Nutrition

Invited author **James A Berkley**

Malnutrition, Health and Survival

Why does malnutrition occur?

Poverty, food insecurity, infections, and lack of care are the main direct causes. Underlying factors include global food prices, trade tariffs, d political commitment, political instability, civil unrest, disasters, climate, geography, agricultural factors (e.g. soil characteristics, and factors affecting animal husbandry). Seasonal availability of cereals, fruit, or vegetables affect dietary intake. Cultural preferences include gender inequality, early weaning, lack of exclusive breast feeding, d birth intervals and large family size. d breast feeding because of fears of HIV transmission contribute to malnutrition. Inadequate care is by sick parents, and orphanhood. Certain cereals may limit the bioavailability of micronutrients. Infection d appetite, intake and absorption, i nutrient requirements, and i urinary and intestinal losses. This → a vicious cycle of infection and malnutrition. Infections → malnutrition, esp. persistent diarrhoea, pertussis, measles, intestinal parasites, TB, and HIV/AIDS.

Effects of malnutrition on health and survival

- *Immunity:* Malnutrition kills by i susceptibility to infections. Malnutrition contributes to ~50% deaths among children aged 6 mo - 5 y, and an unknown proportion outside that age range, by d barrier functions of skin, gut and respiratory mucosa, d systemic immunity, and slowing tissue repair after infection. Vit A and Zn deficiency are important.
- *Growth and development:* Dietary and genetic factors affect growth. Better diet before the age of 2 y l lifelong improvements.
- *Organ function:* Severe malnutrition → profound physiological and metabolic disturbances, with failure of normal homeostatic mechanisms (b p 628). Nutritional deficiencies affect the eye (vit A), thyroid gland (iodine), brain (Fe, essential fatty acids), (vit A and Zn), and ovaries (anorexia).
- *Pregnancy outcomes:* Maternal malnutrition affects maternal health, intrauterine development, d birth weight, lactation, and infant/child health.
- *Foetal programming:* Intrauterine malnutrition i risk of cardiovascular diseases, hypertension, diabetes, and stroke in adulthood.

What are the main aspects of treatment?

- *Immediately treat life-threatening complications*
- *Re-establish physiology and metabolism (stabilisation)*
- *Treat infections*
- *Promote growth and repair*

Malnutrition with medical complications should be treated in a hospital or inpatient feeding centre. Uncomplicated moderate acute malnutrition (MAM) or severe acute malnutrition (SAM) may be treated in an outpatient or community setting with Ready to Use Therapeutic Food (RUTF).

Key points in malnutrition

- Most cases of severe malnutrition occur in children <5 y. In older children or adults, suspect an underlying infection (e.g. HIV, TB) or malignancy.
- Complicated severe acute malnutrition (SAM) has a high mortality because immunity, metabolism and physiology are dysfunctional, → ↓ appetite, malabsorption and failure of homeostasis.
- Children and adults with kwashiorkor have severe physiological disturbances and high mortality.
- Assessment and management requires the same rigour of management as severe microbial or metabolic illness. These often co-exist.
- Infections may not demonstrate the usual clinical signs. Children may develop overwhelming sepsis quite suddenly after apparently making progress.
- Medical treatment differs from that for non-malnourished individuals.
- Initially, in complicated SAM, cautious feeding aims to re-start metabolic processes and improve absorption by carefully replacing energy and micronutrients without overloading a fragile system.
- Ready to use therapeutic food (RUTF) enables effective management of uncomplicated SAM in the community.
- Children and adults with moderate acute malnutrition (MAM) are also at i risk of death in the community and in hospital.
- Modern programmes integrate the management of SAM and MAM.
- The HIV epidemic has had a profound impact on the presentation and outcomes of malnutrition.
- After rehabilitation has started, children and adults may return to homes with continuing poor diet and a high burden of exposure to infections before fully recovering immunity.
- Poverty and social exclusion are often underlying factors. Your patient may not be able to afford the fare to come for follow up or buy the drugs that have been prescribed.
- The role of health professionals in advocacy for prevention of malnutrition is vital.
- Obesity is also an increasing problem in resource-poor countries. Over- and under-nutrition may co-exist in the same household.

Measuring nutritional status

Anthropometry (body measurement) quantifies malnutrition according to international standards. In children, measurement of mid-upper arm circumference (MUAC) is increasingly used, and the most simple to undertake. Weight and height measurements are more traditional, but require more equipment. They can be useful to detect wasting and stunting (see below) as well as for individual monitoring over time e.g. growth velocity.

Weight and Height

- Weight for height (W/H): weight relative to standard weight for a child of the same height. W/H indicates acute malnutrition. <2 y, measure length (lying) rather than height (W/L). Low W/H = wasting, and indicates acute malnutrition.
- Height for age (H/A): height relative to the standard height for a child of the same age. Low H/A = stunting and indicates chronic malnutrition. Wasting and stunting may occur together.
- Weight for age (W/A): weight relative to the standard weight for a child of the same age. Low W/A = underweight. W/A does not distinguish acute from chronic malnutrition since a tall thin child may be the same weight as a short fat child of the same age. W/A is thus not used for diagnosis of acute malnutrition, but plotted over time, W/A is a useful marker of progress (see fig 17.3).

W/A, H/A, and W/H are expressed as Z scores, e.g. if weight is 2 SD below the mean weight of normal children of the same age, the Z score is –2 (b p 626); or it can be expressed as centiles.

Mid upper arm circumference (MUAC)

MUAC is measured using a tape or marked plastic strip around the left upper arm. Between ages 6 mo–5 y MUAC increases slowly, so simple cut-off values may be used for nutritional assessment. MUAC <115 mm is equivalent to a W/H Z score of –3 (i.e. SAM). MUAC<125 mm is equivalent to a W/H Z score of –2 (i.e. MAM). MUAC is much quicker and easier than calculating W/H, esp. in sick patients. MUAC is a good predictor of mortality. There is no need to use both MUAC and W/H.

Body mass index (BMI).

BMI assesses under-nutrition or obesity among non-pregnant adults. Normal values vary with age in childhood and BMIis affected by individual variation in build and muscle bulk, so should be interpreted with this in mind. BMI= weight/(height)^2.

'Visible Severe Wasting' (VSW)

VSW = muscle wasting in buttocks, loss of subcutaneous fat, or prominence of bones e.g. ribs/spine/pelvis. VSW is very insensitive, identifying only severe cases of SAM. It is better to use MUAC.

Fig 17.1 Measuring MUAC
source: "How to weigh and measure children" UN 1986

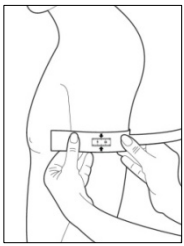

Table 17.1 **Thresholds for malnutrition in children, adults, pregnant women, and the elderly**

Age group	Severe	Moderate	At risk
MUAC			
Infants <6m	-		
Children 6m–5 y	<115 mm	115–124 mm	125–135 mm
Children 5y-18y	-		
Adults*	<160 mm	160–185 mm	-
Elderly*	<160 mm	160–175 mm	-
Pregnant/lact. women*	<185 mm	185-210 mm	210-230mm

* decisions to admit may also be influenced by recent weight loss or chronic illness e.g. HIV

Weight for Height Z Score**			
Infants <6 mo	<-3	-3 to -2	-2 to -1
Children 6mo –5 y	<-3	-3 to -2	-2 to -1
Children 5y -18y	<-3	-3 to -2	-2 to -1
Adults	-		
Elderly	-		
Pregnant/lact. women	-		

** see WHO charts and tables at http://www.who.int/childgrowth/standards/en/

BMI			
Children 0-18y	Varies with age**		
Adults***	<16	16-17	17-18.5
Pregnant/lact. women	-		
Elderly	-		

*** In adults, BMI >25 is overweight and BMI >30 is obese

Kwashiorkor	Kwashiorkor = severe malnutrition at any

age

Reference growth standards and growth charts

Weight-for-length GIRLS Birth to 2 years (z-scores)					Weight-for-length BOYS Birth to 2 years (z-scores)				
cm	-3 SD	-2 SD	-1 SD	Median	cm	-3 SD	-2 SD	-1 SD	Median
60.0	4.5	4.9	5.4	5.9	60.0	4.7	5.1	5.5	6.0
60.5	4.6	5.0	5.5	6.0	60.5	4.8	5.2	5.6	6.1
61.0	4.7	5.1	5.6	6.1	61.0	4.9	5.3	5.8	6.3
61.5	4.8	5.2	5.7	6.3	61.5	5.0	5.4	5.9	6.4
62.0	4.9	5.3	5.8	6.4	62.0	5.1	5.6	6.0	6.5
62.5	5.0	5.4	5.9	6.5	62.5	5.2	5.7	6.1	6.7
63.0	5.1	5.5	6.0	6.6	63.0	5.3	5.8	6.2	6.8
63.5	5.2	5.6	6.2	6.7	63.5	5.4	5.9	6.4	6.9
64.0	5.3	5.7	6.3	6.9	64.0	5.5	6.0	6.5	7.0
64.5	5.4	5.8	6.4	7.0	64.5	5.6	6.1	6.6	7.1
65.0	5.5	5.9	6.5	7.1	65.0	5.7	6.2	6.7	7.3
65.5	5.5	6.0	6.6	7.2	65.5	5.8	6.3	6.8	7.4
66.0	5.6	6.1	6.7	7.3	66.0	5.9	6.4	6.9	7.5
66.5	5.7	6.2	6.8	7.4	66.5	6.0	6.5	7.0	7.6
67.0	5.8	6.3	6.9	7.5	67.0	6.1	6.6	7.1	7.7
67.5	5.9	6.4	7.0	7.6	67.5	6.2	6.7	7.2	7.9
68.0	6.0	6.5	7.1	7.7	68.0	6.3	6.8	7.3	8.0
68.5	6.1	6.6	7.2	7.9	68.5	6.4	6.9	7.5	8.1
69.0	6.1	6.7	7.3	8.0	69.0	6.5	7.0	7.6	8.2
69.5	6.2	6.8	7.4	8.1	69.5	6.6	7.1	7.7	8.3
70.0	6.3	6.9	7.5	8.2	70.0	6.6	7.2	7.8	8.4
70.5	6.4	6.9	7.6	8.3	70.5	6.7	7.3	7.9	8.5
71.0	6.5	7.0	7.7	8.4	71.0	6.8	7.4	8.0	8.6
71.5	6.5	7.1	7.7	8.5	71.5	6.9	7.5	8.1	8.8
72.0	6.6	7.2	7.8	8.6	72.0	7.0	7.6	8.2	8.9
72.5	6.7	7.3	7.9	8.7	72.5	7.1	7.6	8.3	9.0
73.0	6.8	7.4	8.0	8.8	73.0	7.2	7.7	8.4	9.1
73.5	6.9	7.4	8.1	8.9	73.5	7.2	7.8	8.5	9.2
74.0	6.9	7.5	8.2	9.0	74.0	7.3	7.9	8.6	9.3
74.5	7.0	7.6	8.3	9.1	74.5	7.4	8.0	8.7	9.4
75.0	7.1	7.7	8.4	9.1	75.0	7.5	8.1	8.8	9.5

