There is a 90% probability of complete remission, but those presenting over the age of 10 years are more likely to have chronic ITP. ITP that persists for 6 months is defined as chronic. Children with chronic ITP are more likely to have an underlying cause (e.g. autoimmune disease).

Bleeding manifestations include petechiae, purpura, epistaxes, haematuria, gastrointestinal haemorrhage and (rarely) intracerebral haemorrhage. The child has no hepatosplenomegaly and is usually well. Other causes of thrombocytopenia must be excluded. If there is any doubt, a bone-marrow aspirate will show normal haematopoiesis with increased numbers of megakaryocytes in ITP.

Management
- Treatment is based on symptoms, not platelet count, and many patients require no treatment.
- Petechiae on the head and neck, and gastrointestinal and oral bleeding, are indicators for prednisolone (1–2 mg/kg/day after food in two divided doses for no more than 14 days or 4 mg/kg for no more than 4 days; reduce over 5 days and stop irrespective of the platelet count if the patient is asymptomatic). Prednisolone does not alter the course of the disease. The time to remission is very variable.
- Tranexamic acid can be useful in the treatment of mucosal bleeding. Give 10 mg/kg IV slowly over 10 minutes in children 6–18 years (maximum 1 gram) followed by 25 milligrams/kg orally (maximum 1.5 gram) three times daily for 2–8 days.
- Hormonal treatment can benefit girls with menorrhagia. In addition Tranexamic acid 1 gram orally 3 times daily for up to 4 days can help (initiate when menstruation starts).
- Chronic ITP with serious bleeding into the gastrointestinal tract or brain may require splenectomy. However, in resource-limited countries there is a high risk of infection following splenectomy, and long-term penicillin prophylaxis and pneumococcal vaccination are required.

Reference
Grainger JD, Rees JL, Reeves M et al. (2012) Changing trends in the UK management of childhood ITP. Archives of Disease in Childhood, 97, 8–11.

5.12 Gastrointestinal disorders

5.12.A Acute diarrhoea

Introduction
Diarrhoeal diseases are a leading cause of childhood morbidity and mortality in resource-limited countries. In 2001, an estimated 1.5 million children under 5 years of age died from diarrhoea, 80% of them in the first 2 years of life. Around 50% of these deaths are due to watery diarrhoea and occur either because of lack of access to oral rehydration solution (ORS) or because of incorrect case management. About one-third of deaths are caused by persistent diarrhoea and the remainder (approximately 15%) are caused by dysentery.

This section is primarily aimed at the management of the infant and child under 5 years as they are the most seriously affected. There are particular problems in managing children with severe co-morbidities: these include significant malnutrition and anaemia (Hb below 6 G/dL, see Sections 5.10.B and 5.11.A). In these children, assessment is more difficult and there is likely to be an abnormal response to a fluid load because of poor cardiac function. Modifications to the management plans for these children largely involve slower shock management and rehydration, the careful use of blood transfusion and diuretics and very frequent re-assessment.

ORS has been a simple and effective solution, reducing morbidity and mortality in diarrhoeal illness. The new low-osmolarity ORS reduces by 33% the need for supplemental

Important issues
- Shock management, rehydration therapy and continued feeding are key strategies.
- Antibiotics are not given routinely, but they are indicated in bloody diarrhoea (probable Shigella infection) and suspected cholera.
- Antidiarrhoeal drugs and anti-emetics should never be given and can be dangerous in children.
- Zinc supplementation speeds recovery and helps to prevent further episodes.

<table>
<thead>
<tr>
<th>BOX 5.12.A.1 Minimum standards</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduced-osmolarity ORS.</td>
</tr>
<tr>
<td>ReSoMal for children with severe malnutrition.</td>
</tr>
<tr>
<td>IV fluids: Hartmann’s or Ringer-lactate solution with glucose 5% or 10% to prevent hypoglycaemia.</td>
</tr>
<tr>
<td>Potassium: oral and IV.</td>
</tr>
<tr>
<td>ABC resuscitation for shock.</td>
</tr>
<tr>
<td>Antibiotics: co-trimoxazole, amoxicillin, nalidixic acid, ciprofloxacin, cefotaxime, chloramphenicol, erythromycin, metronidazole, tetracycline, vancomycin, doxycycline.</td>
</tr>
</tbody>
</table>
IV fluid therapy after initial rehydration compared with the previous standard WHO ORS solution. The new ORS also reduces the incidence of vomiting by 30% and stool volume by 20%.

In addition, zinc supplementation has been shown to significantly reduce the severity and duration of diarrhoea.

Definition
Diarrhoea is the passage of loose or watery stools, usually at least three times in a 24-hour period. However, it is the consistency of the stools rather than the number that is most important. Mothers usually know when their children have diarrhoea, and may provide useful working definitions in local situations. The volume of fluid lost through the stools in 24 hours can range from 5 mL/kg (near normal) to 200 mL/kg, or more. Dehydration occurs when these losses are not replaced adequately and a deficit of water and electrolytes develops. The concentrations and amounts of electrolytes lost also vary. The total body sodium deficit and electrolytes develops. The concentrations and amounts of electrolytes lost also vary. The total body sodium deficit is usually about 70–110 millimoles/litre of water deficit. Potassium and chloride losses are in a similar range.

The most common causes of diarrhoea are rotavirus, enterotoxigenic E. coli (ETEC) and, during epidemics, Vibrio cholerae O1 or O139.

Classification of diarrhoea
- Acute watery diarrhoea (including cholera): this lasts from several hours to days. The main danger is dehydration, and malnutrition also occurs if feeding is not continued. If there is a current epidemic, cholera is likely and causes severe dehydration with a positive stool culture for Vibrio cholerae O1 or O139.
- Acute bloody diarrhoea, or dysentery (blood is mixed in with stool): the main dangers are intestinal damage, sepsis and malnutrition. Other complications, including dehydration, may also occur.
- Persistent diarrhoea: this is defined as passage of three or more loose watery stools in a 24-hour period, which lasts for 14 days or longer. The main danger is malnutrition and serious non-intestinal infection; dehydration may also occur (see Section 5.12.B).
- Diarrhoea with severe malnutrition (marasmus or kwashiorkor): the main dangers are severe systemic infection, dehydration, heart failure and vitamin and mineral deficiency (see Section 5.10.A).
- Diarrhoea associated with a recent course of broad-spectrum oral antibiotics.

Assessment of the child with diarrhoea
- Fever, vomiting and loose stools are the common symptoms of acute gastroenteritis.
- If possible, rule out other serious illness (e.g. meningitis, malaria, bacterial sepsis).
- Assess for degree of dehydration, bloody diarrhoea, persistent diarrhoea, malnutrition and serious non-intestinal infections.

History
Specific points to enquire about in the history include the following:
- duration of diarrhoea
- presence of blood in the stool
- local knowledge or reports of a cholera epidemic
- recent use of antibiotics
- the presence of fever, cough or other important problems (e.g. convulsions, measles)
- usual feeding practices
- the type and amount of fluids (including breast milk) and food taken during the illness
- drugs or other remedies taken
- immunisation history.

Physical examination
First assess the patient for shock and treat this urgently as a priority if it is present. Children with shock will have reduced consciousness, a high and increasing heart rate, weak pulse, poor skin circulation time with prolonged capillary refill time (> 3 seconds), and low or even unmeasurable blood pressure.

Children with shock require immediate resuscitation (ABC), including high concentrations of oxygen (if available) and an IV bolus of 10–20 mL/kg of either Ringer-lactate or Hartmann’s solution given as rapidly as possible (see Section 5.5.B). If IV access is not possible (often the veins are collapsed), consider the intra-osseous route (see Section 8.4.B). If shock is not relieved by 20 mL/kg, give another bolus of 10–20 mL/kg, but watch very carefully for fluid overload and in particular pulmonary oedema (this is most likely if the patient is also severely anaemic and will be shown by increasing breathlessness, crepitations may be heard).

The examination includes measurement of vital signs together with clinical correlation. The degree of dehydration is graded according to signs and symptoms that reflect the amount of fluid lost (see Table 5.12.A.2). Infants with acute diarrhoea are more apt to dehydrate than are older children, because they have a higher body surface area to weight ratio, have a higher metabolic rate, and are dependent on others for fluid. Although the most accurate assessment of fluid status is acute weight change, the patient’s premorbid weight is often not known.

