5.10 Nutritional disorders

5.10.A Vitamin or mineral deficiencies

**BOX 5.10.A.1 Minimum standards**
- Adequate diet.
- Vitamins A, B, C, D and K.
- Folic acid.
- Zinc.
- Iodine.

**Vitamin A deficiency (VAD)**

**Significance**
- Vitamin A deficiency is the single most important cause of childhood blindness in resource-limited countries.
- It makes a significant contribution to morbidity and mortality from common childhood infections, even at subclinical levels of deficiency.
- A Cochrane review indicates that regular vitamin A supplementation reduces mortality by 24%.

**Prevalence**
- Vitamin A deficiency is endemic in at least 60 countries worldwide, especially in Africa, South and South-East Asia, some areas of South America and the Western Pacific.
- Around 250 million preschool children are at risk.
- It causes 250,000–500,000 cases of blindness per year.

**Good food sources** are red palm oil, mango, pawpaw, dark green leafy vegetables, unskimmed milk, eggs and liver.

**Aetiological factors**
- Persistent inadequate intake of vitamin A exacerbated by insufficient consumption of dietary fat, leading to ineffective absorption.
- Frequent infections, especially measles, gastroenteritis and respiratory infections, resulting in decreased food intake, malabsorption, increased urinary loss, and increased utilisation of vitamin A by the body resulting in depletion of liver stores. The decrease in vitamin A levels in the body in turn predisposes children to infection, and so a vicious cycle is set up.
- Vitamin A deficiency is common in the context of poverty, social under-development, hostile living environments, water shortage and food scarcity, and individual factors such as lack of breastfeeding, inappropriate weaning practices and increased physiological needs during periods of rapid growth.

**Clinical effects**
- Night blindness (decreased ability to generate rhodopsin in the retinal rod photoreceptors essential for vision in dim light).

- Compromised integrity of epithelial surfaces due to loss of mucus-producing goblet cells, leading to ‘dry eye’ (conjunctival xerosis), Bitot’s spots, corneal xerosis, corneal ulceration, and irreversible damage to the eye (keratomalacia).
- Depressed immunity (both innate and adaptive immunity), which results in increased susceptibility, duration and severity of common infections (e.g. acute respiratory infection, diarrhoea, measles).
- Poor growth, apathy and slow development.

**TABLE 5.10.A.1 Signs of vitamin A deficiency in the eyes**

<table>
<thead>
<tr>
<th>Sign</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Night blindness</td>
<td>Inability to see in dim light (e.g. at dawn or dusk). Often occurs in the later part of pregnancy</td>
</tr>
<tr>
<td>Conjunctival xerosis</td>
<td>The conjunctiva looks dry and slightly rough instead of smooth and shiny</td>
</tr>
<tr>
<td>Bitot’s spots</td>
<td>White foamy patches on the conjunctiva. Not always present</td>
</tr>
<tr>
<td>Active corneal lesions:</td>
<td>At this stage the condition can worsen within a few hours and complete or partial blindness can result</td>
</tr>
<tr>
<td>Corneal xerosis</td>
<td>The cornea looks dry and cloudy</td>
</tr>
<tr>
<td>Ulcers on the cornea</td>
<td>Often on the edge of the cornea</td>
</tr>
<tr>
<td>Keratomalacia</td>
<td>The cornea is cloudy and soft like jelly. Rare</td>
</tr>
</tbody>
</table>

**Assessment of vitamin A status**

There are no simple tests for vitamin A deficiency, but it is likely to affect communities where vitamin-A-rich food is scarce and infection and/or malnutrition rates are high.

- Vitamin A deficiency becomes a public health problem when the following are prevalent in the child population:
  - night blindness (> 1%)
  - Bitot’s spots (> 0.5%)
  - corneal xerosis with or without ulceration (> 0.01%)
  - corneal scarring (> 0.05%).

**Prevention**
- Encourage the use of local foods rich in vitamin A.
  - Provide dietary education about vitamin-A-rich foods (e.g. dark green leafy vegetables, carrots, mango, papaya, eggs, orange fruits, liver, red palm oil, fatty fish).
  - Treat the siblings and mother. Mothers are especially vulnerable to vitamin A deficiency, and should be supplemented in the first month of lactation.
Give regular supplementation every 4 to 6 months as described in Table 5.10.A.2.
Prevent recurrent infections by recommending the use of impregnated nets, deworming, using clean water and breastfeeding.

**TABLE 5.10.A.2 Vitamin A supplements to prevent vitamin A deficiency**

<table>
<thead>
<tr>
<th>Target group</th>
<th>Immunisation contact</th>
<th>Vitamin A dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants under 6 months who are not breast fed or breast fed infants whose mothers have not received vitamin A supplements.</td>
<td></td>
<td>50,000 IU</td>
</tr>
<tr>
<td>Infants aged 6–11 months</td>
<td>Measles vaccine contact</td>
<td>100,000 IU</td>
</tr>
<tr>
<td>Children aged 12–59 months</td>
<td>Booster doses</td>
<td>200,000 IU</td>
</tr>
<tr>
<td></td>
<td>Special campaigns</td>
<td>every 4 to 6 months</td>
</tr>
</tbody>
</table>

Regular vitamin A supplementation is advised for all children in resource-limited countries. It has been shown to reduce all causes of mortality, and especially mortality from diarrhoea.

If a child has malnutrition, severe diarrhoea or measles, give one high-dose vitamin A capsule, according to Table 5.10.A.3, unless they have received a dose in the previous month.

**Treatement**
If there are any eye signs, give vitamin A as indicated in Table 5.10.A.3.

**TABLE 5.10.A.3 Doses of vitamin A for treatment of clinical deficiency**

<table>
<thead>
<tr>
<th>Age</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Two weeks later</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 6 months</td>
<td>50,000 IU</td>
<td>50,000 IU</td>
<td>50,000 IU</td>
</tr>
<tr>
<td>6–12 months</td>
<td>100,000 IU</td>
<td>100,000 IU</td>
<td>100,000 IU</td>
</tr>
<tr>
<td>&gt; 12 months</td>
<td>200,000 IU</td>
<td>200,000 IU</td>
<td>200,000 IU</td>
</tr>
</tbody>
</table>

If there are ulcers or the eyes look soft or cloudy, instil atropine 0.1%, three times a day for 3–5 days, and a topical antibiotic. Cover the affected eye with a saline-soaked bandage.

Deep IM injection of vitamin A (retinyl palmitate) 50000 IU for children under 2 years of age, and 100000 IU for those over 2 years, should be given if severe stomatitis, persistent vomiting or malabsorption are present.

**Vitamin B, deficiency: beriberi**

- This may occur in areas of severe nutritional deprivation where little more than polished rice is consumed. It is uncommon in Africa, as the staple is maize or wheat, which contains vitamin B.

- It affects adults, children and breastfed infants of thiamine-deficient mothers.
- It is often mistaken for oedematous malnutrition (kwashiorkor), nephritis, cerebral malaria, encephalopathy or septicaemia.
- It causes wet (cardiac) or dry (neurological) beriberi:
  - cardiac failure with breathlessness, oedema and tachycardia
  - peripheral neuritis, with tingling and burning of feet, and reduced tendon reflexes
  - acute encephalopathy and coma.
- An aphonic form is characterised by a noiseless cry due to laryngeal nerve paralysis.

**Beriberi is rapidly fatal.**
- The initial dose is 50–100 mg thiamine hydrochloride, IM or orally. This is particularly effective in heart failure (facilities for treating anaphylaxis must be available).
- Continue with 10 mg/day for children under 2 years of age, 25 mg/day for those aged 2–12 years, and 50 mg/day for those over 12 years for 3–4 days.
- Patients with beriberi often have other B vitamin deficiencies.
- Good food sources of vitamin B are pork, whole grain cereals, legumes, nuts and liver.

**Nicotinic acid (niacin) deficiency: pellagra**

Nicotinic acid is synthesised from the essential amino acid tryptophan, and pellagra is found where the diet is deficient in either nicotinic acid or tryptophan. It is common where maize is the staple diet, as in many parts of Africa. Maize is deficient in tryptophan, and the nicotinic acid is bound and unavailable.

**Clinical features**

- Dermatosis of parts of the skin exposed to sunlight, namely the neck (Casal’s necklace), face and hands, usually seen in children over 5 years.
- Diarrhoea and malabsorption.
- Encephalopathy, which is rare in children.

**Treatment**

- Nicotinic acid:
  - 10 mg three times daily for 7 days in children under 2 years of age
  - 25 mg three times daily for 7 days in children over 2 years. In severe cases give 100 mg IV.
- Treat other B vitamin deficiencies at the same time (thiamin and riboflavin).
- Improve the diet with protein and green vegetables, peanuts, whole grain cereals, meat, fish, chicken and liver.

**Vitamin C deficiency: scurvy**

This usually presents at the age of 4–10 months. Cow’s milk is low in vitamin C.

- Vitamin C is needed for collagen formation (in bones, cartilage, teeth and capillary walls).
- It is important for the healing of wounds.
- It increases iron absorption.
- It is found in citrus fruits, vegetables and breast milk.
Very little vitamin C is present in cow’s milk, especially if it is heated.
- Vitamin C deficiency is found in severe malnutrition and in children fed on very poor diets in institutions.

Clinical features
- Spontaneous haemorrhages, especially from gums, and defective bone, cartilage and dentine formation.
- Local tenderness and swelling of the legs (due to subperiosteal haemorrhages), which may present as irritability when the child is picked up or moved.
- Pseudo-paralysis of the limbs.
- Haemorrhagic and spongy changes in the gums.
- Petechiae and ecchymoses around the eyes.
- Microscopic haematuria may be present.
- The anterior ends of the ribs swell.
- Mild anaemia.
- Increased risk of fractures.
- Poor healing of fractures and wounds.
- Characteristic X-ray appearance: loss of trabeculae in long bones gives a ground-glass appearance, dense lines of calcification in the epiphysis next to the epiphyseal plate and calcification of subperiosteal haemorrhages.

Treatment
- By mouth
  - Child 1 month–4 years 125–250 mg daily in 1–2 divided doses
  - Child 4–12 years 250–500 mg daily in 1–2 divided doses
  - Child 12–18 years 500 mg–1 g daily in 1–2 divided doses.
- A subsequent improvement in diet is needed, with plenty of fresh fruit and vegetables.