Fig. 17.2 Example W/L reference tables for boys and girls 0–2 y Full tables are found at: http://www.who.int/childgrowth/standards/en/

Pathophysiological consequences of severe malnutrition

- Energy: d energy intake, malabsorption, d liver glycogen stores and gluconeogenesis → i susceptibility to hypoglycaemia and hypothermia.
- Gut: achlorhydria, d gut motility, bacterial overgrowth, villous atrophy and gut enzyme deficiencies impair digestion and absorption.
- Liver: d protein synthesis (e.g. albumin, transferrin) and d detoxification. Abnormal metabolites of amino acids and drugs are produced. i production of acute phase proteins (e.g. CRP, ferritin).
- Renal function: may d with inability to excrete Na+ and phosphate.
- Whole body Na^+↑ and K^+↓. Na^+/K^+ ATPase pumps are impaired, electrolytes are not normally distributed across cell membranes.
- Muscle wasting and diarrhoea l K^+, Zn^{++}, Ca^{++}, Mg^{++} and Cu^{++} deficiency.
- Red cell mass ↓, liberating iron. Unbound iron, free radicals and anti-oxidant deficiency → infections, inflammation and cell membrane dysfunction.

Immunity

- Surface and mucosal barriers are impaired → pathogen/antigen entry.
- Skin excoriation → local sepsis and septicaemia.
- Gut inflammation and damage → bacterial translocation (bacteraemia).
- Cellular immunity: thymic atrophy, ↓ cellular immunty, ↓ neutrophil function.
- Humoral immunity — SAM has little effect on antibody production following immunization. Secretory IgA is ↓.

Kwashiorkor (oedematous malnutrition)

- Kwashiorkor was first described by Dr Cecily Williams in Ghana, in 1935, among children weaned from the breast onto a maize-based diet.
- It is unclear why some children develop kwashiorkor and others not (see b p XX for clinical features of kwashiorkor and comparison with marasmus).
- Dietary deficiencies of energy, protein, and micronutrients are common to both kwashiorkor and marasmus.
- Oedema of kwashiorkor improves on a low protein initial diet.
- Anti-oxidant deficiencies have been postulated as cause of kwashiorkor, However a multiple anti-oxidant supplements do not affect the subsequent incidence of kwashiorkor.
- Kwashiorkor may be related to enteropathy

Pathophysiology of micronutrient deficiencies

- ↓ intake of micronutrients → depletion of body stores → specific clinical signs in severe cases: xerophthalmia (Vit A), pellagra (nia-

cin), scurvy (Vit C), anaemia (iron/folate/Vit B12), rickets (Vit D/calcium), clotting abnormalities (Vit K) and goitre (iodine).
- ↓ intake of protein, essential amino acids and minerals → ↓ growth rate or ↓ weight, usually without specific signs of deficiency.

Clinical assessment of nutrition

History and physical examination

Look for complications, physiological dysfunction, infections, signs of specific nutrient deficiencies, underlying illness, feeding patterns and modifiable risk factors.

History

Concurrent illness/symptoms
- Current or recent illness (diarrhoea, malaria, lower respiratory tract infection (LRTI), etc).
- Duration, frequency, and nature of vomiting or diarrhoea.
- Behaviour and activity changes (crying, irritable, apathy, anorexia).
- Hydration: Recent sinking of eyes, time when urine was last passed.

Feeding history
- Food and fluids taken in past few days? Thirst? Appetite?
- Breastfeeding history: how long for? Mixed or exclusive BF? Is the child breastfeeding now? Age of introduction of complementary feeds?
- Usual diet before current illness, lack of food in the household or the quality of food, recent change in diet

Growth history
- Birth weight; prematurity; whether a twin.
- Review growth chart

Other medical history
- HIV/AIDS status of child or mother
- Development milestones reached (e.g. sitting unsupported 9mo standing unsupported for 1-2 s at 12mo)
- Immunization and Vit A doses up to date? (b p imms section).

Family history
- Deaths of siblings or parent.
- Is the mother ill or malnourished?
- TB contact?

Physical examination
- General appearance, behaviour, mood (apathy, irritability), level of consciousness, facial appearance, signs of kwashiorkor/marasmus.
- Fever or other signs of infection
- Pallor

- Enlarged or tender liver, jaundice; abdominal distension, tenderness.
- Skin changes: desquamation, oedema, rash (e.g. post measles), exfoliation, fungal infection, cancrum oris
- Signs of cerebral palsy or congenital syndrome (e.g. Down's).
- Appetite test (see b p 633).

Clinical features of marasmus

- *Wasting: low MUAC* or W/H Z score
- *Emaciated:* thin, flaccid skin ('little old man' appearance), grossly ↓ fat and muscle tissue; prominent spine, ribs, pelvis.
- *Behaviour:* alert and irritable.
- *Distended abdomen* due to weakened abdominal muscles and gas from small bowel bacterial overgrowth.

Clinical features of kwashiorkor

- *Low or normal MUAC* or W/H Z score
- *Oedema:* bilateral pitting limb oedema; periorbital oedema. May be generalised.
- *Skin changes:* desquamation, often in the flexures and perineum.
- *Hair changes:* dry, thin hair which may become depigmented appearing brown, yellowy-red, or white.
- *Hepatomegaly* is common.
- *Behaviour:* miserable, lethargic, and apathetic with sad facies.

These may occur together: marasmic–kwashiorkor.

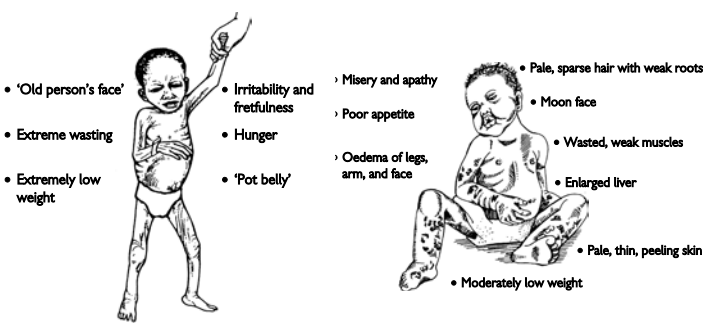

Fig. 17.1 Signs of marasmus (left) and kwashiorkor (right).

Complicated or uncomplicated malnutrition?

Complicated malnutrition indicates severe physiological or metabolic changes, or infection. The child must be treated as an in-patient until stabilized. Uncomplicated malnutrition may be treated in the community.

Children with **uncomplicated malnutrition**:
- Are alert with no respiratory distress, shock, hypoglycaemia, hypothermia, severe diarrhoea, dehydration, convulsions or severe oedema
- and have no other reason for admission to hospital (e.g. pneumonia)
- and pass an appetite test (see box).

Children with **complicated malnutrition** fail one or more of these criteria.

Appetite Test

Appetite is a good marker of metabolic disturbance, and whether a malnourished child needs to be admitted.
- In a quiet area, explain to the mother/carer the purpose of the test.
- The caregiver washes her hands, with soap and water.
- The caregiver either offers the RUTF (see b p 639) from the packet or puts a small amount on her finger and gives it to the child.
- If the child refuses, the caregiver continues to encourage the child. The child must not be forced to take the RUTF.
- A child might refuse to eat the RUTF because of the strange environment - in this case the carer should take the child to a quiet place and gently encourage them.
- Offer plenty of water to drink from a cup while he/she is taking RUTF.
- The mother must be happy that the child is eating the RUTF and the clinic worker should actually see the child eat the RUTF.
- The child should be able to eat about a quarter of a 92g sachet.
- Any child not eating the RUTF has failed and should be admitted.

Laboratory evaluation

The following can be used, but are rarely available, and some are altered by inflammation:
- Serum proteins (e.g. albumin, transferrin).
- Micronutrients (e.g. Vit A, Zn, Vit B12, Vit D, Ca, folate, ferritin).
- Red cell enzymes (e.g. glutathione reductase)
- Urinary micronutrients (e.g. iodine).

Management plan

This depends on whether the child has complicated or uncomplicated malnutrition, and MAM or SAM. Use the algorithm below to classify the malnutrition and decide on type of treatment.

