FRACGP lecture series

Nutrition in childhood

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Importance of adequate nutrition

- Growth
- Brain development
- Tissue repair
- Immune system
- Metabolism
Incidence of acute protein-energy malnutrition in hospitalised children

- < 3 months: 63%
- 3 - 12 months: 73%
- 1 - 5 years: 38%
- 6 - 12 years: 41%
- 13 - 18 years: 44%

Clinical consequences of malnutrition

- Loss of muscle strength: skeletal, respiratory
- Increased incidence of fractures
- Increase in pressure sores
- Delayed wound healing
- Impaired immune function
- Impaired hormone function
- Impaired thermoregulation
- Impaired cardiovascular and gastrointestinal function
- Depression/apathy
- Increase in post-surgical complications
Adverse effects of catabolic state

- Negative nitrogen balance
- Muscle wasting
- Weakness
- Respiratory muscle failure
# Fluid maintenance requirements

<table>
<thead>
<tr>
<th>Body weight</th>
<th>Daily fluid volume</th>
<th>Hourly rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 – 10 kg</td>
<td>100 mls/kg</td>
<td>4 mls/kg</td>
</tr>
<tr>
<td>10 – 20 kg</td>
<td>1000 mls + (50 mls/kg)</td>
<td>40 + 2 mls/kg</td>
</tr>
<tr>
<td>20 or more</td>
<td>1500 mls + (20 mls/kg)</td>
<td>60 + 1 ml/kg</td>
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</tbody>
</table>
Estimation of energy requirements

- Altman-Ditmer equation (children < 1 yr)
- Schofield equation (children > 1 yr)
- Harris-Benedict equation (older children & adults)
- White equation (critically ill children > 2 yrs)
- Stress & activity factors
- Indirect calorimetry

- 90% of enteral requirements = parenteral requirements
## Energy requirements

<table>
<thead>
<tr>
<th>Age group</th>
<th>kcal/kg/d</th>
<th>kJ/kg·d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature infants</td>
<td>120-150</td>
<td>500-630</td>
</tr>
<tr>
<td>Neonates</td>
<td>100-120</td>
<td>420-500</td>
</tr>
<tr>
<td>1 - 12 months</td>
<td>90-100</td>
<td>400-420</td>
</tr>
<tr>
<td>1 - 6 years</td>
<td>75-100</td>
<td>320-400</td>
</tr>
<tr>
<td>7 - 12 years</td>
<td>60-75</td>
<td>250-320</td>
</tr>
<tr>
<td>12 - 18 years</td>
<td>30-60</td>
<td>125-250</td>
</tr>
<tr>
<td>Adults</td>
<td>30-40</td>
<td>125-170</td>
</tr>
<tr>
<td>Age group</td>
<td>Protein [g/kg·d]</td>
<td></td>
</tr>
<tr>
<td>-------------------------</td>
<td>-----------------</td>
<td></td>
</tr>
<tr>
<td>Premature infants</td>
<td>2.5 - 3.0</td>
<td></td>
</tr>
<tr>
<td>Infants (0 - 1 yr)</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>Children 2 - 13 yrs</td>
<td>1.5 - 2.0</td>
<td></td>
</tr>
<tr>
<td>Adolescents / adults</td>
<td>1.0 - 1.5</td>
<td></td>
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</tbody>
</table>
Protein energy malnutrition (PEM)

- Significant cause of mortality in the developing world (children)
- Complications:
  - sepsis
  - pneumonia
  - gastroenteritis
Reasons for PEM in developed countries

- Chronic illness, e.g.
  - chronic infection (CF, HIV, TB)
  - malignancy
  - malabsorption
- Difficult social circumstances / poverty
- Mental illness / psychological problems
- Child abuse
Marasmus

- Severe PEM
- Weight loss
- Loss of muscle and subcutaneous fat
- Bradycardia, hypothermia (severe)
- Oedema absent
Kwashiorkor

- Inadequate protein intake
- Reasonable carbohydrate and fat intake
- Oedema of lower limbs
- Muscle wasting
- Body weight does not represent degree of malnutrition (oedema)
Failure to thrive (FTT) is a term used to describe young children with inadequate weight gain. No valid and reliable definition exists. Protein-energy malnutrition (PEM) is one condition associated with FTT.
Micronutrient deficiencies