In severe dehydration, prolonged skin retraction time and decreased perfusion are more reliably predictive of dehydration than a sunken fontanelle or the absence of tears. A good correlation has been reported between capillary refill time and fluid deficit. However, fever, ambient temperature and age can affect capillary refill time as well. In severe dehydration, shock and death soon follow if rehydration is not started quickly.

Children with some dehydration or severe dehydration should be weighed without clothing when estimating their fluid requirements. If weighing is not possible, the child’s age may be used to estimate their weight:
- Weight = (age in years + 4) × 2 for children less than 10 years old.
- For an infant up to 1 year old, birth weight doubles by 5 months and triples by 1 year.

Treatment should never be delayed because facilities for weighing are not rapidly available.

In addition:
- Look for an abdominal mass or abdominal distension.
- In an infant less than 1 week old, diarrhoea is sometimes a sign of neonatal sepsis (see Section 3.4). In an infant, blood in the stool may be due to an intussusception (see Section 5.19).
Remember other diagnoses, including typhoid, antibiotic-associated colitis and (rarely) inflammatory bowel disease (see Section 5.12.D).

**Investigations**

Laboratory investigations are rarely needed at the outset. Serum electrolytes, especially sodium or potassium concentrations, are useful in severe dehydration and for monitoring progress, if available. Stool cultures should be undertaken if at all possible in dysentery (bloody diarrhoea), but are not needed to initiate treatment in the usual case of acute watery diarrhoea. Stool microscopy can be useful for diagnosing Giardia lamblia, Cryptosporidium and amoebic dysentery.

**Principles of case management**

There are five essential elements of the management of all children with diarrhoea:

- **Resuscitation from shock, if present:** Give IV boluses of Hartmann’s solution or Ringer-lactate solution. This needs to be done rapidly (caution is required in malnutrition and anaemia; see Section 5.10.B). Improvement in conscious level is a good indicator of response to circulatory shock treatment.
- **Rehydration therapy:** this should be done more slowly, so as not to cause rapid metabolic change.
- **Maintenance therapy:** this is to replicate the normal fluid needs and any ongoing extra losses.
- **Zinc supplementation.**
- **Continued feeding.**

**Calculating fluid requirements**

WHO Plans A to C for gastroenteritis in children (see Appendix to this section) include estimates of total fluid requirements, and assume that most children will be drinking by 4 hours into treatment and thus able to ‘self-regulate’. For patients for whom this is not the case, fluid management can be undertaken using the following guidelines.

**Estimating fluid requirements**

The amount of fluid that the child needs over a 24-hour period needs to be calculated. It is the sum of:

- estimated fluid deficit + maintenance requirements + ongoing losses.

**Deficit**

If an accurate recent pre-illness weight is available, subtract the current weight to estimate lost fluid (1 kg = 1 litre of fluid).

For example, a child who weighed 9.2 kg is seen with diarrhoea and weighs 8.3 kg:

estimated fluid loss is (9.2 – 8.3) kg = 0.9 kg = 900 mL deficit, i.e. 10% dehydrated.

If no recent weight is available, or the recorded weight is considered to be unreliable, assess the degree of dehydration as described in Table 5.12.A.2.

Weigh the child (or estimate their weight from their age as follows: weight (kg) = 2 × [age (years) + 4]) if over one year.

Then use the following formula: percentage dehydration × weight (kg) × 10 = deficit (in mL).

For example, a child whose weight is estimated to be 10 kg is 10% dehydrated.

Their estimated fluid loss is 10 × 10 × 10 = 1000 mL (40 mL/hour if replaced over 24 hours).

**Maintenance**

**TABLE 5.12.A.1 Estimated maintenance fluid requirements based on body weight for a child**

<table>
<thead>
<tr>
<th>Body weight</th>
<th>Fluid needed per day</th>
<th>Fluid needed per hour</th>
</tr>
</thead>
<tbody>
<tr>
<td>First 10 kg of body weight</td>
<td>100 mL/kg</td>
<td>4 mL/kg</td>
</tr>
<tr>
<td>Second 10 kg of body weight</td>
<td>50 mL/kg</td>
<td>2 mL/kg</td>
</tr>
<tr>
<td>Subsequent kg</td>
<td>20 mL/kg</td>
<td>1 mL/kg</td>
</tr>
</tbody>
</table>

**Ongoing losses**

**For each diarrhoeal stool:**

- < 2 years of age: give 50–100 mL or 10 mL/kg
- ≥ 2 years of age: give 100–200 mL or a cup or small glass if drinking or tolerating NG fluid.

**For each vomit:** use 2 mL/kg ORS, and give small frequent volumes (e.g. 5 mL/minute in a child) via a spoon, syringe or cup. Gradually increase the amount given and closely supervise this.

For nasogastric tube aspirates: replace volume for volume with either ORS or Ringer-lactate solution with 5% or 10% glucose or Hartmann’s solution with 5% or 10% glucose.

**Signs of over-hydration**

- Cædematous (puffy) eyelids.
- Heart failure (especially in severe malnutrition), chronic malnutrition or protein-losing enteropathy: look for tachycardia, tachypnoea, crepitations at the lung bases, hepatomegaly or gallop rhythm (see Section 5.4.B).
- A chest X-ray may be helpful in showing pulmonary plethora or oedema.

Stop giving ORS, but give breast milk or plain water, and food.

Do not give a diuretic unless there is pulmonary oedema (lung crepitations), in which case give furosemide 1 mg/kg IV.

**Treatment phases in dehydration with shock**

In the shock phase, the circulating volume must be improved sufficiently to perfuse vital organs, this will be identified by an improvement in conscious level, falling heart rate and stronger pulse volume.

- In the rehydration phase, the fluid deficit should be replaced and clinical hydration achieved.
- In the maintenance phase, adequate dietary and fluid intake should be maintained.
- In all phases, excess fluid losses must be replaced continuously.

**Oliguria**

If the child responds poorly to ORS, test for oliguria (urine output < 1 mL/kg/hour). If present, give 10–20 mL/kg (or 1 mL/kg per hour) Ringer’s lactate or Hartmann’s solution intravenously. Stop if there is an improvement in urine output or other clinical signs. Do not give additional fluids if diuresis ceases. Increasing urine output by 5–10 mL/kg/hour is adequate.
Rehydration therapy is based on degree of dehydration. Reduced-osmolarity ORS is as effective as ORS because it did not reduce stool output or the duration of the diarrhoea. Without glucose, ORS solution would be ineffective in the small intestine. This is true irrespective of the cause of the diarrhoea. Absorbed glucose promotes the absorption of sodium and water and basal deficit acidosis. Glucose is essential because, as it is absorbed, it promotes the absorption of sodium and water in the small intestine. This is true irrespective of the cause of the diarrhoea. Without glucose, ORS solution would be ineffective.

Healthcare workers and mothers criticised standard ORS for preventing and treating diarrhoea, but it also reduces stool output/volume by 25%, reduces vomiting by almost 30%, and reduces the need for supplemental IV rehydration by 33%. This means that there is less need for hospital care, less disruption of breastfeeding, less use of needles and, where IV treatment is not available, less risk of dying from acute diarrhoea.

It is as effective as standard ORS in the treatment of cholera in adults, but may produce transient hyponatraemia. In children it appears to be as effective as standard ORS in cholera, but careful observations for hyponatraemia should be undertaken if possible.