Vitamin D₃ deficiency: rickets
Vitamin D deficiency causes the following:
- rickets (failure of mineralisation of growing bone)
- hypocalcaemic tetany in infancy
- osteomalacia in adults.

Nutritional rickets is most prevalent in North Africa, the Middle East and Pakistan, Asian and Afro-Caribbean children are also at risk in the UK and other countries where there is limited sunshine. Vitamin D deficiency is unusual in African children over 18 months, as at this age they can walk and therefore go out into the sunshine. Older children in Africa with rickets must be investigated for causes of rickets other than vitamin D deficiency, such as dietary calcium deficiency or inherited forms of hypophosphataemic rickets.

Biochemistry
- Vitamin D increases Ca²⁺ absorption from the gut, reabsorption of Ca²⁺ from the kidney, and a phosphate diuresis.
- Vitamin D deficiency reduces Ca²⁺ and increases parathyroid hormone (which increases phosphate loss by the kidney), resulting in low Ca²⁺ and low phosphate levels. Subsequently there is a rise in alkaline phosphatase and then the X-ray features of rickets occur.

Aetiology
- Prolonged breastfeeding, especially if the mother is vitamin D deficient.
- Lack of vitamin-D-containing foods such as oily fish, eggs, butter and margarine.
- Lack of sunlight exposure (UV light) (black- and brown-skinned children living indoors or in countries where there is little sunlight are particularly at risk).
- An infant’s diet contains only small amounts of vitamin D, so fortification of foods and vitamin D supplementation is recommended.
- If a child presents with rickets and has normal exposure to sunlight, consider a hypocalcaemic diet (reported in South Africa and Nigeria). Cereals can bind calcium and prevent its absorption.
- Rarely, there is a metabolic disorder such as familial hypophosphataemic rickets. Where consanguinity is common, renal tubular disorders can produce this.
- Vitamin D deficiency also occurs in chronic renal and liver failure.

Clinical features
- 1,25-Dihydroxyvitamin D crosses the placenta, and the neonate generally has sufficient levels for the first few months of life.
- Disturbance of the normal growth of the epiphyseal plate leads to the formation of inadequately calcified new bone at the diaphysis edge of the plate (so-called osteoid tissue). The proliferating zone on the epiphyseal side of the plate enlarges excessively, producing a swelling of the plate. Osteoid tissue may also form subperiosteally. There is also demineralisation of the skeleton. The following features result from these abnormalities:
  - epiphyseal swelling (especially distal radii at the wrists, and also the ankles and knees)
  - craniotabes (soft areas of the skull bones, especially of the occiput, which when pressed gently are easily depressed)
  - rickety rosary (enlarged costochondral junctions)
  - delayed fontanelle closure
  - curvature of the shafts of the tibia and femur (may occur in severe cases)
  - bossing of the frontal and parietal skull bones due to osteoid formation
  - pigeon chest (pectus carinatum)
  - Harrison’s sulci
  - deformities of the thoracic and lumbar spine can produce kyphoscoliosis and lumbar lordosis
  - pelvic bone deformities in female children can lead to subsequent birthing difficulties due to damage to the inlet and outlet of the birth canal
  - delayed dentition
  - delayed gross motor development with generalised muscle weakness and hypotonia
  - growth retardation
  - occasionally, especially in infants, symptoms of hypocalcaemia.

Diagnosis
- Very elevated plasma alkaline phosphatase activity.
- Usually normal, but possibly slightly low, plasma calcium levels.
- Very low plasma phosphate levels.
Lowered plasma levels of 25-hydroxyvitamin D₃, but often this cannot be tested for.

The best sites to radiologically assess for rickets are those where there is rapid bone growth, namely the wrists and knees.

- Typical X-ray appearance: cupping and fraying of the distal ends of the long bones, such as the ulna and radius.
- There is widening of the metaphyseal plate due to osteoid formation.
- The periosteum may be raised.
- There may be abnormal curvature of bones and generalised under-calcification.

**Prevention measures**

- Exposure to sunlight and foods such as egg yolk, milk and fortified margarine.
- Vitamin D₃ (ergocalciferol) supplementation, 400–600 IU daily.

**Vitamin K deficiency**

- Vitamin K is a cofactor for the hepatic synthesis of clotting factors (prothrombin, and factors VII, IX and X).
- Sources are green leafy vegetables, meat, liver, cheese, and synthesis by gut flora.
- Deficiency may occur as a result of the lack of bile salts and the malabsorption of fats after the use of broad-spectrum antibiotics, or in the breastfed newborn whose gut is not yet colonised with bacteria and therefore does not produce vitamin K.
- Treat bleeding due to vitamin K deficiency with 250–300 microgram/kg (max 10 mg) IV; neonates 1 mg. Repeat doses every 8 hours if needed.
- Prevent haemorrhagic disease of the newborn by giving 1 mg vitamin K to all newborn infants either orally or IM (preterm 400 microgram/kg maximum dose 1 mg).

**Folic acid deficiency**

- The most important issue here is that women who are deficient in folic acid at the time of conception and in early pregnancy are at increased risk of having a baby with a neural tube defect (spina bifida or anencephaly).
- Relative deficiency occurs in haemolytic anaemias and in preterm infants (see Section 3.3 and Section 5.11.C).
- Deficiency occurs in malabsorption syndromes such as coeliac disease and blind loop syndromes.
- Anticonvulsants such as phenytoin may interfere with the metabolism of folic acid.
- Consequences of folic acid deficiency include the following:
  - fetal abnormalities
  - megaloblastic anaemia, neutropenia and thrombocytopenia.
- Sources of folic acid include green leafy vegetables, oranges and other fruit, legumes, nuts, liver and yeast.

**Treatment**

- All women who are anticipating pregnancy should be taking an additional 400 micrograms of folic acid per day before and throughout pregnancy.
- To treat deficiency, give infants 500 micrograms/kg once daily and children over 1 year of age 5 mg once daily.
- Treat for up to 4 months and exclude concomitant vitamin B₁₂ deficiency, which if untreated could result in neuropathy.
- For haemolytic anaemia, treat with 2.5–5 mg orally once a day for children aged 1 month to 12 years, and 10 mg once a day for those over 12 years of age.
- Neonates 50 micrograms once daily or 500 micrograms once weekly.
- Give preterm infants 100–200 micrograms orally per day.

**Zinc deficiency**

- Zinc is an essential trace element required for maintaining cells, bone growth and immune function (it scavenges for free radicals).
- Deficiency often occurs in children living in resource-limited settings, and arises from either insufficient intake of zinc-containing foods or insufficient absorption.
- Foods high in zinc are of animal origin, such as meats, fish and dairy products.
- Dietary fibre and phytates found in cereals and legumes bind zinc and reduce its absorption.
- Zinc deficiency is associated with stunting of growth, impaired immunity and increased risk and severity of diarrhoea and respiratory infections.
- Zinc deficiency is a feature of the rare disease acrodermatitis enteropathica, in which children present with peri-oral and peri-anal rashes.

Therapeutic zinc supplementation is now recommended as an adjunct to oral rehydration therapy for treatment of diarrhoea. Routinely giving 10 mg per day to children under 6 months of age and 20 mg per day to those over 6 months of age for 10–14 days can reduce diarrhoea duration and severity and the likelihood of subsequent infections for 2 to 3 months.
Zinc supplements of 2 mg/kg/day should be an essential component of the mineral mix used in the management of severe malnutrition.

5.10.B Severe malnutrition

**Introduction**
Severe acute malnutrition (SAM) is characterised by oedema or wasting, often with anaemia and infection. The main immediate causes of death are infections, septic shock, hypoglycaemia, electrolyte imbalance, dehydration, hypothermia, cardiac failure and severe anaemia. Every physiological and metabolic function is impaired, so the children affected are extremely fragile, similar to the premature neonate.

In 2009 the WHO and UNICEF defined SAM for children aged 6–60 months as follows:

1. using new weight for length/height charts (see procedures) a cut-off of below minus 3 standard deviations
2. and the mid upper arm circumference (MUAC) less than 115 mm.

Two clinical pictures are seen, with much overlap between them.

- Marasmus (wasting) affects all ages, but young infants are particularly at risk. It is usually due to insufficient intake of growth nutrients after breastfeeding stops. It can also be due to chronic illness. The baby is extremely thin, with loss of subcutaneous fat, resulting in skin wrinkles and folds. Weight for length or height is less than 70% of the median (see Section 9), or the MUAC is less than 115 mm.

- Kwashiorkor (oedematous malnutrition) usually occurs in children aged 2–4 years. It is an acute illness that suddenly appears over a few days. It is thought to be due to a deficit in the antioxidant nutrients. It presents with sodium retention and oedema of various degrees (from pedal to generalised), and skin lesions that are like severe sunburn in a fair-skinned person. It is fatty liver, with low circulating levels of all hepatic export proteins. The hair may be de-pigmented (this has no relation to the prognosis, and should be ignored clinically), and the hair pulls out very easily and painlessly (which is related to the prognosis).

In severe malnutrition, biochemical abnormalities include the following:

- low urea
- severe hypoproteinaemia
- hypokalaemia and hypophosphataemia
- hypomagnesaemia
- hypoglycaemia (see below)
- anaemia (frequently present).

**Principles of treatment**
Early identification and treatment is important, and children are often missed on the general admission wards or in outpatients because they are not measured. Screening using MUAC is helpful for identifying children if length measurement is not easily performed, or weight for height is notcharted.

Treatment is much more successful if standard treatment protocols are followed than if clinical judgements are made on individual patients. This is because the illness itself changes the clinical presentation, signs and symptoms of common complications.

**Inpatient versus outpatient community management of acute malnutrition (CMAM)**
Traditionally, care has been provided for all children in inpatient hospital units, ideally in a defined malnutrition ward. However, carers are less likely to be prepared to attend these until the child is unwell, so patients tend to present late. They also want the child to be discharged as soon as they are clinically stable, so often leave before nutritional deficits have been restored and the child has recovered. This predisposes to higher post-discharge mortality, which is rarely identified by the inpatient programme.