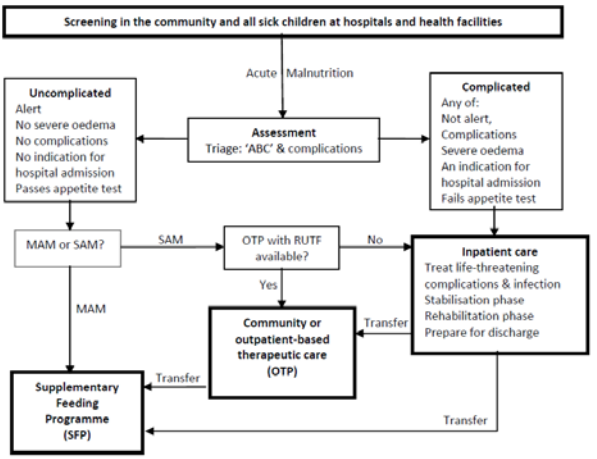

Fig. 17.3 Algorithm for categorization and management of children with malnutrition

Medical management within inpatient therapeutic nutrition programmes

Manage life-threatening complications

See chapter 1 on management of airway, breathing circulation (ABC) and shock in severe acute malnutrition (b p x).Infection, dehydration, hypothermia, hypoglycaemia, severe anaemia and electrolyte abnormalities are common complications. See for management of hypoglycaemia b p x. Other complications are discussed below.

Hypothermia

Defined as rectal temp <35.5°C) it is associated with infection and/or hypoglycaemia. It is a dangerous prognostic sign. Change wet nappies and bedding, ensure child does not get cold during washing. Provide blankets/lamps/heaters esp. at night. Encourage 'kangaroo' technique: mother lies supine with child on her chest, covered by her clothes and blankets. Cover child's head with a cap to d heat loss.

Severe anaemia

Transfuse if Hb<4g/dL; or 4-6g/dL with respiratory distress. If not shocked, give 10ml per kg body weight of whole blood, slowly over 3 hours. If shocked, you should not be reading here yet – see b p X

Dehydration

0 Intravenous fluid resuscitation is controversial and may result in potentially dangerous under- or over-hydration. Use oral rehydration fluids designed for severely malnourished children (ReSoMal) unless there is established shock (b p x).

Dehydration may be difficult to assess as sunken eyes and d skin turgor may be due to acute malnutrition. Assessment is best made on the basis of history or observed fluid losses. Use ReSoMal rather than standard oral rehydration solution (b p 248–251): give 5 ml/kg orally/NG every 30 mins for the first 2 h; aim to give 70–100 ml/kg over 12 h. ReSoMal can be given with F75. Once rehydrated, continue F75 and replace volumes lost in stool. Once rehydrated, continue F75 and replace volumes lost in stool. Continue breastfeeding wherever possible. Reserve IV fluids for children in shock, or maintenance only in those not tolerating oral/NG fluids (with monitoring as described above). Useful signs of rehydration include return of tears, moist mouth, less sunken eyes and fontanelle, and improved skin turgor. Daily weight is a guide to changes in hydration status.

Infection

Bacterial sepsis: a common cause of death in severely malnourished children. Signs of infection may be absent, so give all children with complicated SAM broad-spectrum antibiotics:

- ampicillin (50mg/kg 6 hrly IM/IV) plus gentamicin (7.5 mg/kg IM/IV daily) or ceftriaxone (50 mg/kg daily by IV injection over 2–4 mins) for 7 d for complicated SAM.
- Metronidazole (7.5mg.kg 8-hrly for 7 d) may be added if suspected protozoal (e.g. amoebiasis) or anaerobic infection.
- If receiving cotrimoxazole prophylaxis for HIV, this should continue.

Other infections:
- Blood film/rapid diagnostic test for malaria on admission (b p x).
- Urinary tract infections are common in malnourished children. Do a urine dipstick test and if possible, culture (b p x).
- All severely malnourished children should have a diagnostic testing and counseling (DTC) for HIV performed by the clinician at admission.
- After stabilisation give a single dose of albendazole 400 mg oral to children >24 mo ; (children 12-23 months, 200 mg) to treat helminth infection.
- Screen for other infections as clinically indicated, consider if available chest X-ray; LP; blood cultures; consider TB.

Diarrhoea: is common; may be due to infection, osmotic load of the food, lactose intolerance or premature transfer to the next feeding phase. Cohort patients and wash hands to avoid spread.
- If severe diarrhoea, go back to F-75 or a non-milk-based feed. If possible, test stool for reducing substances (= lactose malabsorption; stool pH usually <5.5). Check volumes of feeds are correct — large boluses may → diarrhoea.
- Severely malnourished children with HIV may have *Cryptosporidium* or other intestinal parasites — do microscopy if possible.
- Treat dysentery with antibiotics according to local protocols b p 221.
- Diarrhoea can be due to systemic infection (e.g. sepsis)
- Zn is already included in therapeutic feeds, no extra Zn is required.

Tuberculosis: Severely malnourished children have ↑ TB risk, but clinical features are non-specific and microbiological diagnosis rarely possible. Consider TB in children who do not respond quickly to standard nutritional and medical treatment, and in those with a history of household TB contact (see b p 158). Cohort patients with TB to avoid nosocomial transmission. Investigations for TB include fine needle aspiration of cervical glands, sputum induction (nebulised hypertonic saline) or gastric washings (b p X).

Electrolyte abnormalities

Hyponatraemia may be present but does not reflect a deficiency in total body Na^+; avoid giving too much Na^+. Hypokalaemia is common, especially with diarrhoea, and contributes to cardiac arrhythmias. Hypomagnesaemia may contribute to cardiac arrhythmias and muscle twitching. Phosphate levels are often low. K, Mg and other electrolytes are contained in therapeutic feeds. ReSoMal and the recom-

mended IV fluids contain less sodium and more potassium than ORS and fluids used for well nourished children.

Inpatient therapeutic nutrition programme

The principles are similar for all ages. Combine therapeutic nutritional treatment with intensive medical care (see b p XX). Management of infants <6 mo is covered on b p XX. Nutritional treatment may be divided into stabilisation, transition, and rehabilitation phases.

1) *Stabilisation phase:* Establish low protein, fat and energy feeds (F75) with just enough micronutrients to restart metabolic and physiological processes. This also allows oedema to clear.
- Give 130ml/kg F75 daily, divided into 3hrly feeds (see Table 17.2).
- Continue feeding at night to prevent hypoglycaemia.
- If unable to take sufficient feed orally, give via NG tube. Offer each feed by mouth and give remainder via NG. NG may be removed when child takes most of daily diet orally.
- Weigh child each morning before a feed; assess oedema daily.
- Monitor vital signs at least twice a day.
- Give folic acid 5 mg on admission.
- Give Vit A (age <6mo 50,000 iu; 6–12mo 100,000 iu; >1 y 200,000 iu) on day 1 except those who had Vit A <1 mo or with severe oedema
- If signs of Vit A deficiency, (see b p 652 give on d 1, 2 and 14.
- All other micronutrients are contained in commercial F75.
- Iron should not be given in the stabilisation phase as it is pro-inflammatory, pro-oxidant and can promote infection (e.g. *Salmonella*).

2) Transition phase: Once appetite returns, oedema starts to clear and medical complications are treated (usually after 73–7 d), child may enter transition phase in which dietary intake ↑ under close monitoring. Problems can occur with ↑ dietary Na^+, fluid, osmolality, and amino acids. Key components of transition phase:
- Switch to the same amount of F-100 therapeutic milk.
- Continue to feed regularly (e.g. 8 feeds in 24 h).
- Gradually ↑ successive feeds by 10 ml until refusal.
- Monitor vital signs at least twice a day.
- Weigh and assess oedema daily.
- Once tolerating feeds, provided improving and not losing weightl rehabilitation phase.

Note: oedematous children lose weight before starting to gain.

0 Failure to lose oedema or gain weight: is almost always because of inadequate intake of feeds or infection.

Therapeutic and supplementary feeding products

Milk-based products

F75 and F100 are specially formulated milks, available commercially or locally prepared (see b p 646). F75 (75 kcal and 0.9g protein/100 ml) is used for cautious feeding in the stabilisation phase to restart metabolic processes, ↓oedema and regain appetite. F100 (100kcal and 2.9g protein/100ml) is for catch up growth in the transition and rehabilitation phase. F75 and F100 contain a balance of protein, energy, fats and commercial (but not all local) preparations contain micronutrients. They do not contain additional iron. Because of a short shelf life they are not suitable for use at home. Commercial F75 and F100 contain maltodextrin instead of sugar, which ↓ osmolarity and ↓ risk of osmotic diarrhoea.

Ready to use therapeutic food (RUTF)

RUTF is a paste based on peanuts, or cereals/legumes that is lipid, protein, energy, and micronutrient-rich. RUTFs are very palatable, with a nutritional composition similar to F100 (incl. extra K^+). RUTF is resistant to microbial contamination (it can be stored safely at ambient tropical conditions for many months), lower osmolarity than F100 and micronutrients (e.g. Vit A) are preserved. Typically, a 92g sachet = 500kcal.

RUTF can eaten from the packet and needs no cooking.

Encourage breastfeeding and adequate water intake; other foods should not be encouraged until weight has been restored. RUTF can be used at home for uncomplicated SAM or interchangeably with F100 in hospital. There is no RUTF equivalent of F75 and RUTF is unsuitable for the stabilisation phase of complicated SAM.