Use ReSoMal instead of low-osmolarity ORS in severe acute dehydration and when to return

<table>
<thead>
<tr>
<th>Degree of dehydration with diarrhoea</th>
<th>Symptoms and signs present</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>No dehydration</td>
<td>None Increased thirst</td>
<td>Treat at home with extra fluids. WHO Treatment Plan A (see below)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Breastfeeding or standard diet must continue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Warn mother about danger signs of some or severe dehydration and when to return</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Zinc supplements</td>
</tr>
<tr>
<td>Some dehydration (5–9% fluid deficit)</td>
<td></td>
<td>Treat with WHO Treatment Plan B in hospital for at least 24 hours (if feasible)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Give ORS or ReSoMal if there is malnutrition</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Breastfeeding or standard feeding to continue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Zinc supplements</td>
</tr>
<tr>
<td>Severe dehydration (10% or greater)</td>
<td>Two or more of the following signs:</td>
<td>WHO Treatment Plan C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rapid IV rehydration, giving ORS while IV cannula is put in place</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Test for and treat any hypoglycaemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Breastfeeding or standard feeding as soon as possible</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Zinc supplements</td>
</tr>
<tr>
<td>Shock</td>
<td>As above: High and increasing heart rate; weak pulse volume</td>
<td>Urgent IV or intra-osseous access</td>
</tr>
<tr>
<td></td>
<td>Low or even unmeasurable blood pressure</td>
<td>Urgent IV/intra-osseous fluid bolus of 10 mL/kg</td>
</tr>
<tr>
<td></td>
<td>Very reduced conscious level or coma</td>
<td>Ringer-lactate or Hartmann’s solution</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Repeat 10 mL/kg boluses if remains shocked, up to a total of 40 mL/kg, then beware of fluid overload</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Then rehydrate more slowly</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Use NG or oral ORS/breast milk as soon as tolerated</td>
</tr>
</tbody>
</table>

Rehydration therapy is based on degree of dehydration.

**Treatment with low-osmolarity ORS**

The formula for standard ORS and the latest low-osmolarity ORS recommended by the WHO and UNICEF is given in Table 5.12.A.3. The quantities shown are for preparation of 1 litre of ORS, by adding one sachet of oral rehydration salts to 1 litre of clean water.

When prepared and given correctly, ORS provides sufficient water and electrolytes to correct the deficits associated with acute diarrhoea. Potassium is provided to replace the large potassium losses associated with acute diarrhoea, especially in infants, thus preventing serious hypokalaemia. Citrate (or bicarbonate) is provided to prevent or correct base deficit acidosis. Glucose is essential because, as it is absorbed, it promotes the absorption of sodium and water in the small intestine. This is true irrespective of the cause of the diarrhoea. Without glucose, ORS solution would be ineffective.

Healthcare workers and mothers criticised standard ORS because it did not reduce stool output or the duration of diarrhoea. Reduced-osmolarity ORS is as effective as standard ORS for preventing and treating diarrhoea, but it also reduces stool output/volume by 25%, reduces vomiting by almost 30%, and reduces the need for supplemental IV rehydration by 33%. This means that there is less need for hospital care, less disruption of breastfeeding, less use of needles and, where IV treatment is not available, less risk of dying from acute diarrhoea.

A child’s fluid deficit can be estimated as follows:

- Mild or no signs of dehydration: < 5% fluid deficit; < 50 mL/kg.
- Some dehydration: 5–10% fluid deficit; 50–100 mL/kg.
- Severe dehydration: > 10% fluid deficit; > 100 mL/kg.

**Table 5.12.A.2 Estimated degrees of dehydration with symptoms, signs and treatment**

<table>
<thead>
<tr>
<th>Degree of dehydration with diarrhoea</th>
<th>Symptoms and signs present</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>No dehydration</td>
<td>None Increased thirst</td>
<td>Treat at home with extra fluids. WHO Treatment Plan A (see below)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Breastfeeding or standard diet must continue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Warn mother about danger signs of some or severe dehydration and when to return</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Zinc supplements</td>
</tr>
<tr>
<td>Some dehydration (5–9% fluid deficit)</td>
<td></td>
<td>Treat with WHO Treatment Plan B in hospital for at least 24 hours (if feasible)</td>
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<tr>
<td></td>
<td></td>
<td>Give ORS or ReSoMal if there is malnutrition</td>
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<td></td>
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<td>Breastfeeding or standard feeding to continue</td>
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<tr>
<td></td>
<td></td>
<td>Zinc supplements</td>
</tr>
<tr>
<td>Severe dehydration (10% or greater)</td>
<td>Two or more of the following signs:</td>
<td>WHO Treatment Plan C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rapid IV rehydration, giving ORS while IV cannula is put in place</td>
</tr>
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<td>Test for and treat any hypoglycaemia</td>
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<tr>
<td></td>
<td>Low or even unmeasurable blood pressure</td>
<td>Urgent IV/intra-osseous fluid bolus of 10 mL/kg</td>
</tr>
<tr>
<td></td>
<td>Very reduced conscious level or coma</td>
<td>Ringer-lactate or Hartmann’s solution</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Repeat 10 mL/kg boluses if remains shocked, up to a total of 40 mL/kg, then beware of fluid overload</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Then rehydrate more slowly</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Use NG or oral ORS/breast milk as soon as tolerated</td>
</tr>
</tbody>
</table>

**Table 5.12.A.3 Composition by weight of WHO/UNICEF oral rehydration salts to be dissolved in boiled water to produce 1 litre**

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Original standard ORS (grams/litre clean water)</th>
<th>New and recommended low-osmolarity ORS (grams/litre clean water)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium chloride</td>
<td>3.5</td>
<td>2.6</td>
</tr>
<tr>
<td>Trisodium citrate dihydrate</td>
<td>2.9</td>
<td>2.9</td>
</tr>
<tr>
<td>Potassium chloride</td>
<td>1.5</td>
<td>1.5</td>
</tr>
<tr>
<td>Glucose anhydrous</td>
<td>20</td>
<td>13.5</td>
</tr>
</tbody>
</table>
Instead of citrate.

If using bicarbonate ORS there are 30 mmol/litre of bicarbonate (276–295 mOsm/litre).

Hyperosmolar with respect to plasma osmolality (normal = 285–295 mOsm/litre). 

**TABLE 5.12.A.4** Resulting molar concentration of components of standard and reduced-osmolarity WHO oral rehydration solutions

<table>
<thead>
<tr>
<th>ORS</th>
<th>Standard osmolarity (mEq/litre)</th>
<th>Reduced osmolarity (mEq/litre)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>111</td>
<td>75</td>
</tr>
<tr>
<td>Sodium</td>
<td>90</td>
<td>75</td>
</tr>
<tr>
<td>Chloride</td>
<td>80</td>
<td>65</td>
</tr>
<tr>
<td>Potassium</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Citrate</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Osmolarity</td>
<td>311* mOsm/litre</td>
<td>245 mOsm/litre</td>
</tr>
</tbody>
</table>

* Hyperosmolar with respect to plasma osmolality (normal = 276–295 mOsm/litre).

If using bicarbonate ORS there are 30 mmol/litre of bicarbonate instead of citrate.

Children with severe malnutrition, as this product is specifically designed for such children.

**Zinc supplementation**

Zinc is an important micronutrient for children’s overall health and development. It is lost in greater quantity during diarrhoea. Replacing the lost zinc is therefore important both for helping the child to recover and for keeping them healthy in the coming months. It has been shown that zinc supplements given during an episode of diarrhoea reduce the duration and severity of the episode, and lower the incidence of diarrhoea in the following 2–3 months. For these reasons, all patients with diarrhoea should be given zinc supplements as soon as possible after the diarrhoea has started. Give 10 mg/kg for infants less than 6 months old and 20 mg/kg for older infants and children for 14 days.

**Treatments for different degrees of dehydration with/without shock**

Dehydration does not neatly fit into discrete categories, although texts such as this one and the WHO publications show practicality in this way for clarity and guidance. Similarly, it can be very difficult to distinguish severe dehydration from shock, and the two ‘categories’ overlap. The essential point to understand is that each severely ill patient must be reassessed frequently to ascertain whether the treatment protocol is having the desired effect of reversing the life-threatening signs of fluid loss. Look for the following:

- Increasing awareness and response to stimuli
- Gradually strengthening pulse with a decreasing rate of heart rate, weak pulse, pale skin circulation time with prolonged capillary refill time (> 3 seconds), depressed conscious level, and low or even unmeasurable blood pressure.

Children severely dehydrated with shock: shock treatment phase

Children with shock will have a high and increasing heart rate, weak pulse, poor skin circulation time with prolonged capillary refill time (> 3 seconds), depressed conscious level, and low or even unmeasurable blood pressure.

These children require immediate resuscitation (ABC) and emergency treatment (see also Section 5.5.B).

**Airway (if patient has a reduced conscious level)**

- Use an opening manoeuvre if the airway is not open or if it is partially obstructed. Then keep the airway open. If there is immediate improvement but the airway closes without active opening support, consider using airway adjuncts to support the airway.
  - Suction if necessary, but not routinely.
  - If the child is deeply unconscious (P or less on the AVPU scale), the airway may need to be secured by intubation using experienced senior help (if available).