There has been a change to management of the malnourished child who is not unwell, through CMAM programmes. This is sometimes referred to as community-based therapeutic care (CTC). These programmes separate children into those with complications or severe oedema (complicated malnutrition), and those with uncomplicated malnutrition. Children with uncomplicated malnutrition have a reasonable appetite, and on formal testing are able to eat a portion of ready-to-use therapeutic food (RUTF). Children with fever, poor appetite, diarrhoea or dehydration, or who are not fully alert or have generalised oedema are identified and referred to inpatient care (a stabilisation centre), where initial management is delivered.

If a CMAM programme is operating in your area, children with complicated malnutrition will be sent to the hospital, and you may be able to direct those with uncomplicated malnutrition to the CMAM programme after hospital care.

**Useful website**

---

**5.10.8.1 Minimum standards**

- Scales (accurate to 5 gram), metre length board, MUAC tapes, care charts.
- ReSoMal.
- Vitamin and mineral mixtures.
- Antibiotics.
- IV 10% glucose.
- Anthelmintic drugs.
- F-75 and F-100 feeds.
- Barrier skin cream.
- Sources of heat (blankets, hat, warm room, clothes).

** BOX 5.10.8.1 Minimum standards**
admission for an illness, or if they present with complicated malnutrition, once they are stabilised and on phase II feeds (see later).

This subsection will deal with the care of children managed in an inpatient hospital unit.

Inpatient management
The inpatient treatment of severe malnutrition is divided into two phases, which are separated by a transition phase.

Phase I (initial treatment)
Specific objectives: return of normal homeostasis and treatment of complications.
- The immediate treatment of life-threatening complications: hypoglycaemia, hypothermia, heart failure, septic shock, infections and infestations, severe dehydration and very severe anaemia.
- The prevention of hypoglycaemia and hypothermia.
- Nutritional treatment based on a maintenance diet (total 100 kcal/kg/day), divided into frequent meals (eight meals per 24 hours).

Transition phase: the diet is gradually increased over 4–5 days.

Phase II (rehabilitation or catch-up growth)
Specific objectives: promotion of rapid weight gain (10–20 g/kg/day) and preparation for discharge.
- A nutritional treatment based on a high energy intake (160–200 kcal/kg/day) divided into six meals a day.
- Emotional and physical stimulation.

The treatment in phase II can be given as ready-to-use therapeutic food (RUTF) in the community, or through an Outpatient Therapeutic Programme (OTP), either administered through a hospital clinic, or preferably in community-based clinics. If RUTF or an equivalent (such as a high-energy biscuit) is not available, children continue on F-100 until nutritional cure is achieved. This is usually as a high-energy biscuit (see later).

Ongoing nutritional support
After discharge from this therapeutic programme, it is good practice to link the child to a supplementary feeding programme, which gives a food ration to the family for up to 4 months following discharge. This is a means of ensuring food security for the vulnerable child. Programmes with this safety-net provision often discharge children at 80% of median weight for height.

Admission criteria
- Weight for height less than 70% of the median.
- Oedema (exclude nephritic syndrome and other clinical conditions).
- MUAC of less than 110 mm if the child is over 65 cm in length.

Assessment of nutritional status and recovery
For practical procedures relating to nutrition measurement, see Section 9.

Discharge criteria
These depend upon the quality of the follow-ups. If adequate follow-up services and a Supplementary Food Programme (SFP) are available, the discharge criteria are as follows:
- weight for height of more than 80% of the median for 3 days (85% if there is no SFP)
- and no oedema for 10 days
- and no medical complications.

Medical and nutritional history and examination
The pro-forma history and examination sheet (see Appendix, Section 9) should be filled in by the admitting physician or an experienced nurse.

Key points in the history
- Recent intake of foods and fluids.
- Usual diet before current illness.
- Whether breastfeeding or not.
- Duration and frequency of diarrhoea and vomiting.
- Type of diarrhoea (watery/bloody).
- Appetite.
- Family circumstances.
- Previous attempts at treatment, local drugs and/or traditional medicines given.
- History of chronic cough or contact with TB.
- History of contact with measles.
- Potential HIV infection (including mother’s status and whether parents are alive).

Key points on examination
- Oedema.
- Dehydration (this is very difficult to diagnose, and impossible in the oedematous child).
- Shock (often gives the appearance of dehydration in a child with oedema).
- Severe palmar pallor.
- Eye signs for vitamin A deficiency (dry eyes, Bitot’s spots, corneal ulceration, keratomalacia) (see Section 5.10.A).
- Signs of local infection (ear, throat, skin, pneumonia).
- Signs of HIV (adenopathy, oral candida, chronic ear discharge) (see Section 6.2.D).
- Fever.
- Hypothermia (oral temperature < 35.5°C, axillary temperature < 35°C).
- Mouth ulcers, Candida or other oral problems.
- Skin changes of kwashiorkor (hypo- or hyperpigmentation, desquamation, ulceration, exudative lesions resembling burns, often with secondary infections such as Candida).

Children with vitamin A deficiency are likely to be photophobic and will keep their eyes tightly closed. Examine their eyes carefully to prevent corneal rupture.

Laboratory tests
Laboratory tests are not needed to guide or monitor treatment. Electrolytes and haemoglobin are difficult to interpret and can easily be misleading. If haemoglobin is measured this should be done on admission only, and a transfusion should be given at this time if essential. The patient should not be given a blood transfusion after the first 48 hours.
following admission. The haemoglobin level nearly always falls after admission due to haemodilution with expansion of the circulation during mobilisation of oedema and export of sodium from inside the cells in marasmus. At this time, with expansion of the circulation, there is such a grave danger of precipitating heart failure that a transfusion should rarely be given, even for very severe anaemia.

In endemic areas, a malaria smear or rapid test is useful if malaria treatment is not given as part of the routine management of all severely malnourished children.

In regions where HIV is prevalent, HIV testing (serology using two tests in children over 1 year of age, or serology and PCR for children under 1 year) is informative for ongoing care, initiating co-trimoxazole prophylaxis, and determining eligibility for antiretroviral (ARV) therapy. The mother of a seropositive child is invariably HIV infected, and mothers of seropositive children should be offered an HIV test. Services vary, but would normally include counselling prior to voluntary HIV testing. Where testing is routinely offered, uptake is usually high. CD4 counts are not usually required for the initial management of severe acute malnutrition, as this follows the standard protocols, but may be relevant when considering initiating ARV therapy (see Section 6.2.D).

**Details of treatment**

In Phase I (initial phase) the aim is to restore nutritional imbalances and metabolic function and treat complications. Phase II (catch-up growth) is a period of rapid weight gain. There is a ‘transition phase’ between these phases.

### TABLE 5.10.B.1 Phases of malnutrition treatment

<table>
<thead>
<tr>
<th>Phases of treatment</th>
<th>Phase 1 (1–7 days)</th>
<th>Transition phase (3–4 days)</th>
<th>Phase 2 (usually 14–21 days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treat dehydration</td>
<td></td>
<td>Correct nutrient deficiencies</td>
<td></td>
</tr>
<tr>
<td>Treat hypoglycaemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treat hypothermia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treat infection</td>
<td></td>
<td>Treat helminths</td>
<td></td>
</tr>
<tr>
<td>Do not give iron</td>
<td>Do not give iron</td>
<td>Correct iron deficiency</td>
<td></td>
</tr>
<tr>
<td>Correct electrolyte problems</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diet is maintenance intake</td>
<td>Diet is moderate intake</td>
<td>Diet is high intake</td>
<td></td>
</tr>
<tr>
<td>Stimulate the child</td>
<td>Stimulate the child</td>
<td>Stimulate the child</td>
<td>Provide physical activities</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Prepare for discharge</td>
</tr>
</tbody>
</table>

- **There are routine measures** that are systematically implemented for all malnourished children, and additional routine treatments that are often included.
- **Specific treatments**: these include emergency management of life-threatening complications and of specific diseases.
- **General points**: on admission, severely malnourished children should be separated from those with infections and kept in a warm room (25–30°C) without draughts. Washing should be minimal and when possible with warm water, and the child immediately dried. The mother should be encouraged to stay with her child.

### Intravenous infusion and blood transfusion

Intravenous infusions are to be avoided whenever possible in all severely malnourished children. The risk of precipitating heart failure is very high because of their atrophic heart muscle and high intracellular sodium and electrolyte imbalance.

- The only indication for IV infusion in severely malnourished children is unconsciousness due to circulatory collapse or shock. This is a condition which is difficult to diagnose.
- The only indication for blood transfusion is when anaemia is present on admission and is life-threatening.
- Cannulas should not have IV fluids running after the prescribed treatment has been given. If flushed for IV treatment, they should be removed when not required.

Nasogastric tube feeding is recommended in cases of:

- anorexia with an intake of less than 70 kcal/kg (70% of phase I feed prescribed)
- severe dehydration with inability to drink
- inability to drink and eat because of weakness or clouded consciousness
- painful or severe mouth lesions (herpes, cancrum oris)
- repeated, very frequent vomiting.

Try to not tube-feed for more than 3–4 days. Always explain the reason to the mother.

Try to breastfeed or feed by mouth every time, and top up by nasogastric tube.

### Dehydration with severe malnutrition

Dehydration from diarrhoea is common in severely wasted children (with marasmus) on admission. The treatment of dehydration is not the same as in the non-malnourished child (with the exception of cholera). This section does not apply to mild diarrhoea occurring during transition from one phase to another, which is a common event.

### Signs of dehydration in malnutrition

The normal signs used to assess dehydration are all unreliable in severe malnutrition.

Assume that all children with acute watery diarrhoea have some dehydration.

The interpretation of the signs relies on the history. The specific signs are as follows:
Reduced skin turgor and sunken eyes (that are long-standing symptoms) are features of malnutrition itself. It is not possible to adequately determine the degree of dehydration in the severely malnourished child.

The appearance of dehydration in children without watery diarrhoea or in those with oedema can be caused by a toxic shock with dilatation of the blood vessels. These patients should not be treated as if they have dehydration, but as cases of septic shock (see later).

Note that low blood volume can occur with oedema.

**Oral treatment of dehydration in malnutrition**

Standard WHO oral rehydration solutions (ORS) have too high a sodium content and too low a potassium content for children with severe malnutrition. 