RUTF can be produced locally, but the commercial formulations are usually widely available, often supported by NGOs or UNICEF. Commercial RUTF contains maltodextrin instead of sugar, which ↓osmolarity. Local production requires a properly formulated vitamin and mineral mix, which must be bought. Aflatoxin contamination can be a risk. Mechanical mixing is required. Instructions are available on the WHO website:

http://www.who.int/nutrition/topics/backgroundpapers_Local_production.pdf

Supplementary feeds

Supplementary feeds are used to treat moderate acute malnutrition (MAM) in supplementary feeding programmes (SFP). They typically include micronutrient fortified, blended cereals and pulses as dry rations and vegetable oil. Local food habits may be important in determining appropriate rations. Ready to use supplementary food is also available, and is specially designed for nutritional requirements in MAM.

3) Rehabilitation phase: When ↑appetite, ↓oedema and medical complications are stable, child may enter the rehabilitation phase, which aims to promote rapid growth.
- Gradually ↑ to ≥200ml/kg/d F100 (=200kcal/kg/d)
- 5 feeds per d are usually needed.
- RUTF and F100 are interchangeable: 20g RUTF = 100ml F100.
- Give iron 3 mg/kg/d, except when using RUTF, which contains 11.5mg iron per sachet.
- Record the dietary intake.
- Give albendazole as described above b p XX.
- Check temperature and general condition daily.
- Check weight and oedema 3 x per wk.
- Encourage play therapy and physical activity to promote speech and motor development.
- Promote breastfeeding (see box).

4) Preparing for discharge from inpatient care

Discharge should be planned in advance with the mother/carer. Refer to an outpatient therapeutic nutrition programme (OTP) or a supplementary feeding programme (SFP) if now moderately malnourished. Key issues during this phase include:
- Stable children may be discharged to OTP, even if they still meet criteria for SAM. However, they must pass an appetite test with RUTF (see b p 6xx).
- Discharge to SFP means being completely stable, and eating well.
- Provide nutrition and health education, cooking demonstrations
- Support increasing household food security.
- Support families with serious social problems.
- Give Vit A, if not given at admission.
- Encourage regular visits to under-5 clinics, immunization, and regular Vit A prophylaxis (3 times/y).
- Vaccinate all children aged 9 mo –15 y against measles, unless proof of previous vaccination. Children immunized <9 mo should be re-vaccinated as their previous immune response may be inadequate.

5) Follow-up
- Follow up should ideally be through an established OTP or SFP.
- If no OTP/SFP is available, therapeutic feeding should continue until the WHZ is > -2 or MUAC >12.5cm and oedema has resolved for at least 2 wks. See child after 1 wk then every 2 wks to check weight gain (after nutritional recovery child should grow at 1–2 g/kg body weight/day). Check for dietary/medical reasons for poor weight gain.
- Ensure child is integrated into clinic or community-based programme for monitoring progress of 'at risk' children, and can be referred back in case of problems.

0 ***Deterioration... l ABC, complications l stabilization phase.***

Table 17.2 Amounts of F75 required for stabilisation phase

Weight (kg)	2-hrly feed	3-hrly feed	4-hrl feed		Weight (kg)	2-hrly feed	3-hrly feed	4-hrly feed
2.0	10	0	45		**6.2**	70	100	135
2.2	25	35	50		**6.4**	70	105	140
2.4	25	40	50		**6.6**	75	110	145
2.6	30	45	55		**6.8**	75	110	150
2.8	30	45	60		**7.0**	70	115	155
3.0	35	50	65		**7.2**	80	120	160
3.2	35	55	70		**7.4**	80	120	160
3.4	35	55	75		**7.6**	85	125	165
3.6	30	60	80		**7.8**	85	130	170
3.8	40	60	85		**8.0**	90	130	175
4.1	45	65	80		**8.2**	90	135	180
4.2	45	70	90		**8.4**	90	140	185
4.4	55	70	95		**8.6**	95	140	190
4.6	50	75	100		**8.8**	95	145	195
4.8	55	80	10		**9.0**	100	145	200
5.0	55	80	110		**9.2**	100	150	200
5.2	55	85	115		**9.4**	105	155	205
5.4	60	90	120		**9.6**	105	155	200
5.6	60	90	125		**9.8**	110	160	215
5.8	65	95	130		**10.0**	110	160	220
6.0	65	100	130					

Table 17.3 Amounts of RUTF required for rehabilitation phase

Weight (kg)	92g Sachets per day	92g Sachets per week
3.5 – 3.9	1.5	11
4.0 – 5.4	2	14
5.5 – 6.9	2.5	18
7.0 – 8.4	3	21
8.5 – 9.4	3.5	25
9.5 – 10.4	4	28
0.5 – 11.9	4.5	32
≥12	5	35

Outpatient therapeutic nutrition programme (OTP)

The aim of an OTP is to conduct screening, identify the severity of malnutrition and plan a treatment regimen. For uncomplicated SAM, provide medicines and therapeutic feeds, and advise patients/carers.

1. Treat infection (often subclinical)

- Treat all children with SAM for infection whether they have clinical signs or not. For uncomplicated SAM, give cotrimoxazole oral for 5 d. If the child is on cotrimoxazole prophylaxis, this should continue, and give amoxicillin oral for 5 d.
- Give a single dose of albendazole (doses see b pXX).
- Test for HIV and refer to comprehensive care services if HIV+.
- Diagnose and treat malaria and UTIs, URTIs, skin infections etc.
- Ensure child is up to date with immunizations.
- Give Vit A (doses see b pXX) on day 1 except those had Vit A within a month; if signs of Vit A deficiency, give on days 1, 2 and 14.
- Additional iron and folate are not needed, they are in RUTF.
- Follow up the child weekly until adequate weight gain is achieved.

2. Outpatient RUTF programme

The aim of this programme is to manage children with SAM at home provided they can eat adequate amounts of RUTF.

Give sufficient RUTF till the next visit:

- Follow up every 2 wks until weight gain satisfactory (e.g. >5 g/kg/d).
- Advise carer to give up to 100 kcal/kg body weight/d (using tea-spoon equivalents of RUTF) until oedema has resolved.
- If no oedema, or once oedema resolved, give 150–220 kcal/kg body weight/d.
- RUTF of 200 kcals/kg body weight/day can → a daily weight gain of up to 20 g/kg body weight.
- Advise carer of need to feed frequently, to keep child warm (esp. at night), and to come back to the clinic if the child develops an infection or refuses RUTF.
  RUTF is sometimes shared with other children. If the intake of RUTF falls to 100–150 kcals/kg body weight, daily weight gain to falls to 75–10 g/kg body weight/d. Give cereal/legume (e.g. corn-soy blend) supplement to the family to ensure other children in the family do not eat RUTF intended for the index child.

Give nutritional guidance to improve dietary intake

- Advise on need to give extra food in convalescent phase of an illness and how to ↑ the protein and micronutrient content of traditional diet if possible.
- Advise on access to local programmes which ↑ food security (using local community development programme and/or a local programme of food supplements).

- Provide a 'take home' ration of food to provide protein, energy and micronutrients in a form which is palatable and can be stored safely without refrigeration (e.g. nutritional pastes).
- Discharge to SFP when appropriate (MAM).
- For kwashiorkor, therapeutic feeding should continue until oedema has resolved for >2 wks.

Supplementary feeding programmes (SFP)

The aim of an SFP is to conduct screening, identify the severity of malnutrition; plan a treatment regimen (refer to paediatric ward, OTP, SFP or home). For MAM, provide supplementary feeds, identify and advise patients/carers. MAM ↑ risks of infectious and development of SAM.

Food rations include cereals, pulses, legumes, oil, sugar and micronutrients, e.g. corn-soy blend (see b p 641). In some areas, ready to use supplementary foods (RUSF) may be available. RUSF is similar to RUTF but designed for MAM and to be used alongside other foods.

Medical treatment:
- Vit A, as above – unless already given in the last month.
- Albendazole as above, unless already given
- Iron and folate as per national recommendations.
- Measles immunization unless proof of previous immunization. Children immunized <9mo should be re-immunized.

Nutrition counselling:
The mother or carer should be advised on giving a healthy diet using locally available foods. Work with a nutritionist to make a chart of foods available rather than trying to 'educate' mothers about carbohydrates, fats, protein etc.

Discharge criteria: Children age 6 - 59 mo: MUAC >12.5cm; children of any age: W/H >-2 z scores; adults: MUAC >18.5cm, or if pregnant or HIV+, MUAC >23cm.

HIV/AIDS and malnutrition

Among severely malnourished children in sub-Saharan Africa, the prevalence of HIV approaches 15% in community treatment programmes, and 60% in complicated SAM. Almost all severely malnourished HIV+ children have low CD4 counts and need anti-retroviral treatment (ART). CD4 counts are not low in severely malnourished children without HIV.

All severely malnourished children should be tested for HIV because selective testing increases fear and stigma. A +ve test in the child reveals the mother is also HIV+. The benefits of making the diagnosis include appropriate treatment of OIs, co-trimoxazole prophylaxis, ART, improving mothers' health, advice on infant feeding, and prevention of future mother-to-child transmission (see HIV ch b p XX).

Specific issues in HIV+ children:

- Poor dietary intake due to weakness, painful oral lesions (e.g candidiasis), anorexia due to fever/infections, and sickness of a parent or guardian (often also HIV+) → limited care and food provision.
- Malabsorption and chronic diarrhoea due to intestinal parasites (e.g. *Cryptosporidium*) → nutrient losses from the intestine.
- ↑ energy expenditure due to intercurrent infections.
- Severe weight loss and growth faltering are common.
- Micronutrient deficiencies (incl. vit A, Zn) are common.
- Anaemia is common in HIV +ve children, usually the result of chronic inflammation rather than micronutrient deficiency. Iron supplements may be harmful in HIV due to the ↑ in oxidative stress and HIV viral load.