**Breathing**

- Give 100% oxygen (mask with reservoir and flow rate of at least 6 litres/minute) regardless of SpO2 (this increases oxygen delivery as well as improving tissue oxygenation).
- For inadequate ventilation or depressed conscious level (as indicated by the AVPU score) with hyperventilation, respiration should be supported with oxygen via a bag and mask, and experienced senior help summoned (if available).

**Circulation**

- Obtain vascular access to give boluses quickly. Insert an IV cannula and if facilities available send blood for a full blood count, urea and electrolytes, glucose, cross-matching (if anaemic) and clotting. If peripheral veins are difficult to access an intra-osseous infusion (e.g. EZIO) is rapid and effective. In the absence of IO equipment, the external jugular vein or long saphenous vein cut-down are good alternatives (see Section 5.8.B for circulatory procedures). If a skilled operator is available, an internal jugular vein central line is ideal, once an initial rapid infusion has been given, if the patient is very severely shocked and likely to need ongoing high dependency care, as it can also allow CVP measurements (if available).
- Give an initial rapid bolus of 10 mL/kg of Ringer-lactate or Hartmann’s solution and reassess. Do not use 5% glucose or 0.18% saline/4% glucose solutions for resuscitation, as these can cause hyponatraemia and cerebral oedema. Boluses should be manually pushed in using a 20- to 50-mL syringe (utilising a three-way tap and link to an IV giving set).
  - The re-assessment after the first bolus allows the clinician to ascertain whether the child has any contraindications to large volume resuscitation. Assess for:
    - malnutrition (this should be obvious; see Section 5.10.B) severe anaemia or cardiac problem. Rapid fluid infusion can be fatal in malnutrition, severe anaemia or cardiac problems. Stop the rapid infusion and proceed more slowly with reference to Sections 5.10.B malnutrition, 5.11.A anaemia and 5.4.B heart failure and consider a blood transfusion.
    - Further 10 mL/kg boluses with reassessment will usually be required if shock continues. In a child with shock from severe dehydration caused by diarrhoea, it would be very unusual to need more than 30–40 mL/kg to improve the child’s circulation. Reconsider the diagnosis. For example:
      - surgical abdominal pathology (e.g. intussusception or volvulus) (see Section 5.19)
      - additional pathology e.g septicaemia (see Section 5.5.C)
      - ongoing severe diarrhoea, particularly if there is a cholera epidemic.
Once a total of 40 mL/kg of boluses have been given IV, complications such as pulmonary oedema may occur. If available, expert help (including CVP monitoring and facilities for positive pressure ventilation) is essential; if expert help is not available and there is ongoing severe diarrhoea, continue with fluid resuscitation until there is some improvement in conscious level.

If a blood glucose shows hypoglycaemia (<2.5 mmol/L) or glucose stick test has not been available, give a dose of 5 mL/kg of 10% glucose IV to any child who still has a depressed conscious level, as hypoglycaemia may be contributing to this problem. Increased alertness confirms hypoglycaemia (and see below).

Keep the patient warm, but do not overheat them as this will cause peripheral vasodilatation and reduce the blood supply to vital centres. Hypothermia will exacerbate poor peripheral perfusion, acidosis and coagulation abnormalities.

Elevate the legs (raise the foot of the bed).

Give a 10 mL/kg bolus of fresh blood as soon as possible if severe anaemia is present, but watch for circulatory overload.

Consider using broad-spectrum IV antibiotics.

Monitor urine output.

If the child has a reduced level of consciousness or has a convulsion, particularly if they are an infant or young child, hypoglycaemia may be present. Always measure the blood glucose level in this situation. However, if blood glucose measurement is not possible, always treat as for presumed hypoglycaemia and, in addition to the IV fluids given above, give 5 mL/kg of 10% glucose IV or, if there is no IV access, by intra-osseous needle.

As shock is being treated, reassess the child’s vital signs: alertness, pulse, respiratory rate etc. after each bolus and at least every 15–30 minutes until signs of shock are improving. Increased alertness, lower pulse and respiratory rate are encouraging signs, but the easiest and most sensitive to recognise is the degree of responsiveness.

### Children severely dehydrated with shock: rehydration phase

The best route for rehydration is the oral or nasogastric one, but in children who were sick enough to require rapid IV boluses, further IV fluid is likely to be needed initially.

At this stage, also, there is a need to again consider hypoglycaemia (which may have been identified earlier on stick testing). See below.

### Fluid requirement for replacing in the rehydration phase

Fluid requirement falls into the three categories mentioned above:

1. Correction of deficit
   - Weigh the child again or estimate the weight as above
   - Re-assess the clinical signs of dehydration as shown in Table 5.12.A.2 and estimate the percentage of dehydration: fluid deficit in mL = weight in kg × % dehydrated × 10
   - e.g. a 6 kg child with a 5% dehydration will have 6 × 5 × 10 = 300 mL deficit.
2. Replacement of ongoing losses
   - For each diarrhoeal stool: < 2 years of age: give 50–100 mL or 10 mL/kg and ≥ 2 years of age: give 100–200 mL
   - For each vomit: use 2 mL/kg ORS
   - For nasogastric tube aspirates: replace volume for volume
   - e.g. a 6 kg child with 5 loose watery stools will need another 300 mL as replacement.

### Maintenance fluids (see Table 5.12.A.5)

<table>
<thead>
<tr>
<th>TABLE 5.12.A.5 IV maintenance fluids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
</tr>
<tr>
<td>--------</td>
</tr>
<tr>
<td>First 10 kg of body weight</td>
</tr>
<tr>
<td>Second 10 kg of body weight</td>
</tr>
<tr>
<td>Subsequent kg</td>
</tr>
</tbody>
</table>

The 6 kg child will need 600 mL in 24 hours for maintenance.

Total fluid in 6 kg child with 5 loose watery stools who is 5% dehydrated is 300 + 300 + 600 mL = 1200 in 24 hours. The IV would be set to run at 50 mL/hr. Initially, adjustments to the volume will have to be made in the presence of further large watery stools or vomits or nasogastric aspirate. If available, a check on the plasma electrolytes is very useful at least daily to monitor response to treatment and to guide further therapy. Clinical observations should be done at least hourly and include looking for evidence of urine output.

### Choice of IV fluid

As described before, a solution such as Ringer’s lactate or Hartman’s solution is preferable to Normal (0.9%) Saline as it contains less chloride and contains potassium which is vital in diarrhoea treatment. If N saline must be used, add 10 mmol of potassium chloride to each 500 mL bag once urine has been passed. If Ringer’s lactate or Hartman’s solution are being used, add 5 mmol to each 500 mL bag once urine has been passed.

There is an advantage in managing these children with a urinary catheter as urine volume measurement is a useful guide to fluid need in the absence of cardiac failure but its use must be weighed against the risk of infection.

There is always a possibility of hypoglycaemia as the child is not eating (see below) so for this reason, add glucose to the infusion fluid.

To make a 5% solution of dextrose in Ringer’s lactate, Hartman’s solution or N saline, remove 50 mL from the 500 mL bag and replace with 50 mL of 50% dextrose.

To make a 10% solution of dextrose in Ringer’s lactate, Hartman’s solution or N saline, remove 100 mL from the 500 mL bag and replace with 100 mL of 50% dextrose.

Start the rehydration fluid regime, review the child’s vital signs at least hourly, including assessing urine output and looking for signs of fluid overload, such as puffiness face or limbs or increased breathlessness. Also review if there is any change reported by the mother. Once the child is regaining a degree of responsiveness and has a gag reflex, consider introducing oral or nasogastric (enteral) fluids to replace the IV route.

### Re-introduction of enteral fluid

Re-assess the child’s dehydration status by checking skin pinch, level of consciousness, and ability to drink, at least...
every hour, in order to confirm that hydration is improving. Sunken eyes recover more slowly than other signs, and are less useful for monitoring.

