**Nephrology** ⇒ severe renal disease & tubulopathy, renal failure

**Hypercatabolism** ⇒ burns, polytrauma, post-surgery

### Digestive indications:

Malabsorption syndromes

Need for bowel rest

Congenital or acquired neonatal pathology of the GI tract

**Table 1**

PN the major source of calories is D-Glucose in the form of monohydrate dextrose. It offers 3.4 kcal/g and contributes to most of the osmolality of the PN solution. Even if central venous accesses can tolerate a solution up to 30–35% of dextrose, it is rare in clinical practice to use dextrose at more than 20% concentration. The upper limit of dextrose prescription is determined by its oxidation rate. The maximum oxidation velocity tolerated by young children is 12–15 mg/kg/min, which is higher compared to adults' (3–5 mg/kg/min).

If glucose is prescribed within this limit, its oxidation produces energy and glycogen; whereas any excess is directed to lipogenesis and fat deposition. Restoring fat deposits may represent a goal in refeeding a child but lipogenesis from excess glucose increases the energy expenditure and triggers steatosis, where the newly produced fatty acids are stored in the liver. Moreover, excessive glucose intake may contribute to increase CO<sub>2</sub> production and minute ventilation, worsening any respiratory acidosis in those patients with respiratory difficulties.

Protein metabolism is also impaired by excessive glucose intake, since for PN patients it is dependant on the composition of energy intake. Nitrogen balance is improved by reduced glucose intake and/or the addition of lipid emulsions.

It is important to control the glycaemia in patients on PN since it has direct effects on complications and mortality. PN induced hyperglycemia is associated with increased length of hospital stay and risk of complications such as infections. Hyperglycaemia alters the immune functions by impairing leucocyte function, phagocytosis and chemotaxis; and the inflammatory response by increasing inflammatory cytokines and oxidative stress.

## Parenteral energy needs

| Age (years) | Total kcal/kg of body weight/day |
|-------------|----------------------------------|
| Preterm     | 110–120                          |
| 0–1         | 90–100                           |
| 1–7         | 75–90                            |
| 7–12        | 60–75                            |
| 12–18       | 30–60                            |

**Table 2**

## ESPGHAN/ESPEN practical recommendations on starting glucose infusion

|            | Day 1 | Day 2 | Day 3 | Day 4 |
|------------|-------|-------|-------|-------|
| Up to 3 kg | 10    | 14    | 16    | 18    |
| 3–10 kg    | 8     | 12    | 14    | 16–18 |
| 10–15 kg   | 6     | 8     | 10    | 12–14 |
| 15–20 kg   | 4     | 6     | 8     | 10–12 |
| 20–30 kg   | 4     | 6     | 8     | <12   |
| >30 kg     | 3     | 5     | 8     | <10   |

**Table 3**

## Cyclical PN

Regular discontinuation of the PN infusion (cyclical PN) is well tolerated in children and should be prompted as soon as possible. Cyclical PN may lead to higher glucose infusion rates, causing hyperglycemia and a risk of hypoglycemia at discontinuation.

To avoid that the maximal glucose infusion rate should be <1.2 g/kg/h and the infusion rate should be increased/decreased in a stepwise fashion.

Cyclical PN has many advantages: alternating fasting to nutrition allows changes in the insulin/glucagon balance, reducing lipogenesis (in both subcutaneous tissue and liver). An early intervention with cyclical PN may help in protecting the liver when bilirubin (Brb) is increasing. Adult studies shown benefits from this practice when Brb is <20 mg/dl.

Being disconnected from the pump allows the patient to gain some physical activities with great influence on growth (protein synthesis) and psychological wellbeing.

## Practical points: glucose (Glc)

- In preterm infants: Glc infusion should start with 4–8 mg/kg/min  
Max Glc oxidation rate is 8.3 mg/kg/min
- In critically ill children Glc should be limited to 5 mg/kg/min
- In full term neonates and children up to 2 years Glc should not exceed 18 mg/kg/day
- Glc should provide 60–75% of non-protein calories
- Cyclical PN may be used from 3 to 6 months of age
- Stepwise increase and decrease is recommended at onset and discontinuation of glucose infusion rate
- Avoid hyperglycaemia causing glycosuria and/or hypoglycaemia
- Insulin use should be restricted to conditions where adaptation of glucose

## Lipids

Lipid solutions are the non-carbohydrate source of energy and they should contribute to 25–40% of the total caloric requirement of paediatric patients on PN. Lipids provide 9 kcal/g; more calories in a lower volume and with lower osmolality than carbohydrates. The use of lipids in PN reduces insulin secretion, CO<sub>2</sub> production and thermogenesis and they improve net-nitrogen balance. They are also a source for essential fatty acids (EFAs): omega-6 fatty acids (linoleic acid) and omega-3 fatty acids (linolenic acids).

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