Management

- Give co-trimoxazole prophylaxis (b pXX HIV chapter)
- Give stabilisation therapy as above
- During rehabilitation, aim at 220kcal/kg/day
- Manage diarrhoea energetically. Persistent diarrhoea due to *Cryptosporidium* may not improve until ART is started.
- Treat infections and OIs vigorously (b p 83–103).
- Refer to an ART clinic; measure CD4 count if possible.
- Optimum time to start ART is unknown. Because of metabolic effects of ART, children should ideally have stabilized, appetite returned and oedema resolved.
- Failure to gain weight is common in HIV. Investigate co-morbidities incl. TB and intestinal parasites, and consider early ART.
- Prevent mother-to-child transmission — see b p 132.
- Provide health care for the mother and/or carer, incl. HIV testing ± CD4 count to assess need for ART. A healthy mother/carer is crucial to the child's recovery.

During Follow up

- Ensure nutrition during OIs.
- Ensure best possible food and drinking water hygiene.

Outcome: HIV roughly doubles the case fatality of SAM. Weight gain is often slower and may not increase until ART is started.

Nutrition in people with HIV/AIDS

Good nutrition is essential, in order to maintain immune competence and strength and minimize the impact of infections. HIV/AIDS patients (even when asymptomatic) need ↑ food intake:
- At least the recommended daily allowance (RDA) of vits A, B, C, E, folic acid; and minerals (e.g. selenium, Zn).
- Even more during recovery from an infection.

Nutritional education should start once a person is identified as HIV+. Focus on how to meet ↑ dietary needs and prevent OIs and improve hygiene. Support the entire family, including food security, hygiene, and psycho-social care of the HIV+ individual.

To optimize intake: eat small, frequent meals; make food softer. Include body-building food (legumes, cereal, animal products), protective foods (fruits and vegetables, fortified food), and energy foods (sugar, starch and fat, staple foods).

Problems
- Nausea and vomiting: eat frequent small meals and avoid fatty food.
- Mouth sores: avoid hot and spicy foods; eat soft, mashed or liquid food.
- Anorexia: eat frequent small meals. Time ART to minimize impact of GI side-effects (e.g. nausea) on meals.

Breast feeding and HIV/AIDS

- HIV+ mothers are usually worried about transmission during breast feeding. Mixed breast/replacement feeding has greater HIV transmission than exclusive breast feeding or replacement feeding.
- Many mothers end up mixed feeding because they cannot afford to sustain replacement feeding.
- Exclusive breast feeding has lower risks of inadequate intake and infection (contamination).
- Cow's milk is especially hazardous. Although recipes for modification for infant use are available, these are impractical for poor mothers.
- Clinical trials of prophylactic ART given to the mother or child reduces transmission to 72% during breastfeeding, with few maternal side effects. Where ART prophylaxis to mother or breast feeding is available this is preferable to replacement or mixed feeding (see b p Ch 3).
- WHO recommend exclusive breast feeding up to 6 mo and partial breastfeeding continue to at least 12 mo, with maternal ART or infant Nevirapine until 1 wk after breastfeeding ceases to prevent transmission.
- Replacement feeding should not be used unless it is acceptable, feasible, affordable, sustainable and safe – in resource poor coun-

tries these criteria are very rarely met.

Severe malnutrition in infants < 6 months old

Feeding problems, incl. sickness or absence of the mother, insufficient breast milk (stress, war, drought), inappropriate alternative infant feeding (unsafe bottle feeding, use of cow's milk, early introduction of complementary (weaning) foods) and inappropriate attempts to avoid breast milk transmission of HIV, may cause SAM and illness in infants. Diagnosis of SAM <6 mo is based on:

- W/L <–3 z scores
  or
- Bilateral oedema of kwashiorkor
  or
- Weight loss and too weak to suckle effectively.

Breast feeding

Breast feeding ↑ immunity, is hygienic, clean, and cheap, and there is usually a good supply, although mothers need support. Artificial feeding risks contamination (teats, bottles, milk left standing too long, unclean water) and dilution (cost, sharing with siblings). This can → malnutrition through inadequate intake, wrong concentration and repeated episodes of diarrhoea.

During insecurity, anxiety, and migration, breast milk might be d. Mothers often think they produce less breast milk because they are themselves malnourished. Milk quantity is usually only reduced once maternal energy intake is <1600 kCal/d. Breast milk quality (esp. of micronutrients) is quickly affected by the mother's diet. A mother's complaint that she does not have enough milk should be properly investigated. When the milk production is reported to be d or stopped, breastfeeding should be encouraged and the mother supported with nutritious food. Only if there is no other option should artificial feeding be used and then the mother (or caretaker) must be trained in using the milk safely.

Medical treatment

Severely malnourished young infants require similar medical treatment regimes to older infants and children with severe malnutrition:

- Antibiotics — (b p x–x).
- Vit A (50,000 iu)
- Folic acid 2.5 mg on admission

- Note: commercial F100 already contains iron.
- Ensure that the mother has her illnesses diagnosed and treated. Maternal depression affects infant feeding and growth. Test the infant for HIV and counsel the mother appropriately.

Nutritional treatment

Aim to re-establish breast feeding whilst treating the infant. There is no stabilisation phase unless the infant has kwashiorkor.

- *S*upplement breast milk with diluted F100 (see b p 650) 130 ml/kg/d. Kwashiorkor, use F75 (130ml/kg/d) until oedema is resolving then use diluted F100. Breast feed as often as possible.
- Use a *supplemetary suckling technique* to continue to stimulate breast milk production: http://www.docstoc.com/docs/48486395/UNICEF-IMAM-Publication-pdf-National-Guideline-for--malnutrition
- When infant is gaining weight at 20g/kg/d, gradually ↓ diluted F100 until the infant is gaining weight on breast milk alone.

If no prospect of breast feeding

Start with diluted F100 (or F75 if kwashiorkor) 160 ml/kg/d. When stable and tolerating, gradually ↑ diluted F100 up to 320 ml/kg/d. Once gaining weight for 3 consecutive days, very gradually replace diluted F100 with 'normal' breast milk substitute. ↑ from 120 kCal/kg/d (normal intake) to 150 kCal/kg/d for extra growth until recovered.

Monitoring

Monitor weight gain daily. If an infant loses weight for 3 consecutive days, check the amount of food offered is not enough (breast milk plus therapeutic milk), re-screen for infections or medical causes of poor feeding. Address social problems.

Discharge

The following conditions should be met before discharge:

- Clinically well; no infections.
- Weight gain ≥100–125 g/wk without therapeutic milk supplementation for 7 d (min 5 g/kg/d, target 10 g/kg/d).
- Breastfed infants: active suckling; established breast milk production.
- Non-breastfed infants: supply of breast milk substitutes must be ensured and the caretaker should understand hygienic preparation and the dangers of artificial feeding.

Complications of severe malnutrition in infants

- *Hypothermia:* a major cause of mortality in malnourished infants. Keep infants warm: skin-to-skin contact (kangaroo position); provide blankets and caps.
- *Dehydration:* use Re-So-Mal.
- *Anaemia:* iron is only given as treatment for anaemia - not as routine
  therapy. Give iron 2 mg/kg tds (preferably in a suspension) for >3 mo, but only start after 14 d of nutritional treatment. If Hb <5 g/dl, consider blood transfusion (see P XXX).

- *Candidiasis:* is frequent in newborns — treat with nystatin.

Pregnancy

- **Nutritional counselling:** there is no strong evidence that nutritional advice to ↑ energy and protein intake during pregnancy → better outcome for infants or mothers.
- **Energy/protein supplementation:** trials have reported ↓ in small for gestational age (SGA), ↓ stillbirths and possibly ↓ neonatal deaths. There is no effect on prematurity or on long term nutritional status in mothers or infants, or improved neuro-cognitive development.
- **Iron and folic acid** in pregnancy ↓ risk of maternal anaemia, maternal mortality and stillbirth. Give iron 60mg and folic acid 400µg daily.
- **Multiple micronutrient supplementation:** is widely used in wealthy communities during pregnancy. However, in developing countries has very little benefit over iron and folate alone. Multiple micronutrient supplementation cannot be universally recommended.
- Energy/protein supplementation during pregnancy is justified in malnourished women as part of an SFP. Advise non-acutely malnourished pregnant women on a healthy, balanced diet rather than any specific regime.
- Avoid Vit A, except for overt Vit A deficiency (see b p 652).
- Cut off values used for MUAC to diagnose SAM and MAM are higher than for non-pregnant adults (see b p 625)

Breastfeeding: key issues promoted by WHO/UNICEF

- Early discontinuation of breastfeeding is associated with ↑ risk of death. Give infants no food and drink other than breast milk, unless medically indicated = exclusive breastfeeding up to 6 mo.
- Train healthcare staff and have a written breastfeeding policy
- Inform all pregnant women about the benefits of breastfeeding.
- Help mothers initiate breastfeeding within a half-hour of birth.
- Show mothers how to breastfeed and how to maintain lactation even if they may be separated from their infants.
- Promote 'rooming in': mothers and infants should remain together 24 h a day wherever possible. Encourage breastfeeding on demand.
- Give no artificial teats or pacifiers (also called dummies or soothers) to breastfeeding infants.
- Foster the establishment of breastfeeding support groups.

Nutrition in emergencies

Emergencies are typically due to famine, natural disasters, epidemics, armed conflict, and population displacement (refugees), and are exacerbated by poverty, long term food insecurity, weak infrastructure and endemic diseases e.g. HIV or kala azar (see Page X – refugee emergencies) Acute malnutrition (SAM and MAM) are common in emergencies and outbreaks of micronutrient deficiencies occur where diet is restricted. High rates of malnutrition and mortality occur during emergencies.