- **Vitamins**
  - water-soluble ($B_1, B_6, B_{12}, C, folate$)
  - fat-soluble ($A, D, E, K$)

- **Minerals and trace elements**
  (e.g. iron, copper, zinc, magnesium, selenium, iodine)
<table>
<thead>
<tr>
<th>Micronutrient</th>
<th>Clinical manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>Night blindness</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>Rickets</td>
</tr>
<tr>
<td>Vitamin K</td>
<td>Coagulopathy</td>
</tr>
<tr>
<td>Iron</td>
<td>Anaemia</td>
</tr>
<tr>
<td>Iodine</td>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>Zinc</td>
<td>Acrodermatitis enteropathica</td>
</tr>
</tbody>
</table>
Percentile curves

- Crossing of more than 2 percentile lines
- Correct for prematurity
- Weight falling below the 3rd percentile ('wasting')
- Fall-off in linear growth after ~3 months ('stunting')
- Head circumference not usually affected (intrauterine growth retardation or severe PEM)
Length

Weight

NORMAL
FAILURE TO THRIVE

Length

Weight
### Z scores

<table>
<thead>
<tr>
<th>Percentile</th>
<th>Z Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>3rd</td>
<td>-2</td>
</tr>
<tr>
<td>50th</td>
<td>0</td>
</tr>
<tr>
<td>97th</td>
<td>+2</td>
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</tbody>
</table>

**Z score formula:**

\[
Z \text{ score} = \frac{\text{actual weight} - \text{median weight}}{\text{standard deviation}}
\]
Z scores for weight-for-age in 18 infants with Multiple Food Protein Intolerance

Non-FTT (n = 14)
FTT (n = 4)
Body mass index

BMI = \frac{\text{Weight [kg]}}{\text{Height [m]}^2}

BMI (Adults) Categories

- < 18.5: Underweight
- 18.5 - 24.9: Ideal
- 25.0 - 29.9: Pre-obese
- 30.0 - 34.9: Obese Class I
- 35.0 - 39.9: Obese Class II
- > 40.0: Obese Class III
Body composition

- Anthropometry
  - skin folds
  - mid-arm circumference

- Bioimpedence studies

- Dilution methods (total body K)
  Bone densitometry
Nutritional assessment

◆ Estimation of energy requirement
  - Activity factors
  - Stress factors
  - Catch-up growth

◆ Assessment of dietary intake
  - 3-day diary

◆ Specific nutrient requirements
Malnutrition and immunodeficiency

- Poor nutrition
- Increased susceptibility to infection
- Infection
- Deterioration of nutritional status

Increased susceptibility to infection leads to infection, which leads to poor nutrition, which leads to deterioration of nutritional status, which leads to increased susceptibility to infection.
Micronutrients and immune function

Micronutrients that affect immune function:
- Zinc
- Selenium
- Iron
- Copper
- Magnesium
- Vitamin A
- Vitamin C
- Vitamin E
- Vitamin B6
- Folic acid

- Antioxidants
- Co-factors for metalloenzymes involved in nucleic acid synthesis and cell replication
- Other specific direct effects on immune functions
Zinc and vitamin A deficiency

MORTALITY

<table>
<thead>
<tr>
<th>Condition</th>
<th>Zn</th>
<th>Vit A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diarrhoea</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>HIV</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>Measles</td>
<td>↓</td>
<td>↓</td>
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</tbody>
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Selenium deficiency

Keshan disease

- Cardiomyopathy
- Keshan province in China
- Endemic selenium deficiency
- Coxsackie B virus infection
- Selection of more virulent strain of virus
- ? Antioxidant deficiency
**Thymus and T-lymphocytes**

- Immature bone marrow-derived T-cell precursors
- Thymus
  - Maturation of T-cells in cortex
- Thymic hormones
- Cytokines
- Chemokines
- Stem cell factor
- Migration to peripheral lymphatic tissues
- Mature lymphocytes in medulla

Thymus and malnutrition

- Thymic atrophy (lymphoid compartment)
- Occurs in protein, zinc, magnesium, iron and vitamin deficiency states in children
- Severe thymic atrophy with cortical thymocyte depletion (autopsy findings)
- Massive apoptosis of \( \text{CD}4^+\text{CD}8^+ \) cells
- Decreased thymocyte proliferation
- Process reversible after nutritional rehabilitation

Thymic atrophy

- Thymus size in severely malnourished children
- Weekly mediastinal ultrasound
- Smaller thymus
- More immature lymphocytes and fewer mature T-lymphocytes
- Nutritional rehabilitation within 1 month
- Thymic recovery after about 2 months
- Thymic size as marker of immunological recovery?