As has been mentioned earlier, enteral fluid is the safest way to rehydrate the child. Enteral rehydration can be achieved when:

- The child is conscious enough to be fed by a nasogastric tube without aspiration i.e. there is a gag reflex present OR
- The child is conscious enough to take sufficient fluid orally AND
- The child is not vomiting a significant volume of the fluid

The enteral rehydration fluid should be reduced osmolarity ORS (or ReSoMal if malnutrition is present). ORS should be introduced while the IV infusion is still running and the IV fluid volume reduced accordingly. Allow the child to breast feed whenever they want.

Once volumes approaching those required (see WHO Plan B in the Appendix to this section) are reached, the IV infusion can be discontinued and WHO Plan B rehydration continued alone.

All the WHO Plans for rehydration with details on prevention fluids, home fluids and advice for parents can be found in the Appendix to this section (see below).

**Hypoglycaemia in diarrhoea (blood glucose < 2.5 mmol/L or < 45 mg/dL)**

If the child has a reduced level of consciousness or has a convulsion, particularly if they are an infant, hypoglycaemia may be present. Always measure the blood glucose level in this situation. However, if blood glucose measurement is not possible, always treat as for presumed hypoglycaemia. Give 2–5 mL/kg of 10% glucose IV or, if there is no IV access, by intra-osseous needle. If there is no circulatory access, while further attempts are made to access the circulation, any hypoglycaemia can be temporarily managed as below, if there are sufficient staff.

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**Sublingual sugar (sucrose) for treatment of hypoglycaemia**

- Sublingual sugar may be used as an immediate 'first-aid' measure for managing hypoglycaemia in an unconscious child in situations where IV administration of glucose may be impossible or delayed.
- Give 1 teaspoonful of sugar, moistened with 1–2 drops of water, under the tongue. More frequent repeated doses are needed to prevent relapse. Children should be monitored for early swallowing, which leads to delayed absorption, and in this case another dose of sugar should be given. If sublingual sugar is given, repeat doses at 20-minute intervals.
- Recheck the blood glucose concentration in 20 minutes, and if the level is low (< 2.5 mmol/litre or < 45 mg/dL) repeat the sublingual sugar.
- Clearly, once an IV or IO access has been established, glucose can be given into the circulation if necessary.

**Electrolyte disturbances in dehydration from diarrhoeal illnesses**

Knowledge of the levels of serum electrolytes rarely changes the management of children with diarrhoea. Indeed, these values are often misinterpreted, leading to inappropriate treatment. The disorders described below are usually adequately treated by oral rehydration therapy (ORT).

**Hyponatraemia**

Some children with diarrhoea develop hyponatraemic dehydration, especially when given drinks that are hypertonic due to their sugar content (e.g. soft drinks, commercial fruit drinks) or salt. These draw water from the child’s tissues and blood into the bowel, causing the concentration of sodium in extracellular fluid to rise. If the solute in the drink is not fully absorbed, the water remains in the bowel, causing osmotic diarrhoea.

Children with hyponatraemic dehydration (serum Na+ > 150 mmol/litre) have thirst that is out of proportion to other signs of dehydration. Their most serious problem is convulsions, which usually occur when the serum sodium concentration exceeds 165 mmol/litre, and especially when intravenous therapy is given. Seizures are much less likely to occur when hyponatraemia is treated with ORS, which usually causes the serum Na+ concentration to become normal within 24 hours.

**Hypernatraemia**

Children with diarrhoea who drink mostly water, or watery drinks that contain little salt, may develop hypernatraemia (serum Na+ < 130 mmol/litre). Hypernatraemia is especially common in children with shigellosis and in severely malnourished children with oedema. It is occasionally associated with lethargy and (less often) with seizures. ORS is safe and effective therapy for nearly all children with hypernatraemia. An exception is children with oedema, for whom ORS may provide too much sodium. ReSoMal (see Section 5.10.B) may be helpful here.

**Hypokalaemia**

Inadequate replacement of potassium losses during diarrhoea can lead to potassium depletion and hypokalaemia (serum K+ < 3 mmol/litre), especially in children with malnutrition. This can cause muscle weakness, paralytic ileus, impaired kidney function and cardiac arrhythmias. Hypokalaemia is worsened when base (bicarbonate or lactate) is given to treat acidosis without simultaneously providing potassium. Hypokalaemia can be prevented, and the potassium deficit corrected, by using ORS for rehydration therapy and by giving foods rich in potassium during diarrhoea and after it has stopped (e.g. bananas, coconut water, dark green leafy vegetables).

It is also essential to check blood potassium levels, especially if the child has not passed urine, prior to replacing potassium IV, in order to avoid complications of hyperkalaemia secondary to pre-renal failure.

If it is necessary to give potassium intravenously (e.g. if serum K+ is < 2.0 mmol/litre or there are ECG signs of hypokalaemia, namely ST depression, T-wave reduction and prominent U waves), great care must be taken. In acute depletion, an infusion at the rate of 0.2 mmol/kg/hour can be used and the serum K+ level checked after 3 hours. The potassium for injection must be diluted before use and thoroughly mixed before being given. The maximum...
concentration of potassium that can be given through a peripheral vein is 40 mmol/litre. The maximum infusion rate of potassium is 0.5 mmol/kg/hour. The recommended concentration is 20 mmol/litre.

Note: The injectable form of KCl usually contains 1.5 grams (i.e. 20 mmol of potassium in 10 mL), and can be given orally. The daily potassium requirement is 2.5–3.5 mmol/kg.

Supportive treatments

Dietary therapy
During diarrhoea, a decrease in food intake, lack of nutrient absorption and increased nutrient requirements combine to cause weight loss and failure to grow. In turn, malnutrition can make the diarrhoea more severe, more prolonged and more frequent, compared with diarrhoea in non-malnourished children. Therefore give nutrient-rich foods during the diarrhoea and when the child is recovering.
- Breastfed infants: continue feeding on demand.
- Bottle-fed infants: administer full-strength formulas immediately after rehydration (no longer than 4 hours). Lactose intolerance may develop and cause an exacerbation of diarrhoea with a lactose-containing formula. If this happens, temporarily reduce or remove lactose from the diet.
- Older children: continue their usual diet during diarrhoea. Recommended foods include starchy, cereals, yoghurt, fruits and vegetables. Foods high in simple sugars and fats should be avoided. Excess fluid losses via vomiting or diarrhoea must be replaced with ORS (see above).

Zinc treatment
Zinc is an important micronutrient which is lost in diarrhoeal illnesses. Replacement speeds recovery and reduces severity as well as reducing the frequency of diarrhoeal illnesses in the ensuing 2 to 3 months.

Dose under 6 months of age 10 mg (½ tablet) daily for 10–14 days; dose over 6 months of age 20 mg (1 tablet) daily for 10–14 days.

Drug therapy: use of antimicrobial and ‘anti-diarrhoeal’ drugs
Antimicrobial drugs should not be used routinely. This is because, except as noted below, it is not possible to distinguish clinically episodes that might respond, such as diarrhoea caused by entero-toxigenic E. coli, from those caused by agents unresponsive to antimicrobials, such as rotavirus or Cryptosporidium. Moreover, even for potentially responsive infections, selecting an effective antimicrobial drug requires knowledge of the likely sensitivity of the causative agent, and such information is usually unavailable. In addition, use of antimicrobials adds to the cost of treatment, risks adverse reactions and enhances the development of resistant bacteria.

Antimicrobial drugs are reliably helpful only for children with bloody diarrhoea (probable shigellosis), suspected cholera with severe dehydration, and serious non-intestinal bacterial infections such as pneumonia. Antiprotozoal drugs are rarely indicated except as described below when a definite diagnosis is available.

Antimicrobial drugs for acute diarrhoea

Neonates
Diarrhoea and vomiting may be a symptom of septicaemia. If septicaemia is suspected, parental antibiotics are required (see Section 3.4).

Bloody diarrhoea
- Bacterial causes: Campylobacter jejuni, Shigella sonnei, Shigella flexneri and Shigella dysenteriae, and less commonly Salmonella, E. coli 0157:117 and Aeromonas.
- May be accompanied by abdominal pain and rectal prolapse.
- As culture facilities may not be available, sick toxic children with bloody diarrhoea should be treated for shigella dysentery.
- Children with diarrhoea and blood in stool (dysentery) should be treated with ciprofloxacin as first-line treatment and ceftaxone as second-line treatment if they are severely ill and local antimicrobial sensitivity is not known. Where local antimicrobial sensitivity is known, local guidelines should be followed:
  - ciprofloxacin: 20 mg/kg/dose twice daily for 5 days
  - gatifloxacin: 80 mg/kg IV or IM once daily for 5 days.