Current strategies are broad-based including:

- Nutritional surveillance, baseline data and early warning systems.
- National programmes on food security and training in nutrition in emergencies.
- Standardised, rapid nutrition assessments (e.g. MUAC)
- Food distribution incl. general foodstuffs, food for work, and school-based programmes.
- Targeted therapeutic and supplementary feeding with RUTF or blended dry rations with micronutrients as described below.
- Provision of other aspects of healthcare
- Promotion of breast feeding
- Non-food interventions, e.g. livelihood generation, agricultural improvement.
- Coordination between humanitarian agencies, health providers, social welfare institutions and government, and occasionally military authorities.

A set of resources can be found at:

http://www.unscn.org/en/resource_portal/index.php?themes=203

Recipes and formulas for management of malnourished children

Electrolyte/mineral solution (EMS)

This is used in the preparation of starter (F-75) and catch up (F-100) feeding formula and ReSoMal (low Na^+ oral rehydration solution [ORS], see b p 250). Sachets containing these formulae are manufactured but if not available, prepare by dissolving the following ingredients in cool, boiled water made up to 2500 ml solution.

EMS ingredients	amount (g)	mol/20 ml
Potassium chloride: KCl	224	24 mmol
Tripotassium citrate	81	2 mmol
Magnesium chloride: $MgCl_2$, $6H_20$	76	3 mmol
Zinc acetate: Zn acetate, $2H_20$	8.2	300 µmol
Copper sulphate: $CuS0_4$, $5H_20$	1.4	45 µmol
Water make up to	2500 ml	

If possible, add selenium (28 mg of sodium selenate, $NaSe0_4.10H_20$) and iodine (0.012 g of potassium iodide, KI) per 2500 ml.

Store EMS in sterilized bottles in the fridge to retard deterioration. Discard if turns cloudy and make fresh each month.

Commercial mineral and vitamin mix may be available; this is preferable as it contains all necessary micronutrients.

Nutritional rehabilitation formulas F-75 and F-100

Ready-made sachets of F-75 and F-100 are widely available but where these are not, they can be prepared using the following ingredients by mixing the milk, sugar, oil, and electrolyte mineral solution (EMS or commercial mineral and vitamin mix) into a paste, and then slowly adding warm, boiled water to make up to 1000 ml. If available, use an electric blender or hand whisk.

Commercial packets of F-75 starter formula have lower osmolality because maltodextrins replace sugar, and already contain the required micronutrients.

Alternative milk ingredients

- If only whole dried milk (WDM) available, an alternative to F-75 may
  be prepared using 35 g WDM, 100 g sugar, 20 g oil, 20 ml EMS, and water up to 1000 ml. Similarly, to prepare an alternative to F-100, use
  110 g WDM, 50 g sugar, 30 g oil, 20 ml EMS, and water up to 1000 ml.
- If only fresh cow's milk available, another alternative to F-75 may be prepared using 300 ml milk, 100 g sugar, 20 g oil, 20 ml EMS, and

water up to 1000 ml. Similarly, to prepare an alternative to F-100, use 880 ml milk, 75 g sugar, 20 mg oil, 20 ml EMS, and water up to 1000 ml. The use of fresh cow's milk carries ↑ risk of microbial contamination.

Diluted F100 (infants only)

This is a 75% dilution of F-100 used for severely malnourished infants (see b p 646). It is made by adding 350 ml water to 1 litre of prepared F-100. It supplies 75 kCal/100 ml, 10 kCal % protein, 50 kCal % fat, and is isotonic with a medium Na^+ concentration.
Diluted F100 is used because infants <6 mo cannot handle the renal solute load of full strength F100.

Nutritional rehabilitation formulas

Ingredients	F-75	F-100
Dried skimmed milk (g)	25	80
Sugar (g)	100	50
Vegetable oil (g)	27	60
*Electrolyte/mineral soln (ml)	20	20
Water: make up to (ml)	1000	1000

Nutritional contents of F-75 and F-100

Contents per 100 ml	F-75	F-100
Energy (kcal)	75	100
Protein (g)	0.9	2.9
Lactose (g)	1.3	4.2
K^+ (mmol)	4.0	6.3
Na^+ (mmol)	0.6	1.9
Mg (mmol)	0.43	0.73
Zinc (mg)	2.0	2.3
Copper (mg)	0.25	0.25
% energy from protein	5	12
% energy from fat	32	53
Osmolality (mOsm/1)	413	41

*** commercial mineral and vitamin mix is often available, use one 'red' scoop (6.35mg) per 2000ml of F75 or F100 instead of EMS.**

Vit A deficiency

In resource-poor regions, >80% vit A is derived from dietary carotenoids found in breast milk, dark green vegetables, and yellow and orange fruits. Margarine and meat (esp. liver) are also sources. Vit A deficiency → ↑ morbidity and mortality among children and is a preventable cause of blindness from xerophthalmia. Xerophthalmia is classified as follows:

- *Night blindness* (XN): individual bumps into objects in poor lighting.
- *Conjunctival xerosis* (X1a): dry conjunctiva has glazed appearance.
- *Bitot's spots* (X1b): white foamy spots on conjunctival surface, commonly at the corneoscleral junction on the temporal side.
- *Corneal xerosis* (X2): dry cornea, associated with the onset of visual impairment. Most common in children aged 2–4 y.
- *Corneal ulceration* (X3a): often worse in measles; central corneal ulceration may profoundly affect vision.
- *Keratomalacia* (X3b): severe destruction of the eye with blindness; occurs esp. in severe malnutrition precipitated by measles.
- *Corneal scarring* (XS): follows healing after vit A replacement, often with permanent visual impairment.

Treatment:

For xerophthalmia, severe malnutrition, measles, and pneumonia/ diarrhoea in HIV-infected children:

- Give 3 doses of oral vit A at d 1, d 2, and in wk 3 as per box opposite.
- For xerophthalmia, also give topical antibiotic eye ointment (e.g. tetracycline 1% or chloramphenicol 1%) for 10 d.
- If cornea is involved, close the eye and gently cover with an eye pad.

Pregnancy: Vit A is teratogenic and high doses are contraindicated in pregnancy. However a pregnant woman with xeropthalmia should receive Vit A 5,000–10,000 IU oral od for ≥4 wks.

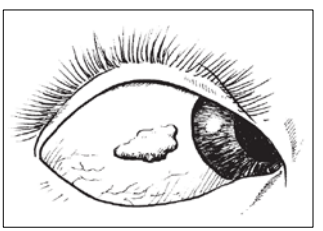

Figure 17.X Bitot's spot, xeropthalmia and conjunctival xerosis.

Source: WHO pocketbook

Curative doses for Vit A deficiency

0–6 mo	50,000 IU*
6–12 mo	100,000 IU*
>1 y (incl. adults)*	200,000 IU*
Vitamin A deficiency in pregnant women	10,000 IU**

* Give 3 doses of oral vit A (d 1, d 2, and in wk 3)

** Give daily dose for ≥ 4wks, but see notes above.

Prevention of Vit A deficiency PUBLIC HEALTH NOTE

- ↑ dietary Vit A: carotenoids in breast milk, spinach, carrots, sweet potatoes, mangos, papaya, milk, eggs, red palm oil, liver, fish liver oils.
- Prophylactic supplementation (200,000 iu) given 2–3 times a year to children aged 6 mo–5 y in endemic deficiency areas.

Vit B_1 (thiamine) deficiency: beriberi

Thiamine is widely available but deficiency may occur when cereals e.g. rice are highly milled. Deficiency may also complicate alcoholism and nitrofurazone therapy for trypanosomiasis. (b p X–X).

Clinical syndromes

- Dry beriberi: peripheral sensory and motor neuropathy: gradual onset of distal limb weakness and wasting with 'glove and stocking' sensory loss; foot drop and calf wasting common. Affected muscles may show oedema and painful contraction when hit. Reflexes and joint position sense are ↓ or lost; ataxia ± incontinence may develop in the later stages. Death occurs due to generalized and diaphragmatic paralysis.
- Wet beriberi: high-output cardiac failure. Typically, peripheries are warm with a bounding pulse, due to peripheral vasodilation In acute, fulminant beriberi, peripheries become cold due to poor cardiac output. Death occurs due to CCF.
- Infantile beriberi: occurs in infants breastfed from a thiamine-deficient mother and is an important cause of infant mortality in parts of Asia. Irritability and oedema typically occur aged 2–3 mo and may be confused with kwashiorkor; progressive heart failure occurs (± convulsions due to CNS involvement) and death is due to cardio-respiratory failure.
- Wernicke's encephalopathy classically complicates thiamine deficiency in chronic alcohol abuse, but may also be precipitated by infections or by administration of carbohydrate (incl. IV dextrose) before thiamine replacement. Clinical features: confusion, ataxia, nystagmus, and ophthalmoplegia due to haemorrhagic degeneration in the midbrain and mamillary bodies. Korsakoff's psychosis may also occur with confusion, confabulation, and loss of short term memory — this is reversible with thiamine replacement, unlike the other clinical features.

Diagnosis is usually clinical. CXR shows cardiolmegaly and pulmonary oedema in cardiac beriberi. Plasma pyruvate and lactate are ↑, red cell transketolase levels are low. Thiamine deficiency may be confirmed *in vitro* by ↑ activation of red cell transketolase after addition of thiamine.

Management:

- *Acute fulminant beriberi:* Thiamine 50–100 mg IV tds until acute symptoms improve followed by 10–25 mg/d oral
- *Chronic beriberi:* Thiamine 10–25 mg/d oral for ≥6 wks. Pain in limbs is relieved rapidly; peripheral neuropathy may take months to years to resolve.