P Chevalier et al., Santé 1996;6:201-208
Leptin

- Leptin is an adipocyte-derived hormone
- Regulates food intake
- Low leptin levels during starvation and malnutrition
- Metabolic and endocrine functions
- Regulates immunity, inflammation and haematopoiesis
- Similarities with pro-inflammatory cytokines
- Proliferative and anti-apoptotic effect on T-lymphocytes, leukaemia cells and stem cells
- Affects macrophage/monocyte activation
Hormonal control of thymus

- Gluco-corticoid levels increased in PEM
- Leptin levels decreased in PEM
- Imbalance of glucocorticoid and leptin homoeostasis

Low-birth weight infants

- Birth weight < 2500g
  - Western countries 7%
  - Developing world 40%
- Upper and lower respiratory infections
  3 times more frequent
- Decreased levels of maternal IgG (placenta)
- Thymic atrophy and impaired cellular immunity
- Reduced thymopoietin levels
- Decreased number of T-lymphocytes
- Persists for several months or even years
- Zn supplementation

RK Chandra et al., Lancet 1992; 340:1124-1127
Short bowel syndrome
Definition of SBS

- Malabsorptive state following extensive small intestinal resection or congenital anomalies
- Malabsorption prolonged or permanent if resection > 50% of small intestine
Aetiology of SBS

**Neonates**
- NEC
- Gastrochisis
- Intestinal atresias
- Midgut volvulus
- Hirschsprung’s disease

**Children and adults**
- Extensive Crohn’s disease
- Abdominal radiation
- Mesenteric infarction
- Abdominal trauma
INTESTINAL FAILURE

- Short bowel syndrome
  - Small bowel resection
  - Extensive Crohn’s disease

- Persistent motility disorders
  - Long segment Hirschsprung’s disease
  - Intestinal pseudo-obstruction
  - Mitochondrial disorders

- Transport defects
  - Congenital chloride diarrhoea
  - Glucose-galactose malabsorption

- Epithelial dysplasias
  - Microvillus inclusion disease
  - Tufting enteropathy
Normal jejunal mucosa

Normal small intestinal function

- Absorptive surface
  - Villus height
  - Lymphatics
  - Disaccharidases
  - specific transporters
  - barrier function

- Peristalsis
- Gut hormones
- Bacterial milieu
Intestinal adaptation

A  Normal mucosa  B  Villus and crypt hyperplasia
Intestinal adaptation

Morphologic

Functional

Clinical outcome
Colonic function

Reabsorption of

- Water
- Sodium
- Fermented SCFA

- “Ileal break”
- Ileocaecal valve
- Continence
Approach to the treatment of SBS

- Optimise mechanical aspects of gastrointestinal function
- Enhance intestinal adaptation
- Limit complications of parenteral nutrition therapy
Optimise mechanical aspects of gastrointestinal function:

- Increase mucosal surface area
- Slow intestinal transit
- Improve motility
- Relieve mechanical or functional obstruction
- Small bowel transplantation
Adaptation in gastrointestinal function

- peristaltic function
- digestive function
- absorptive function
- gastrointestinal hormone receptor interactions
- apoptosis
- mucosal barrier function
Complications of SBS

- Loss of absorptive surface -> malabsorption
- Growth failure / short stature
- Electrolyte imbalance / dehydration
- Micronutrient deficiencies
- Gastric acid hypersecretion
- Cholelithiasis
- Chronic liver disease
- Metabolic bone disease
- PN-related complications
**Bacterial overgrowth**

- Intestinal stasis / dysmotility
- Absent ileocaecal valve
- Chronic diarrhoea
- D-lactic acidosis

\[ \text{Ix: Breath } H_2 \text{ test} \]
\[ \text{Urine metabolic screen} \]
\[ \text{Culture duodenal aspirate} \]
Common micronutrient deficiencies in SBS