- Mild infections due to Shigella sonnei are usually self-limiting. Shigella in resource-limited countries is commonly resistant to co-trimoxazole and ampicillin. Nalidix acid, ciprofloxacin, ceftaxone or the antibiotic of choice for the area should be used for a 5-day course.
- In infants and young children, exclude surgical causes (e.g. intussusception) (see Section 5.19).

Salmonella
If non-typhoidal Salmonella is suspected in infants under 1 year of age or in immunocompromised children, blood cultures should be undertaken. If these are positive or the infant is toxic, an appropriate parenteral antibiotic should be given (e.g. chloramphenicol, cefixime or ciprofloxacin) for 7–10 days. Be alert for pneumonia or metastatic abscesses in bone, brain or elsewhere. Otherwise Salmonella gastroenteritis is not treated with antibiotics.

Systemic Salmonella infection is common in malnutrition, HIV infection, sickle-cell disease and schistosomiasis.

Campylobacter jejuni (and also Shigella and Salmonella) may cause severe abdominal pain, mimicking a surgical emergency. Otherwise the disease is self-limiting and does not require antibiotics. If treatment is considered appropriate, erythromycin (12.5 mg/kg four times daily) for 5 days is the antibiotic of choice.

Other causes of diarrhoea that warrant antimicrobial treatment
- Amoebic dysentery: this is diagnosed by microscopy of fresh warm stool. Treatment is with metronidazole 10 mg/kg three times daily (maximum dose 2 grams) for 5–7 days.
- Cholera: this is usually only diagnosed during epidemics. If the child has severe watery diarrhoea, suspect cholera or enterotoxigenic E. coli (only diagnosed by specialist laboratories). Treatment for cholera is with tetracycline 12.5 mg/kg four times a day for 3 days in children aged over 8 years. The alternative for young children is chloramphenicol 25 mg/kg 8-hourly for 3 days. In addition to rehydration, give an antibiotic to which local
strains of *Vibrio cholerae* are sensitive. These include tetracycline, doxycycline, co-trimoxazole, erythromycin and chloramphenicol.

- **Giardiasis:** this is diagnosed by microscopy of stool, and is usually self-limiting or asymptomatic. If symptomatic in a malnourished child or the disease is prolonged, it is justified to treat with metronidazole for 5 days (as for amoebic dysentery). Tinidazole is an alternative (50–75 mg/kg once only (maximum dose 2 grams), a second dose may be given if necessary).

- **Clostridium difficile** usually occurs after a course of antibiotics for some other illness, and is associated with antibiotic-associated pseudomembranous colitis (there is a danger of bowel perforation). Antibiotics, especially clindamycin, may alter the flora of the gastrointestinal tract and allow overgrowth of *C. difficile*. The latter produces a toxin which causes damage to the gut mucosa, resulting in pseudomembranous colitis. Confirmation is by culture of *C. difficile* in the faeces. Treatment is with oral vancomycin for 7–10 days, which clears *C. difficile* from the gut. The doses are orally:
  - **Child 1 month–5 years:** 5 mg/kg 4 times daily for 10–14 days (increased up to 10 mg/kg 4 times daily if infection fails to respond or is life threatening)
  - **Child 5–12 years:** 62.5 mg 4 times daily for 10–14 days (increased up to 250 mg 4 times daily if infection fails to respond or is life threatening)
  - **Child 12–18 years:** 125 mg 4 times daily for 10–14 days (increased up to 500 mg 4 times daily if infection fails to respond or is life threatening).

### Symptomatic drugs

‘Antidiarrhoeal’ drugs and anti-emetics have no practical benefits for children with acute or persistent diarrhoea. They do not prevent dehydration or improve nutritional status, which should be the main objectives of treatment. Some, like loperamide, have dangerous and sometimes fatal side effects. These drugs should never be given to children under 5 years of age.

### Treatment of rectal prolapse

Gently push back any tissue that has come out of the anus using a surgical glove or wet cloth, or if it is oedematous and cannot be reduced, warm compresses of magnesium sulphate may reduce the oedema.

### Haemolytic–uraemic syndrome

If laboratory tests are not available, suspect this syndrome when purpura, pallor, altered level of consciousness and low or absent urine output are present. If laboratory tests are available, blood smear shows fragmented red cells and decreased or absent platelets. There will be an increase in blood urea and creatinine levels (see Section 5.6.A).

### Appendix

#### WHO Treatment Plan A: home therapy to prevent dehydration and malnutrition

Children with no signs of dehydration need extra fluids and salt to replace their losses of water and electrolytes due to diarrhoea. If these are not given, signs of dehydration may develop.

Mothers should be taught how to prevent dehydration at home by giving the child more fluid than usual, how to prevent malnutrition by continuing to feed the child, and why these actions are important. They should also know what signs indicate that the child should be taken to a health worker. These steps are summarised in the four rules of Treatment Plan A.

#### Rule 1: Give the child more fluids than usual, to prevent dehydration

**What fluids to give**

Many countries have designated recommended home fluids. **Wherever possible, these should include at least one fluid that normally contains salt** (see below). Plain clean water should also be given. Other fluids should be recommended that are frequently given to children in the area, that mothers consider acceptable for children with diarrhoea, and that mothers would be likely to give in increased amounts when advised to do so.

**Suitable fluids**

Most fluids that a child normally takes can be used. It is helpful to divide suitable fluids into two groups:

- **Fluids that normally contain salt, such as:**
  - ORS solution
  - salted drinks (e.g. salted rice water or a salted yoghurt drink)
  - vegetable or chicken soup with salt.
  
  Insert

Teaching mothers to add salt (about 3 g/L) to an unsalted drink or soup during diarrhoea is also possible, but requires a sustained educational effort.

A home made solution containing 3 g/L of table salt (one level teaspoon) and 18 g/L of common sugar (sucrose) is effective but is not generally recommended because the recipe is often forgotten, the ingredients may not be available or too little may be given.

**Fluids that do not contain salt, such as:**

- plain water
- water in which a cereal has been cooked (e.g. unsalted rice water)
- unsalted soup
- yoghurt drinks without salt
- green coconut water
- weak tea (unsweetened)
- unsweetened fresh fruit juice.

**Unsuitable fluids**

A few fluids are potentially dangerous and should be avoided during diarrhoea. Especially important are drinks sweetened with sugar, which can cause osmotic diarrhoea and hypernatraemia. Some examples are:

- commercial carbonated beverages
- commercial fruit juices
- sweetened tea.

Other fluids to avoid are those with stimulant, diuretic or purgative effects, for example

- coffee
- some medicinal teas or infusions.

**How much fluid to give**

The general rule is: give as much fluid as the child or adult can comfortably drink.
wants until the diarrhoea stops. As a guide, after each loose stool, give:

- children under 2 years of age: 50–100 mL (a quarter to half a large cup) of fluid
- children aged 2 up to 10 years: 100–200 mL (a half to one large cup)
- older children and adults: as much fluid as they want. Insert

**Rule 2: Give supplemental zinc (10–20 mg) to the child every day for 10 to 14 days**

Zinc can be given as a syrup or as dispersible tablets, whichever formulation is available and affordable. By giving zinc as soon as diarrhoea starts, the duration and severity of the episode as well as the risk of hydration will be reduced. By continuing zinc supplementation for 10–14 days, the zinc lost during diarrhoea is fully replaced and the risk of the child having new episodes of diarrhoea in the following 2 to 3 months is reduced.

**Rule 3: Continue to feed the child, to prevent malnutrition**

The infant’s usual diet should be continued during diarrhoea and increased afterwards. Food should never be withheld, and the child’s usual foods should not be diluted. Breastfeeding should always be continued. The aim is to give as much nutrient-rich food as the child will accept. Most children with watery diarrhoea regain their appetite after dehydration is corrected, whereas those with bloody diarrhoea often eat poorly until the illness resolves. These children should be encouraged to resume normal feeding as soon as possible.

When food is given, sufficient nutrients are usually absorbed to support continued growth and weight gain. Continued feeding also speeds the recovery of normal intestinal function, including the ability to digest and absorb various nutrients. In contrast, children whose food is restricted or diluted lose weight, have diarrhoea of longer duration, and recover intestinal function more slowly.