ReSoMal (rehydration solution for malnutrition; see below) is a special solution for this situation.

**TABLE 5.10.B.2 Composition comparison of ReSoMal, standard WHO ORS and reduced-osmolarity WHO ORS**

<table>
<thead>
<tr>
<th>Composition</th>
<th>ReSoMal (mmol/litre)</th>
<th>Standard ORS (mmol/litre)</th>
<th>Reduced-osmolarity ORS (mmol/litre)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>125</td>
<td>111</td>
<td>75</td>
</tr>
<tr>
<td>Sodium</td>
<td>45</td>
<td>90</td>
<td>75</td>
</tr>
<tr>
<td>Potassium</td>
<td>40</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Chloride</td>
<td>70</td>
<td>80</td>
<td>65</td>
</tr>
<tr>
<td>Citrate</td>
<td>7</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Magnesium</td>
<td>3</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Zinc</td>
<td>0.3</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Copper</td>
<td>0.045</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Osmolarity (mOsm/litre)</td>
<td>300</td>
<td>311</td>
<td>245</td>
</tr>
</tbody>
</table>

**Children with watery diarrhoea in an adequate clinical state**

At admission, give one dose of ReSoMal orally or by nasogastric tube and start to feed the child with the phase I diet. Feed smaller amounts more frequently if they are vomiting. Further ReSoMal can be given after each stool or vomit.

- Give a 50-mL dose for children less than 2 years or less than 85 cm in length.
- Give 100 mL for children over 2 years or over 85 cm in length.

**Children with watery diarrhoea in a poor clinical state**

Start rehydration with ReSoMal immediately. Give 10 mL/kg/hour for the first 2 hours, and then 5 mL/kg/hour until rehydration is complete.

This rate is slower than for normally nourished and dehydrated children.

**Completed rehydration**

The rehydration is completed when the child is alert, no longer thirsty, and has passed urine. There should be less sunken eyes and fontanelle and improved skin turgor.

(Note that loss of sunken eyes in a severely wasted patient or the worsening of oedema can be a sign of over-hydration.)

The diet should now be given.

**Monitoring**

ReSoMal at 70 mL/kg weight per day is usually enough to restore hydration. However, be careful, as rehydration can quickly lead to fluid overloading, causing cardiac failure or sudden death. Malnourished children do not excrete excess sodium well. The clinical state of the child should be reassessed every 30 minutes during the first 2 hours, and then every hour. The best way to monitor the child is by regularly measuring their weight; this gives “fluid balance” directly and accurately, without having to measure any stool or vomit. The ReSoMal should be stopped immediately if:

- the body weight increases by 10% or more
- the respiratory rate or pulse rate increase
- the jugular vein becomes engorged
- oedema appears or the eyelids become puffy
- the liver enlarges by more than 2 cm (mark its position on the skin with marker pen at the onset of rehydration).

**Note:** It is common for malnourished children to pass many small unformed stools. This must not be confused with profuse watery stools, and does not require fluid replacement.

**Feeding and rehydration**

- Breastfeeding should continue during rehydration.
- Phase I diet should start immediately when the child is alert.
- If the child has had severe dehydration, feeding should start as soon as the child is alert and the severe dehydration has been treated (2–3 hours).

**Rehydration solutions**

If no commercial ReSoMal is available, a solution can be made. (Note that this is double the quantity of water normally used, i.e. 2 litres, so the solution is effectively half strength.)

To 2 litres of boiled filtered water add:

- 1 sachet of ORS (3.5 grams sodium chloride, 2.9 grams trisodium citrate dihydrate, 1.5 grams potassium chloride, 20 grams glucose)
- 50 grams of sugar
- 40 mL of combined mineral mix* (or commercial CMV if available).

* See below for the recipe for the electrolyte/mineral solution. If this cannot be made up, use 45 mL of potassium chloride solution (100 grams of KCl in 1 litre of water) instead.

**Formula for concentrated electrolyte/mineral solution**

This is used in the preparation of starter and catch-up feeding formulas and ReSoMal. Sachets containing pre-mixed electrolytes and minerals are produced by some manufacturers. If these are not available or affordable, prepare the solution (2500 mL) using the ingredients shown in Table 5.10.B.3.
TABLE 5.10.B.3 Electrolyte and mineral mixture

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Grams</th>
<th>Concentration/20mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium chloride (KCl)</td>
<td>224</td>
<td>24 mmol</td>
</tr>
<tr>
<td>Tripotassium citrate</td>
<td>81</td>
<td>2 mmol</td>
</tr>
<tr>
<td>Magnesium chloride</td>
<td>76</td>
<td>3 mmol</td>
</tr>
<tr>
<td>Zinc acetate</td>
<td>8.2</td>
<td>300 micromol</td>
</tr>
<tr>
<td>Copper sulphate</td>
<td>1.4</td>
<td>45 micromol</td>
</tr>
</tbody>
</table>

Water: make up to 2500mL.

If available, also add selenium (0.028 grams of sodium selenate) and iodine (0.012 grams of potassium iodide) per 2500mL.
- Dissolve the ingredients in cooled boiled water.
- Store the solution in sterilised bottles in the fridge to slow down deterioration. Discard if it turns cloudy.
- Make fresh solution each month.
- Add 20mL of the concentrated electrolyte/mineral solution to each 1000mL of milk feed.

If it is not possible to prepare this electrolyte/mineral solution, and pre-mixed sachets are not available, give potassium, magnesium and zinc separately. Make a 10% stock solution of potassium chloride (100 grams in 1 litre of water) and a 1.5% solution of zinc acetate (15 grams in 1 litre of water).

Emergency IV treatment of severe dehydration with shock in severe malnutrition

IV infusion should be administered only in the case of circulatory collapse severe enough to reduce consciousness. **Alert children should never be given an infusion.**

The main signs are as follows:
- cold hands and feet with increased capillary refill time > 3 seconds
- weak or absent radial pulse
- diminished consciousness.

Severe dehydration and septic shock are difficult to differentiate in children with severe malnutrition. They both present with signs of hypovolaemic shock. The following points help to differentiate them:
- Eyelid retraction associated with a history of diarrhoea is a sign of severe dehydration. The child with septic shock has eyelids that droop.
- If the child is unconscious (or asleep) without having the eyelids together (a sign of excess adrenaline), either dehydration or hypoglycaemia is present.
- Superficial veins are always constricted in severe dehydration, but may be dilated in septic shock.

Treatment protocol for life-threatening dehydration with shock in severe malnutrition

Immediate treatment should be given as follows:
1. Give 15 mL/kg IV over 1 hour. The recommended solution is Ringer-lactate or Hartmann’s solution, each with 5% glucose.
2. At the same time, insert a nasogastric tube and give ReSoMal at 10mL/kg per hour.
3. Monitor carefully for signs of over-hydration, reassessing respiratory rate and heart rate every 15 minutes.
   - **If after 1 hour the child is improving but still severely dehydrated**, continue nasogastric ReSoMal 10mL/kg/hour for up to 5 hours.
   - **If after 1 hour the child has not improved** (i.e. radial pulse is still weak), assume that they have septic shock and treat it accordingly (see below for treatment of septic shock). Since hypoalbuminaemia is likely also to be present, 4.5% albumin 5–15mL/kg IV over 1 hour may also be helpful in intractable shock but this approach requires urgent research.

Electrolyte problems in SAM

All severely malnourished children have deficiencies of potassium and magnesium that may take 2 weeks or more to correct. Oedema is partly a result of these deficiencies. **Do not treat oedema with a diuretic.** Excess body sodium exists even though the plasma sodium levels may be low. **Giving high sodium loads could kill the child.**

**Treatment**
- Give extra potassium (3–4 mmol/kg daily).
- Give extra magnesium (0.4–0.6 mmol/kg daily).
- The extra potassium and magnesium are present in commercial F-75 and F-100 feeds, but if making from ingredients locally should be added to the feeds during their preparation. See Table 5.10.B.3 for a recipe for a combined electrolyte/mineral solution. Add 20mL of this solution to 2.5 litres of feed to supply the extra potassium and magnesium required.
- Prepare food without adding salt.

Infections in SAM: treatment and prevention

All malnourished children must be assumed to have an infection. Because of the lack of an inflammatory response, clinical signs of infection may be entirely absent in a malnourished child with severe systemic infection. If untreated, this may cause mortality, morbidity and poor weight gain.

All children with severe acute malnutrition should routinely be given broad-spectrum antibiotics.

Protocol for treatment

**Specific infections**
Children with specific infections should receive the appropriate antibiotic according to local guidelines.

**No specific infection and no suspected septic shock**

The principle is to have a first-line treatment and a second-line treatment.
- **First-line treatment** is routinely given on admission to all severely malnourished children without complications such as septic shock, hypothermia, hypoglycaemia or a specific infection (skin, eyes). This is usually oral amoxicillin or co-trimoxazole.
- **Second-line treatment** is given after 48 hours to children who do not respond to the first-line treatment, and to all children with complications. This usually includes a parenteral antibiotic, although absorption of oral ciprofloxacin and chloramphenicol is excellent, so these can...
be used orally once the child is stabilised. Some units routinely give metronidazole 7.5 mg/kg orally 8-hourly for 7 days in addition to the above.

The choice of the antibiotics used in first-line and second-line treatment is based on local guidelines, which are ideally informed by local resistance patterns. Factors such as route of administration, availability and cost of the drugs are all relevant. It should be a broad-spectrum antimicrobial route of administration, availability and cost of the drugs informed by local resistance patterns. Factors such as line treatment is based on local guidelines, which are ideally informed by local resistance patterns.

Septic shock: recognition
Septic shock is a very common cause of deaths in these patients. The signs are as follows:
- clauding of consciousness
- rapid respiratory rate:
  - 50 breaths/minute for children aged 2–12 months
  - 40 breaths/minute for children aged 12 months to 5 years
- rapid pulse rate
- cold hands and feet with visible subcutaneous veins and prolonged capillary refill time > 3 seconds
- signs of dehydration but without a history of watery diarrhoea
- hypothermia or hypoglycaemia
- poor or absent bowel sounds
- an abdominal splash when the child is shaken.