- **Vitamins**
  A, D, E, K, B12

- **Minerals**
  Zinc, Magnesium, Calcium, Selenium
Prognostic factors

- Length of remaining bowel
- Peristaltic function
- Presence of ileocaecal valve and colon
Clinical outcome in children with SBS

- **Long-term survival in > 80%**

- **Mortality**
  - Sepsis
  - Cholestatic liver disease
  - Metabolic complications
  - Lack of central venous access sites

- **Quality of life**
ENTERAL NUTRITION
Enteral formulas in SBS

- Human milk
- Polymeric infant formulas
  - cow’s milk or soy formulas
- Extensively hydrolysed formulas
  - casein (Pregestimil, Nutramigen)
  - whey (Alfaré)
- Elemental formulas (amino acid-based)
  - Neocate or Paediatric Vivonex
- Polymeric adult formula
  - Osmolyte, Pediasure, Ensure plus
Feeding tubes

- Nasogastric
- Nasojejunal
- Gastrostomy
- Gastrojejunal
- Jejunostomy
Enteral formulas in SBS

- **Specific nutrients**
  - Protein content
  - CHO base (lactose-free)
  - MCT fat

- **Route of delivery**
  - oral
  - tube

- **Mode**
  - bolus vs continuous
Modular formulas

- **Protein source**
  - polymeric (protein intact)
  - hydrolysed (extensively or partially)
  - elemental (amino acid-based)

- **Lipid**
  - MCT
  - LCT

- **Carbohydrate**
  - Glucose polymer
  - Fructose
Gastrostomy

- Surgical gastrostomy (Stamm)
- Percutaneous Endoscopic Gastrostomy (PEG)
  - tube
  - low-profile device (button)
Parenteral nutrition in infants and children
Historical overview of PN

- **1950s** Development of isotonic glucose-protein hydrolysate solution

- **1960s** Infusion of hypertonic glucose protein hydrolysate solution into SVC (beagles)

- **1968** First clinical use in infant with neurological impairment (normal growth while on PN for 22 mo)
General indications for PN

- No oral intake
- Anticipated need for PN
  > 2 days neonate
  > 5 days child
- Weight loss of >10% or failure to gain weight in a patient with a poorly or non-functioning GIT
Extra-intestinal indications for PN

- Premature infants
- Major surgery
- Severe sepsis
- Acute organ failure
- Extensive burns
- Inborn errors of metabolism
Gastrointestinal indications for PN

INTESTINAL FAILURE

Short bowel syndrome
Intractable diarrhoea
Chronic intestinal pseudo-obstruction
Inflammatory bowel diseases
Special considerations for paediatric patients on PN Therapy

- Sensitive to fluid shifts
- Require close monitoring
- Blood volume limitations for monitoring
- Higher incidence for technical and septic complications
- Lack of oral stimulation
- Psychological impact
Fluid volume restriction

- Congestive heart failure
- Cerebral oedema
  - head injury
  - meningitis
  - fluid overload
- Respiratory failure
- Sepsis
What is PN?

**Nutrient Solution**
- Amino acids
- Glucose
- Electrolytes
- Minerals
- Vitamins
- Trace elements
- Heparin
- Water

**Lipid Emulsion**
- +/- fat soluble vitamins

> 50 ingredients
Energy content of macronutrients

- 1 g amino acid / protein = 4 kcal
- 1 g glucose = 4 kcal
- 1 g lipid = 10 kcal
Neonatal solutions

- Vaminolact® and Primene®
  - suitable for premature infants with high protein requirements
  - modelled on cord blood AA profile
  - reduced phenylalanine levels and risk of toxicity
  - able to deliver higher dose of AA/kg
  - no prospective data on outcomes
Lipid emulsions

- **Intralipid 10%, 20% & 30%**
  - soybean oil
  - long-chain triglycerides
  - egg phospholipid / emulsifier

- **Clinoleic 20%**
  - olive oil 80% and soybean oil 20%
  - mono- and polyunsaturated fats > 80%

- **MCT / LCT formulations (Lipofundin)**

- **Structured lipids (Structolipid)**
Electrostatic forces in lipid emulsion

STABLE

UNSTABLE
Example 1

- Premature infant 34/40 – BWt 1680 g
- Fluid requirement 100 mls/kg = 168 mls/d
- EER 90-95 kcal/kg·d (380-400 kJ/kg·d)