**What foods to give**

This depends on the child’s age, food preferences and pre-illness feeding pattern; cultural practices are also important. In general, foods suitable for a child with diarrhoea are the same as those required by healthy children. Specific recommendations are given below.

**Milk**

- Infants of any age who are breastfed should be allowed to breastfeed as often and as long as they want. Infants will often breastfeed more than usual; this should be encouraged.
- Infants who are not breastfed should be given their usual milk feed (or formula) at least every three hours, if possible by cup. Special commercial formulas advertised for use in diarrhoea are expensive and unnecessary; they should not be given routinely. Clinically significant milk intolerance is rarely a problem.
- Infants below six months of age who take breast milk and other foods should receive increased breastfeed-

ing. As the child recovers and the supply of breast milk increases, other foods should be decreased (if fluids other than breast milk are given, use a cup, not a bottle). This usually takes about 1 week. If possible, infants of this age should become exclusively breastfed.

There is no value in routinely testing the stools of infants for pH or reducing substances. Such tests are oversensitive, often indicating impaired absorption of lactose when it is not clinically important. It is more important to monitor the child’s clinical response (i.e. weight gain, general improvement). Milk intolerance is only clinically important when milk feeding causes a prompt increase in stool volume and a return or worsening of the signs of dehydration, often with loss of weight.

**Other foods**

If the child is at least 6 months old or is already taking soft foods, he or she should be given cereals, vegetables and other foods, in addition to milk. If the child is over 6 months old and such foods are not yet being given, they should be started during the diarrhoea episode or soon after it stops.

Recommended foods should be culturally acceptable, readily available, have a high content of energy and provide adequate amounts of essential micronutrients. They should be well cooked, and mashed or ground to make them easy to digest; fermented foods are also easy to digest. Milk should be mixed with a cereal. If possible, 5–10 mL of vegetable oil should be added to each serving of cereal. (Most staple foods do not provide enough calories per unit weight for infants and young children. This is improved by adding some vegetable oil.) Meat, fish or egg should be given, if available. Foods rich in potassium, such as bananas, green coconut water and fresh fruit juice, are beneficial.

**How much food and how often**

Offer the child food every three or four hours (six times a day). Frequent small feedings are tolerated better than less frequent large ones.

After the diarrhoea stops, continue giving the same energy-rich foods and provide one more meal than usual each day for at least 2 weeks. If the child is malnourished, extra meals should be given until the child has regained normal weight for height.

**Rule 4: Take the child to a healthcare worker if there are signs of dehydration or other problems**

The mother should take her child to a healthcare worker if the child:

- starts to pass many watery stools
- has repeated vomiting
- becomes very thirsty
- is eating or drinking poorly
- develops a fever
- has blood in the stool
- does not get better in 3 days.

**WHO Treatment Plan B: oral rehydration therapy for children with some dehydration**

Children with some dehydration should receive oral rehydration therapy with ORS in a healthcare facility following the treatment plan described below.

Children with some dehydration should also receive zinc supplementation as described above.
giving ORS or home fluids according to Treatment Plan A. Do not give a diuretic. When the oedema has gone, resume stop giving ORS, but give breast milk or plain water, and food. They may be a sign of chronic malnutrition. If this occurs, the exact amount of solution required will depend on the child’s dehydration status. Children with more marked signs of dehydration, or who continue to pass frequent watery stools, will require more solution than those with less marked signs or who are not passing frequent stools. If the child wants more than the estimated amount of ORS, and there are no signs of over-hydration, give more. Oedematous (puffy) eyelids are a sign of over-hydration. They may be a sign of chronic malnutrition. If this occurs, stop giving ORS, but give breast milk or plain water, and food. Do not give a diuretic. When the oedema has gone, resume giving ORS or home fluids according to Treatment Plan A.

**How much ORS is needed?**

Use Table 5.12.A.6 to estimate the amount of ORS needed for rehydration. If the child’s weight is known, this should be used to determine the approximate amount of solution needed. The amount may also be estimated by multiplying the child’s weight in kg by 75 mL. If the child’s weight is not known, select the approximate amount according to the child’s age.

The exact amount of solution required will depend on the child’s dehydration status. Children with more marked signs of dehydration, or who continue to pass frequent watery stools, will require more solution than those with less marked signs or who are not passing frequent stools. If the child wants more than the estimated amount of ORS, and there are no signs of over-hydration, give more.

Oedematous (puffy) eyelids are a sign of over-hydration. They may be a sign of chronic malnutrition. If this occurs, stop giving ORS, but give breast milk or plain water, and food. Do not give a diuretic. When the oedema has gone, resume giving ORS or home fluids according to Treatment Plan A.

**How to give ORS**

A family member should be taught to prepare and give ORS. The solution should be given to infants and young children using a clean spoon or cup. Feeding bottles should not be used. For babies, a dropper or syringe (without the needle) can be used to put small amounts of solution into the mouth. Children under 2 years of age should be offered a teaspoonful every 1 to 2 minutes. Older children (and adults) may take frequent sips directly from the cup.

Vomiting often occurs during the first hour or two of treatment, especially when children drink the solution too quickly, but this rarely prevents successful oral rehydration, as most of the fluid is absorbed. After this time vomiting usually stops. If the child vomits, wait 5–10 minutes and then start giving ORS again, but more slowly (e.g. a spoonful every 2–3 minutes).

**Monitoring the progress of oral rehydration therapy**

Check the child from time to time during rehydration to ensure that ORS is being taken satisfactorily and that signs of dehydration are not worsening. If at any time the child develops signs of severe dehydration, switch to WHO Treatment Plan C.

**After 4 hours,** reassess the child fully, following the guidelines in Table 5.12.A.2. Then decide what treatment to give next:

- If signs of severe dehydration have appeared, intravenous (IV) therapy should be started following Treatment Plan C. This is very unusual, however, occurring only in children who drink ORS poorly and pass large watery stools frequently during the rehydration period.
- If the child still has signs indicating some dehydration, continue oral rehydration therapy by repeating Treatment Plan B. At the same time start to offer food, milk and other fluids, as described in Treatment Plan A (see above), and continue to reassess the child frequently.

If there are no signs of dehydration, the child should be considered fully rehydrated. When rehydration is complete:

- the skin pinch is normal
- thirst has subsided
- urine is passed
- the child becomes quiet, is no longer irritable and often falls asleep.

Teach the mother how to treat her child at home with ORS and food following Treatment Plan A. Give the mother enough ORS sachets for 2 days. Also teach her the signs that mean she should bring her child back.

- Use the patient’s age only when you do not know their weight. The approximate amount of ORS required (in mL) can also be calculated by multiplying the patient’s weight in kg by 75.
- If the patient wants more ORS than is shown above, give more.
- Encourage the mother to continue breastfeeding her child.
- For infants under 6 months who are not breast fed, if using the old WHO ORS solution containing 90 mmol/L of sodium also give 100–200 mL clean water during this period. However, if using the new reduced (low) osmolality ORS solution containing 75 mmol/L of sodium, this is not necessary.

**How to give ORS**

A family member should be taught to prepare and give ORS. The solution should be given to infants and young children using a clean spoon or cup. Feeding bottles should not be used. For babies, a dropper or syringe (without the needle) can be used to put small amounts of solution into the mouth.

Children under 2 years of age should be offered a teaspoonful every 1 to 2 minutes. Older children (and adults) may take frequent sips directly from the cup.

Vomiting often occurs during the first hour or two of treatment, especially when children drink the solution too quickly, but this rarely prevents successful oral rehydration, as most of the fluid is absorbed. After this time vomiting usually stops. If the child vomits, wait 5–10 minutes and then start giving ORS again, but more slowly (e.g. a spoonful every 2–3 minutes).

**Monitoring the progress of oral rehydration therapy**

Check the child from time to time during rehydration to ensure that ORS is being taken satisfactorily and that signs of dehydration are not worsening. If at any time the child develops signs of severe dehydration, switch to WHO Treatment Plan C.