- *Infantile beriberi*: Thiamine 25–50 mg given IV slowly followed by 10 mg IM daily for 1 wk; then 3–5 mg/d orally for 6 wks. Treat mother with thiamine 10 mg/day oral for 7 d, then 3–5 mg/d for 6 wks.

Vit B2 (riboflavin) deficiency

Riboflavin is found in meat, vegetables, milk, and wholemeal flour. Overt deficiency is uncommon. Some drugs e.g. phenothiazines and tricyclic antidepressants interact with riboflavin.

Clinical features: Angular cheilosis/stomatitis, sore red lips, atrophic glossitis. There may be plugging of sebaceous glands, giving a roughened appearance to the skin, and scrotal dermatitis. Anaemia occurs because riboflavin deficiency → poor iron absorption.

Management: Riboflavin up to 30 mg oral od. Usually rapidly cured.

Vit B_3 (niacin) deficiency: pellagra

Niacin and its precursor tryptophan are found in meat, fish, nuts, fruits, and vegetables are good sources of preformed niacin. Deficiency → pellagra, which is common in communities where maize or sorghum are the staple, as bioavailability of niacin in maize is low, and ↑ leucine levels in sorghum ↓ nicotinic acid and tryptophan metabolism; deficiency may be prevented by dietary tryptophan e.g. in beans. Pellagra also occurs in malabsorption, isoniazid therapy, alcoholism; it may contribute to diarrhoea, depression and skin disorders in HIV/AIDS.

Clinical features:
The classical triad is of dermatitis, diarrhoea, and dementia.

- *Skin:* a photosensitive, sunburn-like rash at sun-exposed sites; there may be a collar-like ring around the neck (Casal's necklace). Lesions are sensitive/inflamed, later becoming scaly and desquamate. Atrophic patches of skin remain between the fingers; the nails become brittle and atrophic.
- *Gastrointestinal:* gingival swelling ± bleeding; raw, fissured tongue; dysphagia; villous atrophy and malabsorption; diarrhoea and nausea.
- *Neurological:* insomnia, anxiety, depression, memory loss, photophobia; mania or psychosis (which may be permanent); pyramidal and
  extra-pyramidal signs; frontal reflexes. Confusion can precede death. Peripheral and cranial neuropathies also occur.
- *Eyes:* conjunctival oedema, corneal dystrophy, and lens opacities extending from the periphery to the centre.

Management: treatment with nicotinamide 500 mg daily until complete recovery (at least 3–4 wks).

Prevention: In confirmed outbreaks, consider vit B complex supplements for the entire population as a short-term measure

Vit B_6 (pyridoxine) deficiency

Clinical signs of deficiency are rare, except as peripheral neuropathy during isoniazid therapy, and pyridoxine antagonists e.g. pyrazinamide and cycloserine may → sideroblastic anaemia. Dietary sources include. meat and vegetables.

Management: Pyridoxine 50–150 mg/d oral in divided doses are widely used, but little evidence for efficacy. Doses up to 400 mg/d may be partially effective in idiopathic and hereditary sideroblastic anaemia. Give pyridoxine 10 mg/d during isoniazid therapy and to malnourished alcoholics.

Toxicity: Peripheral neuropathy is reported following prolonged high-dose pyridoxine. Improvement is limited even after stopping treatment.

Vit B_{12} deficiency

Vit B_{12} is available in animal products incl. liver, fish, meat, eggs, and dairy products, but not in vegetables. Absorption depends on intrinsic factor from the stomach binding vit B_{12} to facilitate uptake in the terminal ileum. Deficiency may be due to poor dietary intake (e.g. vegans), atrophic gastritis (pernicious anaemia), previous gastrectomy, and terminal ileal disease.

Clinical features

- *General:* Angular cheilosis, glossitis; hyperpigmentation of the hands and feet is noted in some populations.
- *Macrocytic anaemia:* see b p 464.
- *Subacute combined degeration of the cord:* Dorsal column and corticospinal tract degeneration → sensory and both upper and lower motor neuron signs, classically with extensor plantars but absent knee and ankle reflexes, ± ataxia due to ↓ proprioception; pain and temperature sensation are preserved as spinothalamic tracts are not involved. May be precipitated by administration of high doses of folate to patients with combined B_{12} and folate deficiency.
- *Other neurological sequelae:* peripheral neuropathy, optic atrophy, dementia, neuropsychiatric symptoms, neurodevelopmental delay.

Diagnosis: Low serum B12; macrocytosis; anaemia; low WBC and platelets (b p 464). Tests for pernicious anaemia: parietal cell/intrinsic factor Antibodies, Schilling test.

Management: Hydroxycobalamin 1 mg IM 3x/wk for 2 wks to replenish body stores, then 1 mg IM every 3 mos (often needed for life); if neurological involvement, give 1 mg on alternate days until no further improvement, then 1 mg every 2 mos.

Folate deficiency

Leafy green vegetables (e.g. spinach), fruits (like citrus fruits and juices), and dried beans and peas are all natural sources of folate. Folate is heat labile and water soluble so is lost in prolonged cooking or boiling. Deficiency occurs in malabsorption, in pregnancy or haemolysis, and in patients on anti-folate drugs e.g. methotrexate or trimethoprim.

Clinical features: Blood changes are similar to vit B_{12} deficiency (b p 464) but without neurological sequelae. Deficiency in pregnancy ↑ risk of neural tube defects; intrauterine growth retardation, premature delivery, low birth weight.

Diagnosis: ↓ RBC folate; macrocytic anaemia (b p 464).

Management: Treat deficiency with folic acid 5 mg oral od. Folate is increasingly being added to flour as part of national government policies.

Vit C deficiency: scurvy

Vit C (ascorbic acid) is essential to collagen formation, i iron absorption, maintaining healthy epithelial tissues, e.g. mouth and skin, and promoting wound healing. Found in fresh citrus fruit and potatoes but easily destroyed by overcooking. Deficiency occurs when fruit and vegetables are scarce, in the elderly, and in young children (who have ↑ requirements).

Clinical features:

- *General:* weight loss, stiffness, weakness, swollen painful large joints.
- *Skin:* dry skin, hyperkeratosis of hair follicles, 'corkscrew hairs', bruising, perifolicular petechial haemorrhages, poor wound healing.
- *Mouth:* gingivitis, bleeding gums, dental caries, loss of teeth.
- *Anaemia:* microcytic anaemia due to iron deficiency, and/or megaloblastic anaemia as vit C is required for folate metabolism.

Treatment: Oral ascorbic acid, in divided doses per day for 2 wks (infants <1mo 50 mg/d; 1 m–4 y 125–250 mg/d, 4–12 y 250–500 mg/d, adults 500 mg/d).

Prevention: Avoid overcooking vegetables; eat citrus fruit, mangos and guavas. If necessary, supplement with tablets (children and adults 25–75 mg/d). Avoid artificial feeds without fortified vit C.

Scurvy in infants **PAEDIATRIC NOTE**

Infantile scurvy typically presents at 6–12 mo in premature or artificially fed infants. Erupting teeth → bleeding of the gums. Subperiosteal haemorrhages → limb pain and swelling, esp. in the long bones e.g. distal femur and proximal tibia; costochondral beading

may also be palpable (scorbutic rosary). Occasionally, there is bloody diarrhoea. There may be a microcytic and/or megaloblastic anaemia. Plain X-rays of the long bones show epiphyseal changes and ground glass appearance of the shafts.

Vit D deficiency: rickets/osteomalacia

Vit D regulates calcium homeostasis by controlling intestinal absorption and renal excretion of Ca^{++}, and mobilizing Ca^{++} from bone. Vit D is also important in cell signalling, gene expression, platelet aggregation, and host immunity (e.g. TB). The best food source of vit D is oily fish, though most Vit D_3 is formed in the skin by UV light. Deficiency may be due to dietary insufficiency, malabsorption or lack of UV exposure, which is exacerbated by skin-covering and lack of outdoor play. It may also be due to liver disease, renal failure, or anticonvulsant therapy (↑ Vit D metabolism s to enzyme induction). Deficiency → rickets in children and osteomalacia in adults.

Clinical features

- Rickets is due to disordered bone mineralization at the growth plates of growing children, usually <2 y. *Features*: irritability, hypotonia, painful wrists, and tender legs (which may be bowed once the child starts standing); swollen costo-chondral junctions ('rachitic rosary'), pigeon chest, indrawing of the lower ribs (Harrison sulcus), spinal deformities, bossing of the skull and craniotabes. Hypocalcaemia may → tetany and jaw, tongue or laryngeal spasm. Neurodevelopmental delay also occurs. The presentation therefore depends on age.
- Osteomalacia occurs in adults, often in women or in the elderly. It presents with acheing muscles and bones (pelvis, ribs, femora), pathological fractures, proximal myopathy (waddling gait).
- Vit D deficiency is associated with susceptibility to TB and pneumonia.

Diagnosis

Is clinical, aided by the following investigations:

- *X-rays:* Cupping and fraying of the metaphyses with widening of the epiphyses in rickets; osteopenia, Looser's zones (partial fractures without bony displacement e.g. of lateral scapular border, femur, or pelvis), biconcave deformity of the vertebrae.
- *Bloods:* low plasma 25-hydroxy vit D. Only in severe deficiency is there ↓ serum Ca^{++}, ↓PO_4^-, ↑ ALP.
- *Bone scanning* shows characteristic changes.

Management: Oral ergocalciferol (vit D_2): the RDA for adults is 400–800 units. Deficiency should be corrected with a few days of high dose replacement: adults 40,000 units, children <6mo 3,000 units; 6 mo–12 y 6,000 units; 12–18 y 10,000 units daily. A single IM dose of 150,000 – 300,000 iu of vit D_2 in oil will protect for >6 mo. Unless there is plenty of calcium in the diet or water, give calcium 500 mg

oral od for the first 15 days of treatment. Specialist regimens are required if vit D deficiency is due to renal disease or malabsorption.