Energy provided
N 55 kcal/kg·d
L 40 kcal/kg·d
95 kcal/kg·d

Protein 2g/kg·d

Vamin N 20/125: 100 mls/kg·d
Intralipid 20%: 4 g/kg·d
Cycle PN 3 : 1
Example 2

- 8 yr old boy with GVHD – Wt 25 kg
- Fluid requirements $1000 + 500 + 5\times20 = 1600$ mls/d
- Platelet count $12\times10^6$/L
- EER 75 kcal/kg·d (315 kJ/kg·d)

Synthamin 25/200: 70 mls/kg·d

Intralipid 20%: 0.5 g/kg·d

No need to cycle PN

Energy provided

<table>
<thead>
<tr>
<th>N</th>
<th>61 kcal/kg·d</th>
</tr>
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<tbody>
<tr>
<td>L</td>
<td>5 kcal/kg·d</td>
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</tbody>
</table>

| Protein | 1.8 g/kg·d |
Complications of PN

- Metabolic complications
- Micronutrient deficiencies and excess
- Thrombosis and line-related complications
- PN-associated cholestasis
Metabolic complications of PN

- Hyperglycaemia / glycosuria
- Hypertriglyceridaemia
- Electrolyte imbalances
- Acid-base disturbances (acidosis)
- Hypophosphataemia (? Refeeding syndrome)
- Micronutrient deficiency or toxicity (Zn, Se, Fe, Cu, Vitamins A & E)
Adverse effects of catabolic state

- Negative nitrogen balance
- Impaired tissue repair
- Muscle wasting
- Weakness
- Respiratory muscle failure
Central Venous Access Devices are percutaneously or surgically placed catheters that are inserted into the large central veins of the body in order to provide easy access to the venous system.
Clinical use of CVAD

- Delivery of
  - medications
  - IV fluids / resuscitation
  - blood products
  - concentrated nutrient solutions

- Monitoring of haemodynamic variables
  - central venous pressure

- Repeated blood sampling
Complications

- **Mechanical (5-19%)**
  - arterial puncture
  - haematoma
  - hemothorax
  - pneumothorax

- **Infectious (5-26%)**
  - catheter colonisation
  - catheter-related blood-stream infection
  - exit-site infection

- **Thrombotic (2-26%)**

McGee & Gould, N Engl J Med 2003, **348:**1123-1133
Clinical considerations

- **Anatomy**
  - Thrombosis of central veins

- **Clinical aspects**
  - Age of patient
  - Location of CVAD (neck vs femoral)
  - Number of medications required
  - Duration of treatment
  - Monitoring of central venous pressure
  - Repeated blood sampling
PICC

- Peripheral inserted central catheter
- Inserted from cubital fossa
- Tip in SVC/right atrium
- Short-term venous access during intensive treatment
Antimicrobial-impregnated catheters

- Catheters coated with antimicrobial layer:
  - silver sulfadiazine
  - chlorhexidine
  - minocycline
  - rifampicin

- Significant reduction of infection rate
  - 7.6 to 1.6 infections per 1000 catheter-days

- Cost saving (USD 196 per catheter)

Tunnelled catheters

- Silicone catheters (Hickman®, Broviac®)
- Surgical placement
- Tunnelled underneath skin
- Reduced rate of entry site infection
- Medium to long-term use
- Chemotherapy, TPN
Ports

- Implantable chamber (surgical)
- Hidden under skin
- Access with needle into chamber
- Home TPN
- Chemotherapy
Umbilical catheters

- Used in neonatal ICU
- Inserted via umbilical vein
- Tip positioned in IVC
- Administration of medications and blood products
- Short-term
- Significant risk of infection and thrombosis
Line complications

- CVC sepsis
  - gram-negative organisms (? translocation)
  - gram-positive organisms
  - fungal (Candida albicans)

- Line occlusion
  - Fibrin and calcium deposits
  - Clot formation (? repeated blood sampling)

- Extravasation of PN
Prevention of CVC sepsis

- Strict aseptic line care
- Prophylactic antibiotic flushes
- Prevention of thrombosis
Central vein thrombosis in long-term PN

- Underrecognised problem in patients receiving long-term PN
- Aetiology unclear
- Often occurs in relation to CVC sepsis
  - ? hypercoagulability
  - ? other factors
- Loss of vascular access sites long-term
- ? Routine anticoagulation in long-term PN
PN-associated cholestasis