It can be very difficult to distinguish between severe dehydration and septic shock.

Suspected septic shock: treatment
- A broad-spectrum IV antibiotic treatment (ceftriaxone) is started immediately.
- Warm the child to prevent or treat hypothermia (see hypothermia below).
- Feeding and fluid maintenance should be undertaken by nasogastric tube or orally.
- Close monitoring of the vital signs (pulse, respiration and conscious level) is essential.

Circulatory collapse
- Give high-flow oxygen through a face mask with reservoir.
- Give IV infusion as described in the case of circulatory collapse due to severe dehydration. However, as soon as the radial pulse becomes strong and the child regains consciousness, discontinue the infusion and start the diet orally or by nasogastric tube.

Hypothermia: prevention and treatment
Malnourished children have a low metabolic rate. The thermoneutral temperature is 28–32°C. At 24°C they can become hypothermic. Those with infection or extensive skin lesions are at particular risk. A hypothermic malnourished child should always be assumed to have septicaemia.

Signs
The signs of hypothermia are a core temperature (oral) < 35.5°C (with a low-reading thermometer). If the axillary temperature is < 35°C or does not register, assume hypothermia.

Routine prevention
- Cover all children with clothes and blankets. They should wear a warm hat (most heat is lost from the head).
- Ensure that the mother sleeps alongside her child. Do not leave a child alone in bed at night.
- Keep the ward doors and windows closed to avoid draughts.
- Avoid wet nappies, clothes or bedding.
- Do not wash very ill children. Others can be washed quickly, ideally with warm water, and dried immediately.
- Make sure that the child is fed, so that metabolic heat can be produced. Ensure that feeds occur during the night.
- Avoid medical examinations which leave the child feeling cold.

Emergency treatment of hypothermia
- Immediately place the child on the mother’s bare chest or abdomen (skin to skin) and cover both of them. Give the mother a hot drink to increase her skin blood flow.
- If no adult is available, clothe the child thoroughly (including the head) and put them near a lamp or radiant heater.
- Immediately treat for hypoglycaemia (see below) and then start normal feeds.
- Give second-line antibiotics.
- Monitor the temperature every 60 minutes until it is normal (> 36.5°C).

Hypoglycaemia: prevention and treatment
Severely malnourished children easily develop hypoglycaemia. This is associated with serious infection. If available, test blood glucose levels (< 2.5 mmol/litre), or if they are not measurable assume that hypoglycaemia is present.

Signs
The main signs of hypoglycaemia are as follows:
- Lethargy, limpness, loss of consciousness or convulsions
- Drowsiness/unconsciousness with the eyelids partly open, or retraction of the eyelids
- Low body temperature
- Convulsions.

Sweating and pallor do not usually occur in this situation.

Routine prevention
- Give frequent small feeds, day and night.
- Feeding should start while the child is being admitted.
- Treat any infections.

Emergency treatment
If hypoglycaemia is suspected:
- If the child can drink: give therapeutic milk or 50 mL of glucose 10%, or 50 mL of drinking water plus 10 grams of sugar (one teaspoon of sugar in 3.5 tablespoons of clean water). Follow this with the first feed as soon as possible. If achievable, divide the first feed into four and...
give half-hourly. If not, give whole feeds every 2 hours during the day and night for at least the first day.

- **If the child is unconscious or has convulsions:**
  - Give 5mL/kg body weight of glucose 10% IV or by the intra-osseous (IO) route, or if neither of these routes is possible give 5mL/kg of glucose 10% or sugar solution as described above by nasogastric tube.

Continue frequent feeding to avoid a recurrence.

- Give second-line antibiotics.
- If there are convulsions other causes must be excluded, including cerebral malaria, meningitis, encephalitis, thiamine deficiency and hypernatraemic/hyponatraemic dehydration (especially in hot dry climates).
- If blood glucose levels are available and are low, repeat the finger or heel prick after 60 minutes.

### Congestive heart failure

This is a common and dangerous complication that usually occurs several days after admission. The heart muscle is atrophic (effectively there is a cardiomyopathy). During early recovery from severe malnutrition, sodium can be mobilised from the tissues before the kidney recovers sufficiently to excrete the excess. All blood transfusions should be done as soon as possible (in the first 2 days after admission), and should be rarely indicated.

Heart failure is usually caused by inappropriate treatment, including the following:

- Misdiagnosis of dehydration with consequent inappropriate ‘rehydration’
- Very severe anaemia
- Overload due to blood transfusion
- A high-sodium diet, using conventional oral rehydration solution, or excess ReSoMal
- Inappropriate treatment that involves ‘re-feeding diarrhoea’ with rehydration solutions.

#### Signs

Excess weight gain is the most reliable sign, and daily weights should be taken for all malnourished children. Differentiate pneumonia and heart failure by weighing the child. If their weight has increased, particularly if by more than 5%, consider heart failure. If they have lost weight, consider pneumonia.

**First sign:** fast breathing:

- 50 breaths/minute for children aged 2–12 months
- 40 breaths/minute for children aged 12 months to 5 years.

**Later signs:**

- Lung crepitations
- Respiratory distress
- Rapid pulse rate
- Enlargement of the jugular vein
- Cold hands and feet
- Cyanosis or hypoxaemia diagnosed by pulse oximetry if available (SaO₂ < 94% in air at sea level)
- Liver enlarged by > 2 cm from baseline.

### Emergency treatment of congestive cardiac failure

- Give high-flow oxygen.
- Stop all oral intake and IV fluid.

- The treatment of heart failure takes precedence over feeding of the child.
- No fluid at all should be given until the cardiac function improves, even if it takes 24–48 hours.
- Give a diuretic IV, usually furosemide (1 mg/kg). This is the only situation in which diuretics should be used: diuretics should never be given to reduce oedema in malnourished children.

### Measles: prevention and treatment

Measles is especially dangerous in severe malnutrition.

#### Routine prevention

All children over 6 months of age who are admitted with malnutrition should be vaccinated against measles. This is often done weekly, but if measles is being transmitted locally, it should be done on admission. A second dose of vaccine in a previously immunised child is not harmful.

A second dose should be given once recovered or at the normal time, where the prior vaccination state is uncertain or the child was not vaccinated before admission.

#### Treatment of measles

If a case of measles is admitted:

- Isolate the individual and any suspected cases.
- Review the vaccination status of all patients in the ward, and ensure that all are immunised.
- Give two doses of vitamin A separated by 1 day.
- Treat for measles (see Section 6.2.E) as well as for malnutrition.

### Micronutrient deficiencies

All children with acute malnutrition will have these deficiencies. Commercial F-100 and RUTFs contains all of the required micronutrients in the correct amounts.

If not using these, give a daily multivitamin supplement, and add a mineral mix to the feeds. This should contain potassium, zinc, copper, magnesium and ideally selenium. Premixed sachets are available, or a solution can be made. It is important to avoid adding iron to milk-based feeds during the first 2 weeks, and until the child is gaining weight (RUTFs contain iron within the food, and this is safe to use for stable children and in CMAM programmes). After 2 weeks, iron is added to the F-100 feeds. In goitrous regions, potassium iodide should be added to the mineral mixture (12 mg/2500mL), or else the child should be given Lugol’s iodine, 5–10 drops per day.

### Vitamin A: prevention and treatment

#### Routine preventive treatment

Oral vitamin A is particularly important for the severely malnourished child, and one dose should be given routinely to each child admitted with malnutrition.

#### TABLE 5.10.B.4 Vitamin A dosage: preventive treatment

<table>
<thead>
<tr>
<th>Weight</th>
<th>Dose at admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 6kg</td>
<td>50 000 IU once</td>
</tr>
<tr>
<td>6–10kg</td>
<td>100 000 IU once</td>
</tr>
<tr>
<td>&gt; 10kg</td>
<td>200 000 IU once</td>
</tr>
</tbody>
</table>
Treatment of xerophthalmia
If a child shows signs of vitamin A deficiency (xerophthalmia) or has measles, three doses of vitamin A treatment should be given.

<table>
<thead>
<tr>
<th>Weight</th>
<th>Dose on day 1</th>
<th>Dose on day 2</th>
<th>Dose on day 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 6 kg</td>
<td>50,000 IU</td>
<td>50,000 IU</td>
<td>50,000 IU</td>
</tr>
<tr>
<td>6–10 kg</td>
<td>100,000 IU</td>
<td>100,000 IU</td>
<td>100,000 IU</td>
</tr>
<tr>
<td>&gt; 10 kg</td>
<td>200,000 IU</td>
<td>200,000 IU</td>
<td>200,000 IU</td>
</tr>
</tbody>
</table>

If the eyes show signs of inflammation or ulceration, give the following additional care to the affected eye(s) to prevent corneal rupture and extrusion of the lens:
- Instil chloramphenicol or tetracycline eye drops, 2- to 3-hourly as required for 7–10 days.
- Instil atropine eye drops, one drop three times daily for 3–5 days.
- Cover with sterile saline-soaked eye pads.
- Bandage the eye(s).

Note that children with vitamin A deficiency are likely to be photophobic and have their eyes closed. It is important to examine their eyes very gently to prevent corneal rupture.

Treatment of anaemia
The majority of malnourished children have anaemia. This is due to the many deficiencies they have (iron, folic acid, riboflavine, pyridoxine, ascorbic acid, vitamin E, copper) and their inability to metabolise iron. Iron should never be given during Phase I or during the transition phase.

Routine treatment
- Folic acid
  Give 5 mg of folic acid on the day of admission, then 1 mg/day thereafter (in F-100 already).

- Iron
  Iron should never be given during Phase I or during the transition phase. In malnourished patients, iron is not properly metabolised and is therefore dangerous. The free iron enhances the production of free radicals that can damage cell walls. Excess free iron encourages systemic infection.
  Oral iron supplementation should start 14 days after admission. This is best added to the F-100 milk diet at a dose of one crushed tablet of ferrous sulphate (200 mg) to 2 litres of therapeutic milk. Alternatively, it can be given as ferrous sulphate 3 mg/kg/day orally, which should be continued until anaemia has resolved clinically, or ideally on blood test. It is present in adequate amount in RUTF.