Calcium deficiency and rickets **PAEDIATRIC NOTE**

Rickets may occur as a result of calcium deficiency and/or of Vit D deficiency, e.g. in African children fed a maize diet low in calcium.
Vit D deficiency may occur in infancy, due to maternal deficiency and low sunlight exposure.
Calcium deficiency usually occurs later e.g. bow legs.

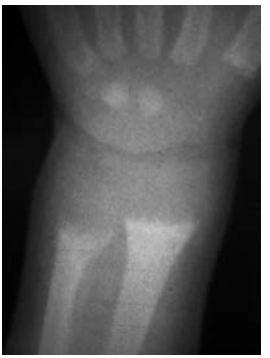

Figure 17.X: Wrist x-ray of a 15mo child with rickets showing cupping and fraying of the distal ends of the radius and ulna.

Vit E (alpha-tocopherol) deficiency

Vit E deficiency develops in patients with fat malabsorption (e.g. cholestatic liver disease) and in patients with congenital abetalipoproteinaemia. Premature infants often have inadequate vit E stores l haemolytic anaemia.

Clinical features: Haemolytic anaemia, ataxia, and peripheral neuropathy.

Diagnosis: Plasma vit E (alpha-tocopherol) level.

Treatment: Few patients need treatment. Treat deficiency with vit E in neonates (10 mg/kg oral od) and children (1mo to 18y 2-10mg/kg oral od, up to 20mg/kg has been used). Children with abetalipoproteinaemia (neonates 100 mg/kg oral od, child 1 mo-18 y, 50-100 mg/kg oral od), Children with cholestasis or severe liver disease treat (neonate 10 mg/kg oral od; children 1 mo-12 y, initially 100 mg oral od adjusted according to response; up to 200 mg/kg od may be required; child 12-18 y, initially 200 mg oral od, adjusted according to response; up to 200 mg/kg oral od may be required.

Vit K deficiency

Vit K is essential for production of clotting factors II, VII, IX, and X, proteins C and S, and for bone growth. It is found in leafy green vegetables and is produced by intestinal bacteria. Deficiency occurs in poorly fed neonates and adults with malabsorption, and → bleeding tendency with ↑ prothrombin time. Neonates have low body stores, esp. those born prematurely, and deficiency causes 'haemorrhagic disease of the newborn.' This can be prevented with vitamin K (see b p493 for details).

Treatment: For Vit K deficiency associated haemorrhage, give vit K (1mo-18y: 250-300 micrograms/kg IV stat, up to a maximum of 10mg; in adults 5-10mg IV) Dietary advice suffices in most non-bleeding cases.

Iodine deficiency

Iodine is essential for thyroid hormone synthesis, brain development and function. Deficiency is usually due to low levels in soil and water, esp. in mountainous areas (e.g. Nepal and Bolivia) and low-lying areas where flooding has washed iodine out of the soil (e.g. Bangladesh). Limited iodine availability can be worsened by eating brassicas, cassava, or soya beans. Deficiency → goitre ± hypothyroidism; it is the commonest cause of preventable mental retardation ('cretinism') worldwide.

Clinical features

- *Goitre:* ↑ thyroid-stimulating hormone (TSH) from the pituitary l thyroid enlargement. Large goitres may → dysphagia and hoarseness (recurrent laryngeal nerve compression). Patients may be euthyroid (most commonly) or hypothyroid. Not associated with ↑ risk of malignancy.
- *Endemic (neurologic) cretinism:* mental retardation, speech and hearing deficits, strabismus, spastic diplegia, and a characteristic apathetic facies with thickened features. May occur as a result of maternal hypothyroidism in any population, even in the absence of iodine deficiency.
- *Hypothyroidism:* clinical features are described on b p 514. Severe hypothyroidism → 'myxoedematous cretinism', with short stature, ataxia, and mental retardation without hearing deficit.

Diagnosis

Is clinical, supported by ↑ TSH ± $\downarrow T_4$; urinary iodine measures of dietary iodine intake.

Treatment

- Iodized oil as a single dose repeated after 1–2y (see box).
- Lugol's iodine (often kept for sterilization) may also be given as 1 drop every 30 d, or 1 daily teaspoon of a solution containing 1 drop of Lugol's iodine in 30 ml of water.
- Surgery may be required for massive goitre.

Prevention of iodine deficiency **PUBLIC HEALTH NOTE**

Visible goitre in >10% of the population indicates severe iodine deficiency and mass prevention should be undertaken with IM injections of iodized oil or oral iodine. Iodine should also be given to pregnant women in endemic areas to prevent congenital hypothyroidism.

- Iodized salt: Satisfactory iodization of salt can be tested using simple
  colour change kits based on starch/iodine interaction colours.
- Iodized poppy seed oil (IPSO) can be used in endemic areas where salt is not iodized: women should take IPSO 400 mg as a single dose,

preferably before conception; children should receive 100mg <12 mo, 200 mg aged 1–5 y, 400mg aged 5–18 y. In areas where intestinal parasites are endemic, give albendazole to ensure absorption. The dose lasts for up to 2 y.
- Over-replacement in endemic areas may → thyrotoxicosis.

Other micronutrients

Zinc

Zinc has antioxidant properties and is essential to several proteins and enzymes, incl. those regulating gene expression. Body stores are minimal so deficiency occurs quickly, esp. in catabolic states or if intestinal losses are high. Zn is found in meat and fish; bioavailability from cereals is often poor because phytate binds Zn. Deficiency occurs in severe malnutrition (esp. oedematous malnutrition) and low birth weight infants.

Clinical features: Failure to thrive, recurrent infections, persistent diarrhoea, scaly lesions (probably due to local *Candida*) on the feet and buttocks, stunting, developmental delay. The classical rash of acrodermatitis enteropathica is rare, usually due to a congenital disorder of Zn malabsorption. Plasma Zn is often ↓ in individuals but single measurements are unreliable as they ↓ in acute infection.

Treatment: Zinc 10 mg/day ↓ frequency/severity of respiratory infections and diarrhoeal disease, including in HIV + individuals, and improves wound healing. A 2-week course of zinc given for diarrhoea ↓ mortality in the following 6 months.

Copper

Cu is important for several enzymes with antioxidant properties and for development of collagen. Cu is widely available in shellfish, liver, kidney, nuts, and wholegrain cereals. Deficiency is uncommon and → osteoporosis and leukopenia with ↑ risk of infection. Cu deficiency may be precipitated by high Zn doses → impaired Cu absorption. Menke's disease is a rare cause due to defective Cu metabolism. Dietary excess (± genetic predisposition) is implicated in Indian childhood cirrhosis (b p 303).

Treatment: Copper should be included in the electrolyte/mineral mix for treatment of severe malnutrition.

Selenium

Several enzymatic processes require Se. Dietary sources include cereals, meat, and nuts. Deficiency occurs where cereals are grown in low Se soils → ↓ antioxidant activity. This may → to coronary artery disease. . Se stimulates immunity and has been advised in nutritional support for HIV.

Clinical features: Se deficiency → cardiomyopathy in China where soils are deficient in Se.

Treatment & prevention: Se should be included in the electrolyte/mineral mix for treatment of severe malnutrition (see above). Fertilizers help mitigate the effect of low Se levels in the soil.

Iron

Iron deficiency is a common cause of anaemia (see b p 460).

Fluoride

Fluoride is essential for mineralization of bones and teeth, and is present in the majority of foods and drinking water. Deficiency contributes to dental caries. Excess dietary fluoride may occur where the drinking water is very high in fluoride (e.g. Rift Valley in E Africa, the Punjab) causing clinical fluorosis.

Clinical features of deficiency include dental caries and softening of long bones with deformity. Conversely, fluorosis is characterized by excess fluoride deposition in teeth and bones, with chalky discolouration of teeth enamel, spinal rigidity, restricted joint movement, ectopic mineralization of tendons, ligaments, and occasionally muscles, ↑ bone density.

Prevention: Add fluoride to drinking water at source where fluoride levels low. Where fluorosis is endemic and fluoride levels in water are high, advise on alternative drinking water sources.

Obesity

The terms overweight and obese are interchangeable. People become obese because they take in more calories than they consume in metabolism and work. There is ↑ evidence for differences between individuals in appetite, fat metabolism, and metabolic responses to a meal. There is a global epidemic of obesity.. Changes in dietary intake in recent years have been associated with ↓ physical activity. Obesity tended in the past to be a disease of the prosperous and urbanization, but the present epidemic of obesity affects the poor who buy cheaper, high-energy foods. Obesity may also result from programming occurring when intrauterine malnutrition, or malnutrition in the first 2 y of life, has been present. Obesity ↑ risk of coronary heart disease, stroke, hypertension, type II diabetes, gallstones and other digestive disorders, back problems, arthritis of the knees and hips, accidents, fractures, and fatigue.

Diagnosis: BMI >25 kg/m^2 indicates probable obesity; >30 definite obesity. Children should be assessed with charts and table of W/H or BMI. Children are obese if they are >+2 Z scores (>97th centile) for W/H (see b p 624).

Management: weight loss is difficult:
- Eat foods containing more fibre and less fat or sugar. Instead of high-energy snacks, eat fruit or maize cobs. Avoid sweets, chips, crisps, and cakes; ↓ alcohol.
- Exercise for >20 mins per day at a level sufficient to raise the pulse and respiratory rates.
- Be realistic and offer encouragement, not scorn.
- Advise stopping smoking to ↓ cardiovascular risk.
- Avoid drugs which suppress appetite and metabolism — the evidence for these is lacking.