- PN-cholestasis affects 40-60% of infants requiring long-term PN for intestinal failure

- **Clinical spectrum**
  - cholestasis
  - cholelithiasis
  - hepatitis fibrosis
  - biliary cirrhosis
  - portal hypertension
  - chronic liver failure

- Aetiology multifactorial

Risk factors for PN cholestasis

- Premature infants or neonates
- Recurrent episodes of (CVC) sepsis
- Previous gastrointestinal surgery
- No oral intake (TPN)
- Lipid emulsion toxicity (RES overload)
  Colomb V et al., JPEN 2000;24:345-50
- Manganese toxicity
Prevention and treatment of PN cholestasis

- Early “minimal” enteral feeding
- Prevention of sepsis
- Cycling of PN
- Avoid energy overload
- ? Ursodeoxycholic acid
- ? Cholecystokinin
- Liver transplantation
Clinical outcome of long-term PN

- **Long-term survival**
  - 1 yr
    - Short bowel syndrome: 94%
    - Motility disorders: 84%
  - 4 yr
    - Short bowel syndrome: 80%
    - Motility disorders: 70%

- **Mortality**
  - Sepsis
  - Liver failure
  - Lack of central venous access sites

- **Quality of life ?**
Clinical outcome in short bowel syndrome

- 44 neonates with small intestinal resection
- PN-dependent for at least 3 months
- 27 / 44 patients off PN < 36 mo
  - 7 / 44 PN-dependent > 36 mo
  - 6 / 44 on PN (younger than 36 mo)
  - 4 / 44 died
- Main predictors of PN dependence:
  - residual bowel length
  - % of energy intake via enteral route

Sondheimer JM et al., J Pediatr 1998;132:80-4
Clinical outcome in short bowel syndrome

- 30 children with SBS studied (1986-1998)
- **Factors associated with shorter duration of PN:**
  - enteral feeding with breast milk
  - enteral feeding with amino acid-based formula
  - longer residual small bowel length
  - % calories received enterally at 6 weeks post surgery
- **Multivariate analysis:**
  - residual small bowel length only independent predictor of PN duration

Andorsky DJ et al., *J Pediatr* 2001;139:27-33
Paediatric intestinal transplantation

- **Indications**
  - Progressive PN-associated liver disease
  - Recurring sepsis (gut-related)
  - Impending loss of central venous access
  - Extreme short bowel
  - Congenital intractable epithelial disorders

- **Contraindications**
  - Profound neurological disabilities
  - Other life-threatening non-correctable diseases
  - Severe immunodeficiencies
  - Non-resectable malignancies
  - Insufficient patency for central vascular access
    > 6 mo post SBTx

Kaufman SS et al., *Pediatr Transplantation* 2001;5:80-87
Paediatric intestinal transplantation

- Reserved for a small group of patients with life-threatening complications of long-term PN
- Overall 50-60% long-term survival
- Survival somewhat better in isolated intestinal graft compared with intestinal-liver or multivisceral Tx
- Graft rejection and lympho-proliferative disease common
- 75% of survivors could cease PN within 2 yrs of ITx
- Outcomes improving due to better post-op care and immunosuppression

Kaufman SS et al., *Pediatr Transplantation* 2001;5:80-87
Home parenteral nutrition

- Children account for 15-20% of HPN programs
- Most common indications \((n=300)\)
  - Short bowel syndrome \(50\%\)
  - Congenital intractable diarrhoea \(15\%\)
  - Chronic intestinal pseudo-obstruction \(15\%\)
  - Other (inflammatory bowel disease, AIDS, metabolic diseases) \(20\%\)
- Usually parent-administered
- Requires close monitoring by multi-disciplinary nutrition support team
- Despite being expensive provides significant cost saving (compared to long-term hospitalisation or transplantation)

**Colomb V et al., Nutrition 1999;15:172-3**
CVC infection in Home PN

- 47 children on HPN (207 CVC-years)
- Age 8.1 ± 5.0 yrs
- Half of hospitalisations due to CVC infection (mainly coagulase-negative *Staphylococcus*)
- No risk factors identified
- Incidence of CVC infection: 2.1 / 1000 HPN days

*Colomb V et al., Clin Nutr 2000;19:355-9*