Emergency treatment of very severe anaemia
Blood transfusion in malnourished children is potentially dangerous because it can precipitate heart failure. There are only two indications for considering blood transfusion, namely:
- the child with a haemoglobin concentration of < 4 grams/100 mL, especially if in shock
- the child with signs of heart failure due to anaemia (at immediate risk of death).

Give 10 mL/kg body weight of packed cells (or whole blood) slowly by partial exchange transfusion. Ideally, and if this can be achieved, use a carefully and continuously observed cannula in an artery or central vein. It is also possible in a vein in the antecubital fossa. First 2.5 mL/kg of anaemic blood is removed and then when 5 mL/kg of appropriately screened and cross-matched blood has been transfused, 2.5 mL/kg is again taken and the cycle is repeated. The child is closely monitored for signs of congestive heart failure.

If partial exchange is not possible and heart failure is present, give 10 mL/kg, ideally as packed cells, otherwise as whole blood. Transfuse over 4 hours and give IV furosemide 1 mg/kg at the start of the transfusion. Monitor carefully for worsening heart failure.

Intestinal parasites
Routine treatment
Routine deworming treatment is given to all children over 1 year of age, but only in Phase II or the transition phase.

- For children over 1 year of age, give mebendazole 100 mg (1 tablet) twice daily for 3 days. Some countries use albendazole 200 mg (for children aged 12–24 months) or 400 mg (for those over 24 months of age) once.

- For children under 1 year of age, give albendazole 100 mg (1 tablet) twice daily for 3 days. Some countries use albendazole 200 mg (for children aged 12–24 months) or 400 mg (for those over 24 months of age) once.

Dermatosis of kwashiorkor
Shedding of the skin in scales or sheets, desquamation, exfoliation, cracking of the skin surface, and ulceration of the genital or perianal areas are all common.
There can be widespread weeping skin lesions that resemble burns.
Zinc deficiency is usual in this situation, and oral zinc supplements improve the skin (give 2 mg/kg/day of elemental zinc).

- Treatment
  - Leave the exposed area open to dry during the day.
  - Apply barrier cream (zinc and castor oil ointment) or petroleum jelly or tulle gras to the raw areas, and gentian violet or nystatin cream to the skin sores twice a day.
  - These children should be on broad-spectrum antibiotics.
  - Do not use plastic pants or disposable nappies for these children.

Continuing diarrhoea
See also Section 5.12.B.
Diarrhoea should subside during the first week of treatment. In the rehabilitation phase, loose or poorly formed stools are normal and do not need treatment provided that weight is increasing.

Treatment
Giardiasis
Giardiasis and mucosal damage are common causes of continuing diarrhoea. Where possible, examine the stools by microscopy.
If cysts or trophozoites of Giardia lamblia are found, give metronidazole (7.5 mg/kg 8-hourly for 7 days). If not
Lactose intolerance
Diarrhoea is only rarely due to lactose intolerance. Only treat for lactose intolerance if the continuing diarrhoea is preventing general improvement. Starter F-75 is a low-lactose feed. In exceptional cases:
- Substitute milk feeds with yoghurt or a lactose-free infant formula.
- Reintroduce milk feeds gradually in the rehabilitation phase.

Osmotic diarrhoea
This may be suspected if the diarrhoea worsens substantially with hyperosmolar F-75, and ceases when the sugar content and osmolarity are reduced. In these cases:
- Use a lower osmolar cereal-based starter F-75 (for the recipe, see Table 5.10.B.7) or, if available, use a commercially prepared isotonic starter F-75.
- Introduce catch-up F-100 gradually.

Malaria: treatment and prevention
In endemic areas, all malnourished children should have a rapid malaria smear or rapid test on admission. Where this is not possible, all malnourished children should receive antimalarial treatment according to local guidelines for the area. The parasitaemia is usually much lower than in normal children. In initially smear-negative children, there can be a recrudescence during nutritional replacement treatment, so consider malaria in children who develop fever.

Children and mothers should sleep under impregnated nets in the wards.

Tuberculosis
In patients treated for malnutrition, tuberculosis (TB) can be a cause of failure to gain weight. In malnourished children, the diagnosis of tuberculosis is particularly difficult and misdiagnosis is common.

How to diagnose pulmonary TB
The signs of TB in malnourished children are often not specific (e.g. anorexia, failure to thrive). Asymmetric chest signs or asymmetric lymph nodes are usually TB. Pneumonia in malnourished children affects both lungs, and HIV gives symmetrical lymphadenopathy.

Consider TB as a possible diagnosis in children who fail to gain weight during admission.

Sputum is rarely available. The Mantoux test can be negative in malnutrition. Undertake a chest X-ray if possible. A family history is often helpful. BCG offers protection against TB, but does not protect completely against infection.

Treatment (see Section 6.1.N)
Children with TB should not be isolated, for the following reasons:
- Young children are not a source of transmission (as it is rarely a cavitating disease).
- Treatment quickly eliminates the risk of transmission.
- An isolated child is stigmatised and neglected in resource-limited settings.

Usually paediatric TB is acquired from a sputum-positive adult, so the TB infected carer is a much higher infection risk to the ward. Take note of the carer on the ward with cough, as they should have a chest X-ray.

Malnutrition and AIDS
Basically, the initial stabilisation phase and nutritional treatment of HIV-infected patients is the same as for any other severely malnourished patient (see Section 6.2.D). They follow the same dietary and initial medical treatments. Many HIV-positive patients will respond well to the nutritional treatment and gain weight.

However, in units where HIV is prevalent, and particularly where there are programmes that offer additional nutritional support, co-trimoxazole, ARV treatment, PMTCT, and counselling on future pregnancies, there are excellent reasons why a carer would choose to have their child identified during admission as infected with HIV. Moreover, where this is routinely offered, such a policy is not found to be stigmatising.

The presentation of HIV-infected children is similar to that of the uninfected, so cannot be easily identified clinically, although there are some conditions that are more common. HIV-infected children are less likely to present with kwashiorcor than with marasmus. They are more likely to have oral candida, discharging ears, lymphadenopathy, chronic cough, persistent diarrhoea and dermatosis. They may have a family member with HIV, or be orphaned. They may present in infancy, while still breastfeeding, which is an uncommon time for presentation with severe acute malnutrition otherwise.

HIV testing should follow counselling, and be voluntary. It is usually done using two rapid ELISA serological tests. In infants under 1 year of age, serology reflects maternal status rather than infection in the infant. To diagnose infection in infants, a PCR test is required. All children identified as infected with HIV (or where PCR is not available as having indeterminate status) should be commenced on prophylactic co-trimoxazole. This has been shown to reduce long-term mortality.

In an HIV-infected infant, or one possibly infected with HIV and presenting with malnutrition, it is not sensible to stop breastfeeding during admission, as this will deprive the infant of an important source of nutrition. For children who are PCR negative, but exposed to HIV, the decision is less clear, although it will depend on the mother’s likely viral load (check whether she is on ARV treatment), the food security of the family, the mother’s ability to provide an alternate breast milk substitute, and her choice. There will usually be guidelines depending on local factors.

If the HIV-infected child is not responding well to malnutrition treatment, this may be because of unidentified infection. Non-typhoidal salmonella (NTS) is more common, as are organisms resistant to commonly used antibiotics. TB is a recognised co-infection, although it may be difficult to identify. Some children do not start gaining weight until ARV drugs are started.

It is not known when it is best to initiate ARV therapy in severe acute malnutrition, although it is generally accepted that children should be on phase II feeds. Some children do not meet the criteria for treatment clinically if they respond well to nutritional support with rapid weight gain. CD4 testing is helpful for determining who would benefit, as not...
all HIV-infected children have severe immunodeficiency, because HIV can be related to malnutrition through food insecurity as well as illness. However, long-term follow-up of infected malnourished children has identified them as being at high risk of mortality, suggesting that earlier ARV treatment might be of greater help in reducing this.

On discharge it is important to ensure that the child is linked into HIV and nutrition support programmes which the family can access, that carers are aware of the ongoing needs of the child, and that the wider family is offered HIV testing.

### Dietary treatment of severe malnutrition

#### Dietary treatment in Phase I

**Objectives**

The aim of this phase is progressive restoration of the electrolyte, metabolic and physiological balance by the frequent feeding of special formula milk.

**Principles**

Severely malnourished children are usually anorexic, and have thin bowel walls, damaged metabolism, and too much sodium in their bodies. Initially they require a low-salt and low-protein diet and are unable to tolerate large amounts of food because their capacity is reduced. Therefore initially a diet high in carbohydrate with low levels of sodium and iron and very modest protein content is given. This diet leads to restoration of metabolic and physiological function, but is insufficient for weight gain.

- Feeding should start quickly after admission.
- It should be divided into many small meals to stay within the absorptive and metabolic capacity of the child and to prevent hypoglycaemia and hypothermia.
- The child should be encouraged to eat, but not be forced to do so. Feeding a malnourished child requires time and patience. Use a cup, bowl, spoon or syringe to feed very weak children. If the child takes less than 70% of the prescribed diet, they should be fed by a nasogastric tube.
- Always continue breastfeeding, and encourage the mother to breastfeed. After the breastfeed give the scheduled amounts of starter formula first (see below).

The following guidelines are also useful:

- Give frequent small feeds of low osmolarity and low lactose content.
- Night feeds are essential.
- Give oral or nasogastric feeds (never parenteral preparations).
- Give 100 kcal/kg/day.
- Protein: give 1–1.5 grams/kg/day.
- Liquid: give 130 mL/kg/day to all children, whether or not oedema is present.

**TABLE 5.10.B.7 Volumes of F-75 per feed**

<table>
<thead>
<tr>
<th>Child’s weight (kg)</th>
<th>2-hourly (mL/meal)</th>
<th>3-hourly (mL/meal)</th>
<th>4-hourly (mL/meal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.0</td>
<td>20</td>
<td>30</td>
<td>45</td>
</tr>
<tr>
<td>2.2</td>
<td>25</td>
<td>35</td>
<td>50</td>
</tr>
<tr>
<td>2.4</td>
<td>25</td>
<td>40</td>
<td>55</td>
</tr>
<tr>
<td>2.6</td>
<td>30</td>
<td>45</td>
<td>55</td>
</tr>
<tr>
<td>2.8</td>
<td>30</td>
<td>45</td>
<td>60</td>
</tr>
<tr>
<td>3.0</td>
<td>35</td>
<td>50</td>
<td>65</td>
</tr>
<tr>
<td>3.2</td>
<td>35</td>
<td>55</td>
<td>70</td>
</tr>
<tr>
<td>3.4</td>
<td>35</td>
<td>55</td>
<td>75</td>
</tr>
<tr>
<td>3.6</td>
<td>40</td>
<td>60</td>
<td>80</td>
</tr>
<tr>
<td>3.8</td>
<td>40</td>
<td>60</td>
<td>85</td>
</tr>
<tr>
<td>4.0</td>
<td>45</td>
<td>65</td>
<td>90</td>
</tr>
<tr>
<td>4.2</td>
<td>45</td>
<td>70</td>
<td>90</td>
</tr>
<tr>
<td>4.4</td>
<td>50</td>
<td>70</td>
<td>95</td>
</tr>
<tr>
<td>4.6</td>
<td>50</td>
<td>75</td>
<td>100</td>
</tr>
<tr>
<td>4.8</td>
<td>55</td>
<td>80</td>
<td>105</td>
</tr>
<tr>
<td>5.0</td>
<td>55</td>
<td>80</td>
<td>110</td>
</tr>
<tr>
<td>5.2</td>
<td>55</td>
<td>85</td>
<td>115</td>
</tr>
<tr>
<td>5.4</td>
<td>60</td>
<td>90</td>
<td>120</td>
</tr>
<tr>
<td>5.6</td>
<td>60</td>
<td>90</td>
<td>125</td>
</tr>
<tr>
<td>5.8</td>
<td>65</td>
<td>95</td>
<td>130</td>
</tr>
<tr>
<td>6.0</td>
<td>65</td>
<td>100</td>
<td>130</td>
</tr>
<tr>
<td>6.2</td>
<td>70</td>
<td>100</td>
<td>135</td>
</tr>
<tr>
<td>6.4</td>
<td>70</td>
<td>105</td>
<td>140</td>
</tr>
<tr>
<td>6.6</td>
<td>75</td>
<td>110</td>
<td>145</td>
</tr>
<tr>
<td>6.8</td>
<td>75</td>
<td>110</td>
<td>150</td>
</tr>
<tr>
<td>7.0</td>
<td>75</td>
<td>115</td>
<td>155</td>
</tr>
<tr>
<td>7.2</td>
<td>80</td>
<td>120</td>
<td>160</td>
</tr>
<tr>
<td>7.4</td>
<td>80</td>
<td>120</td>
<td>160</td>
</tr>
<tr>
<td>7.6</td>
<td>85</td>
<td>125</td>
<td>165</td>
</tr>
<tr>
<td>7.8</td>
<td>85</td>
<td>130</td>
<td>170</td>
</tr>
<tr>
<td>8.0</td>
<td>90</td>
<td>130</td>
<td>175</td>
</tr>
<tr>
<td>8.2</td>
<td>90</td>
<td>135</td>
<td>180</td>
</tr>
<tr>
<td>8.4</td>
<td>90</td>
<td>140</td>
<td>185</td>
</tr>
<tr>
<td>8.6</td>
<td>95</td>
<td>140</td>
<td>190</td>
</tr>
<tr>
<td>8.8</td>
<td>95</td>
<td>145</td>
<td>195</td>
</tr>
<tr>
<td>9.0</td>
<td>100</td>
<td>145</td>
<td>200</td>
</tr>
<tr>
<td>9.2</td>
<td>100</td>
<td>150</td>
<td>200</td>
</tr>
<tr>
<td>9.4</td>
<td>105</td>
<td>155</td>
<td>205</td>
</tr>
<tr>
<td>9.6</td>
<td>105</td>
<td>155</td>
<td>210</td>
</tr>
<tr>
<td>9.8</td>
<td>110</td>
<td>160</td>
<td>215</td>
</tr>
<tr>
<td>10.0</td>
<td>110</td>
<td>160</td>
<td>220</td>
</tr>
</tbody>
</table>

Mix the milk, sugar, oil and electrolyte/mineral solution to a paste, and then slowly add the warm boiled water. Make up to 1000 mL. If available, use an electric blender or hand whisk.

**What food to give**

The special milk for phase I is called F-75. If it is not available, F-100 should be diluted to the same calorie strength as F-75 and given in its place. Alternatively, it can be made from ingredients using the recipe in Table 5.10.B.7 above.
TABLE 5.10.B.8  Homemade recipes for re-feeding formulas F-75 and F-100

<table>
<thead>
<tr>
<th></th>
<th>F-75&lt;sup&gt;a&lt;/sup&gt; (starter)</th>
<th>F-75&lt;sup&gt;c&lt;/sup&gt; (starter: cereal-based)</th>
<th>F-100&lt;sup&gt;d&lt;/sup&gt; (catch-up)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dried skimmed milk (grams)</td>
<td>25</td>
<td>25</td>
<td>80</td>
</tr>
<tr>
<td>Sugar (grams)</td>
<td>100</td>
<td>70</td>
<td>50</td>
</tr>
<tr>
<td>Cereal flour (grams)</td>
<td>–</td>
<td>35</td>
<td>–</td>
</tr>
<tr>
<td>Vegetable oil (grams)</td>
<td>27</td>
<td>27</td>
<td>60</td>
</tr>
<tr>
<td>Electrolyte/mineral solution (mL)</td>
<td>20</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Water: make up to (mL)</td>
<td>1000</td>
<td>1000</td>
<td>1000</td>
</tr>
<tr>
<td><strong>Contents per 100 mL</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy (kcal)</td>
<td>75</td>
<td>75</td>
<td>100</td>
</tr>
<tr>
<td>Protein (grams)</td>
<td>0.9</td>
<td>1.1</td>
<td>2.9</td>
</tr>
<tr>
<td>Lactose (grams)</td>
<td>1.3</td>
<td>1.3</td>
<td>4.2</td>
</tr>
<tr>
<td>Potassium (mmol)</td>
<td>4.0</td>
<td>4.2</td>
<td>6.3</td>
</tr>
<tr>
<td>Sodium (mmol)</td>
<td>0.6</td>
<td>0.6</td>
<td>1.9</td>
</tr>
<tr>
<td>Magnesium (mmol)</td>
<td>0.43</td>
<td>0.46</td>
<td>0.73</td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>2.0</td>
<td>2.0</td>
<td>2.3</td>
</tr>
<tr>
<td>Copper (mg)</td>
<td>0.25</td>
<td>0.25</td>
<td>0.25</td>
</tr>
<tr>
<td>% energy from protein</td>
<td>5</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>% energy from fat</td>
<td>32</td>
<td>32</td>
<td>53</td>
</tr>
<tr>
<td>Osmolality (mOsm/litre)</td>
<td>413</td>
<td>334</td>
<td>419</td>
</tr>
</tbody>
</table>

<sup>a</sup> A comparable starter formula can be made from 35 grams of whole dried milk, 100 grams of sugar, 20 grams of oil, 20 mL of electrolyte/mineral solution, and water to make 1000 mL. If using fresh cow’s milk, take 300 mL of milk, 100 grams of sugar, 20 mL of oil, 20 mL of electrolyte/mineral solution, and water to make 1000 mL.

<sup>b</sup> Isotonic versions of F-75 (280 mOsm/litre) are available commercially, in which maltodextrins replace some of the sugar, and in which all of the extra nutrients (potassium, magnesium and micronutrients) are incorporated. These are of lower osmolarity and therefore less likely to cause osmotic diarrhoea.

<sup>c</sup> Cook for 4 minutes. This may be helpful for children with dysentery or persistent diarrhoea.

<sup>d</sup> A comparable catch-up formula can be made from 110 grams of whole dried milk, 50 grams of sugar, 30 grams of oil, 20 mL of electrolyte/mineral solution, and water to make 1000 mL. If using fresh cow’s milk, take 880 mL of milk, 75 grams of sugar, 20 mL of oil, 20 mL of electrolyte/mineral solution, and water to make 1000 mL.

F-75 contains:
- 75 kcal/100 mL
- 0.9 grams of protein/100 mL (around 5% of kcal provided by protein)
- 2 grams of fat/100 mL (around 32% of kcal provided by fat)
- 13 grams of carbohydrate/100 mL (around 62% of kcal provided by carbohydrates).

**Dietary treatment in the transition phase (for 48 hours)**

**What food to give**

In the transition phase, full-strength F-100 is given in the same volume that was calculated for F-75 in phase I. There is no other change made in the transition phase. F-100 contains:
- 100 kcal/100 mL
- around 2.6 grams of protein/100 mL (10% of kcal provided by protein)
- around 5.6 grams of fat/100 mL (50% of kcal provided by fat)
- around 9.8 grams of carbohydrate (40% of kcal provided by carbohydrate).

There are two forms of F-100.

**Commercial F-100**

This therapeutic milk is prepared in a sachet. All that the nurse has to do is open the packet and dilute the contents in 2 litres of potable (boiled) water. The commercial F-100 has a lower osmolarity to reduce ‘re-feeding’ diarrhoea in the severely malnourished children.

**Example**

A child of 6 kg should receive a diet of 100 kcal/kg/day. The child will be given eight meals of F-75.

Number of kcal/day: 100 kcal × 6 kg = 600 kcal.
Quantity of F-75 per day: 800 mL (798 exactly). Quantity per meal: 800/8 = 100 mL.

Do not exceed 100 kcal/kg/day in this initial phase. Diarrhoea should gradually decrease and oedematous children should lose weight as the oedema disappears. If diarrhoea continues, see above.
Home-made F-100
This can be made from ingredients using the recipe shown in Table 5.10.B.8 above.

Dietary treatment in phase II
Objectives
The aim is catch-up growth of the child with rapid weight gain (10–20 g/kg/day). Usually the appetite has returned.

Principles
The child has re-established their physiological balance and should get enough food to gain weight as quickly as possible. They are given a high-energy diet with normal protein content.

- The intake is increased in quantity (to about 200 kcal/kg/day).
- Reduce meal frequency from eight to six meals per day.
- There should be no limit on the quantity of food given. The child is allowed to eat as much as they want, but must never be forced to eat.
- Breastfeeding continues. Breast milk must always be offered before the high-energy food is given.
- Aim for weight gain of more than 10 grams/kg/day.
- Remain alert for heart failure.

What food to give
The basic diet is composed of F-100 meals. However, when the child is gaining weight quickly, other foods can be introduced – for example:

- enriched porridges (1 mL contains 1 kcal/gram) as one to two meals a day
- enriched biscuits (useful for overnight feeding if phase II is conducted in a day-care centre) or RUTF (see below)
- local meal: composed of the usual food eaten in the area; this should be enriched in the pot with the addition of oil and sometimes DSM.

Quantity of food to give
Dispense and offer 200 kcal/kg of F-100 per kg of body weight per day.

Example of calculation
A child who weighs 9 kg should receive 200 kcal x 9 = 1800 kcal per day. The child will receive six meals per day, and each meal should provide 1800 kcal/6 = 300 kcal.

The diet is composed of six meals of F-100. The enriched porridge or family meal is given in addition if the child wishes to take it.

- F-100 (1 mL of F-100 = 1 kcal): the child should receive 300 mL of F-100 per meal.

Older children and adolescents, when they are gaining weight rapidly, often do not want the milk and demand ‘solid food’. This usually slows the rate of recovery. The solid food should always be enriched.

When developing local recipes the weight gain should be compared with that of children taking F-100 alone. If the weight gain is similar, the recipe for the porridge is adequate.

Ready-to-use therapeutic foods (RUTFs)
RUTFs have been developed to provide the same nutritional content as F-100, but in a peanut-butter-type paste that is not susceptible to pathogens growing in it, due to its low water content. These are usually based on a mix of groundnuts, vegetable oil, dry skinned milk, sugar and micronutrient mix. If this is available, children can be introduced to this when on phase II feeds, and if they have a good appetite, could be followed up after 1–2 weeks in an outpatient- or community-based malnutrition programme. This is referred to as an Outpatient Therapeutic Programme (OTP), CMAM or CTC (see Further Reading at the end of this subsection).

Individual child monitoring
Phase I
A daily medical and nutritional round of all the children in phase I should be done. The children should be carefully monitored every day for:

- oedema
- weight
- appetite: how the child is eating and the quantity eaten
- clinical state: consciousness, diarrhoea, vomiting, skin, etc.
- behaviour: apathetic, alert, crying, etc.
- temperature
- liver size, heart rate and heart sounds.

This information should be recorded every day on an individual chart.

When to pass to the transition phase
Children usually remain in phase I for 1–7 days. The child can pass to the transition phase when:

- they regain appetite
- they are lively and interested
- serious medical complications are under control
- oedema is decreasing (although it may be still present).

If after 5–7 days the child is not ready for the transition phase, they should be completely re-examined and investigated.

After 2 days in the transition phase without experiencing any problem, the child is ready to move to phase II. Oedema should be significantly improved, and the child must be stable, before progressing to phase II.

Phase II
The monitoring in phase II includes the following:

- a daily round by the nurse, who checks the general state of the child, including whether there is oedema, nausea or vomiting, and how the child is eating
- a physician round undertaken weekly if the child is stable
- measurement of the child’s weight twice a week if they are well
- measurement of their height monthly or in each OTP clinic review.

This information should be recorded on the individual chart.

If a child develops a complication in phase II, such as re-feeding diarrhoea or vomiting that requires passage of a nasogastric tube, rehydration solutions, transfusion, etc., they should be returned to phase I and subsequently the transition phase again. The above treatments must never be given to children while in phase II and taking very large amounts of F-100 diet.

When the child can be discharged
Children remain in phase II until they meet the criteria for recovery. The average total length of stay is around 4 weeks.
in traditional inpatient care, and longer if very severe complicated malnutrition, HIV, TB, or underlying disease or disability is present.

Figure 5.10.B.1 shows an example of a typical growth recovery chart.

When the child has reached their target weight and is in a good clinical state, they will be either referred to a supplementary feeding programme or sent directly home with arrangements made for follow-up. Children with long-term illness should be transferred to an appropriate community service.

Failure to gain weight
If the child fails to gain weight they should be investigated. Weight gain is defined as poor if it is less than 5 grams/kg/day, moderate if it is 5–10 grams/kg/day, and good if it exceeds 10 grams/kg/day. The following are the most common reasons for failure to gain weight:

- Food prescription or food preparation (kitchen) is incorrect and the child has not received the right quantity of the right food.
- The child does not eat the amount of food prescribed (e.g. because they dislike the food, or the food is being eaten by other people).
- Suspect hidden acute infections (e.g. urinary tract infection, acute respiratory infection, otitis media, mouth candidiasis, giardiasis).
- There are chronic hidden infections (tuberculosis, HIV).
- Re-examine, do stool and urine microscopy, and take a chest X-ray.

Look for poor feeding techniques, and check that night feeds are occurring.

Emotional and physical stimulation
The severely malnourished child is nearly always psychosocially deprived. The illness itself makes the child unresponsive, and so they do not cry or complain. Because mothers use a cry as the signal to give attention, these children do not receive the attention they need to stimulate them. The neglect is not willful on the part of the mother, but rather it is a failure of the two-way communication between the mother and her child.

Because they do not cry or complain, these children are often also neglected by nurses and staff. This greatly compounds the problems associated with being in a strange environment. It is essential to stimulate these children, particularly the unresponsive ones. The ward should be made as much like home as possible and children should sleep alongside their mothers.

- In phase I it is essential that the mother (or other carer) is present, feeds the child, comforts them, holds them, plays with them, and talks and sings to them.
- In phase II it is important to stimulate the child to move, and to play with other children. A play area should always be present. Staff should be identified who have a responsibility for providing (local) toys and encouraging play.

The daily organisation of the activities
To organise the treatment of malnourished children, a schedule of activities (e.g. care, distribution of meals) must be established. An example is given below.

<table>
<thead>
<tr>
<th>Time (24-hour clock)</th>
<th>Children in phase I and transition phase</th>
<th>Children in phase II (day care)</th>
</tr>
</thead>
<tbody>
<tr>
<td>02.00</td>
<td>Milk distribution</td>
<td></td>
</tr>
<tr>
<td>05.00</td>
<td>Milk distribution</td>
<td></td>
</tr>
<tr>
<td>07.00</td>
<td>Team changeover (day shift)</td>
<td></td>
</tr>
<tr>
<td>07.30</td>
<td>Temperatures</td>
<td>Arrival of children</td>
</tr>
<tr>
<td>08.00</td>
<td>Milk distribution and drugs</td>
<td>Milk distribution and drugs</td>
</tr>
<tr>
<td>09.00</td>
<td>Weight, oedema assessment</td>
<td>Weight, oedema assessment</td>
</tr>
<tr>
<td>09.30</td>
<td>Mother’s meal</td>
<td>Medical round</td>
</tr>
<tr>
<td>10.00</td>
<td>Medical round</td>
<td>Milk distribution</td>
</tr>
<tr>
<td>11.00</td>
<td>Milk distribution</td>
<td>Mother’s meal</td>
</tr>
<tr>
<td>12.00</td>
<td>Milk distribution and drugs</td>
<td></td>
</tr>
<tr>
<td>13.00</td>
<td>Dressings</td>
<td>Dressings</td>
</tr>
<tr>
<td>14.00</td>
<td>Milk distribution and drugs</td>
<td></td>
</tr>
<tr>
<td>15.00</td>
<td>Porridge distribution</td>
<td></td>
</tr>
<tr>
<td>16.00</td>
<td>Mother’s meal</td>
<td>Mother’s meal</td>
</tr>
<tr>
<td>17.00</td>
<td>Milk distribution</td>
<td>Milk distribution</td>
</tr>
<tr>
<td>18.00</td>
<td>Medical round</td>
<td>Departure home with porridge and enriched biscuits for the night</td>
</tr>
<tr>
<td>19.00</td>
<td>Team changeover (night shift)</td>
<td></td>
</tr>
<tr>
<td>20.00</td>
<td>Milk distribution and drugs</td>
<td></td>
</tr>
<tr>
<td>21.00</td>
<td>Close windows, wrap child</td>
<td></td>
</tr>
<tr>
<td>23.00</td>
<td>Milk distribution</td>
<td></td>
</tr>
</tbody>
</table>
Inappropriate practices
- Too much sodium, energy and protein given during phase I of treatment.
- No distinction made between phases I and II.
- Failure to monitor food intake.
- Lack of feeding at night.
- Lack of blankets and hats.
- No daily schedule organised.
- Diuretic given to treat oedema.
- Anaemia treated from time of admission with iron supplements.
- Intravenous fluids given for indications other than circulatory collapse.
- Use of high-sodium diet and standard oral rehydration solution.
- Routine antibiotics not given.
- No vitamin A given.
- No measles vaccine given.

Problems with the management of severe malnutrition
A high level of care is needed. The treatment of a severely malnourished child requires intensive protocol-based care, like that for a premature neonate, with close monitoring, some complex medical care (severe or chronic infections), a diet well enriched in nutrients (F-100, etc.), and an emotionally stimulating, rich and physically warm environment.

The resources are almost always limited. The limited financial resources lead to difficulty in obtaining therapeutic milks and other fortified food, drugs and materials.

However, if staff follow the protocols advocated by the WHO, and described above, outcomes can improve. Staff need to be confident that they can follow the guidelines approved for their unit, and if they are unable to do so, be able to address these deficits in care provision. Nursing staff are often better at following the guidelines than doctors, who may try to individualise treatment as they would for other children. The recording charts, weight charts and pro forma are tools that greatly help in the management of these children.

Analysis has shown that the main reasons for death are inappropriate medical interventions, such as fluid overload from ORS, blood transfusion, and the use of diuretics in oedema. Another reason is failure to adhere to the guidelines, due to either a lack of resources, or a lack of understanding of the differences in the care needs of this group of children. A significant and often unrecognised cause of death and relapse is inadequate discharge planning, or premature discharge.

However, perhaps the greatest problem is posed by the limited human resources on the malnutrition ward, with an insufficient number of skilled personnel, and constant movement of staff as soon as they are trained. The greatest resource that a unit can have is a motivated, trained and experienced staff, who have the basic resources to deliver the care described in this subsection.

Further reading