**After 4 hours,** reassess the child fully, following the guidelines in Table 5.12.A.2. Then decide what treatment to give next:

- If signs of severe dehydration have appeared, intravenous (IV) therapy should be started following Treatment Plan C. This is very unusual, however, occurring only in children who drink ORS poorly and pass large watery stools frequently during the rehydration period.
- If the child still has signs indicating some dehydration, continue oral rehydration therapy by repeating Treatment Plan B. At the same time start to offer food, milk and other fluids, as described in Treatment Plan A (see above), and continue to reassess the child frequently.

If there are no signs of dehydration, the child should be considered fully rehydrated. When rehydration is complete:

- the skin pinch is normal
- thirst has subsided
- urine is passed
- the child becomes quiet, is no longer irritable and often falls asleep.

Teach the mother how to treat her child at home with ORS and food following Treatment Plan A. Give the mother enough ORS sachets for 2 days. Also teach her the signs that mean she should bring her child back.

- Use the patient’s age only when you do not know their weight. The approximate amount of ORS required (in mL) can also be calculated by multiplying the patient’s weight in kg by 75.
- If the patient wants more ORS than is shown above, give more.
- Encourage the mother to continue breastfeeding her child.
- For infants under 6 months who are not breast fed, if using the old WHO ORS solution containing 90 mmol/L of sodium also give 100–200 mL clean water during this period. However, if using the new reduced (low) osmolality ORS solution containing 75 mmol/L of sodium, this is not necessary.

**Meeting normal fluid needs**

While treatment to replace the existing water and electrolyte deficit is in progress, the child’s normal daily fluid requirements must also be met. This can be done as follows:

- **Breastfed infants:** continue to breastfeed as often and for as long as the infant wants, even during oral rehydration.
- **Non-breastfed infants** under 6 months of age: if using the old WHO ORS solution containing 90 mmol/L of sodium also give 100–200 mL clean water during this period. However, if using the new reduced (low) osmolality ORS solution containing 75 mmol/L of sodium, this is not necessary.
- **Older children:** throughout rehydration and maintenance therapy, offer as much plain boiled water to drink as they wish, in addition to ORS.

**If oral rehydration therapy must be interrupted**

If the mother and child must leave hospital before rehydration with ORS is complete:

- Show the mother how much ORS solution to give to finish the 4-hour treatment at home.

---

**TABLE 5.12.A.6 Guidelines for treating children with some dehydration: approximate amount of ORS to give in the first 4 hours**

<table>
<thead>
<tr>
<th>Age</th>
<th>&lt; 4 months</th>
<th>4–11 months</th>
<th>12–23 months</th>
<th>2–4 years</th>
<th>5–14 years</th>
<th>15 years or older</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>&lt; 5</td>
<td>5–7.9</td>
<td>8–10.9</td>
<td>11–15.9</td>
<td>16–29.9</td>
<td>30 kg or more</td>
</tr>
<tr>
<td>Volume (mL)</td>
<td>200–400</td>
<td>400–600</td>
<td>600–800</td>
<td>800–1200</td>
<td>1200–2200</td>
<td>2200–4000</td>
</tr>
</tbody>
</table>
Give her enough ORS packets to complete the 4-hour treatment and to continue oral rehydration for two more days, as shown in Treatment Plan A.

Show her how to prepare ORS solution.

Teach her the four rules in Treatment Plan A for treating her child at home.

**When oral rehydration fails**

With the previous ORS, signs of dehydration would persist or reappear in about 5% of children. With the new reduced (low) osmolality ORS it is estimated that such treatment ‘failures’ will be reduced to 3% or less. The usual causes for these ‘failures’ are:

- continuing rapid stool loss (more than 15–20 mL/kg/hour), as occurs in some children with cholera
- insufficient intake of ORS due to fatigue or lethargy
- frequent severe vomiting.

Such children should be given ORS by nasogastric (NG) tube or Ringer Lactate Solution intravenously (IV) (75 mLs/kg in four hours) usually in hospital. After confirming that the signs of dehydration have improved, it is usually possible to resume ORT successfully.

Rarely, oral rehydration therapy should not be given. This is true for children with:

- abdominal distension with paralytic ileus, usually caused by opiate drugs (e.g. codeine, loperamide) and hypokalaemia
- glucose malabsorption (indicated by a marked increase in stool output, failure of the signs of dehydration to improve, and a large amount of glucose in the stool).

In these situations, rehydration should be given IV until the diarrhoea subsides; nasogastric therapy should not be used.

**Giving zinc**

Begin to give supplemental zinc, as in Treatment plan A, as soon as the child is able to eat, following the four hour rehydration period.

**Giving food**

Except for breast milk, food should not be given during the initial 4-hour rehydration period. However, children who are continued on Treatment Plan B for longer than 4 hours should be given some food every 3–4 hours as described in Treatment Plan A. All children older than 6 months of age should be given some food before being sent home. This helps to emphasise to mothers the importance of continued feeding during diarrhoea.

**WHO Treatment Plan C: intravenous rehydration therapy for patients with severe dehydration**

The preferred treatment for children with severe dehydration is initial rapid intravenous rehydration following Treatment Plan C. If possible, the child should be admitted to hospital. Guidelines for IV rehydration are given in Table 5.12.A.7.

Children who can drink, even poorly, should be given ORS by mouth until the IV drip is running. In addition, all children should receive some ORS solution (about 5 mL/kg/hr) when they can drink without difficulty, which is usually within 3–4 hours for infants and 1–2 hours for older patients. This provides additional base and potassium which may not be adequately supplied by the IV fluid.

**Monitoring the progress of intravenous rehydration**

Patients should be reassessed every 15–30 minutes until a strong radial pulse is present. If it is not, the intravenous drip should be given more rapidly.

When the planned amount of intravenous fluid has been given (after 3 hours for older patients, or 6 hours for infants), the child's hydration status should be reassessed fully as in Table 5.12.A.2.

Look and feel for all the signs of dehydration.

- If signs of severe dehydration are still present, repeat the intravenous fluid infusion as outlined in Treatment Plan C. This is very unusual, however, occurring only in children who pass large watery stools frequently during the rehydration period.
- If the child is improving (able to drink) but still shows signs of dehydration, discontinue the intravenous infusion and give ORS for 4 hours, as specified in Treatment Plan B.
- If there are no signs of dehydration, follow Treatment Plan A. If possible, observe the child for at least six hours before discharge while the mother gives the child ORS, to confirm that she is able to maintain the child's hydration. Remember that the child will require therapy with ORS until the diarrhoea stops.

If the child cannot remain at the treatment centre, teach the mother how to give treatment at home following Treatment Plan A, give her enough ORS packets for two days and teach her the signs that mean she should bring her child back.

**What to do if intravenous therapy is not available**

- If IV therapy is not available at the facility, but can be given nearby (i.e. within 30 minutes), send the child immediately for intravenous treatment. If the child can drink, give the mother some ORS and show her how to give it to her child during the journey.
- If IV therapy is not available nearby, healthcare workers who have been trained can give ORS by NG tube, at a rate of 20 mL/kg body weight per hour for 6 hours (total of 120 mL/kg body weight). If the abdomen becomes sufficiently distended with paralytic ileus, or if the intestinal sounds are absent, then give 70 mL/kg of Ringer's Lactate Solution intravenously.

**Guidelines for intravenous treatment of children with severe dehydration**

<table>
<thead>
<tr>
<th>Age</th>
<th>First give Ringer’s Lactate Solution</th>
<th>Then give 70 mL/kg in:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants under 12 months</td>
<td>1 hour^4</td>
<td>5 hours</td>
</tr>
<tr>
<td>Older</td>
<td>30 minutes^5</td>
<td>Over 2.5 hours</td>
</tr>
</tbody>
</table>

Reassess the patient every 1–2 hours. If hydration is not improving, give the IV drip more rapidly. After six hours (infants) or three hours (older patients), evaluate the patient using the assessment chart. Then choose the appropriate Treatment Plan (A, B or C) to continue treatment

- a. If Ringers Lactate Solution is not available, normal saline may be used
- b. Repeat once if radial pulse is still very weak or not detectable

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**TABLE 5.12.A.7 Guidelines for intravenous treatment of children with severe dehydration**
swollen, ORS should be given more slowly until the abdomen becomes less distended.

- If NG treatment is not possible but the child can drink, ORS should be given by mouth at a rate of 20mL/kg body weight per hour for 6 hours (total of 120mL/kg body weight). If this rate is too fast, the child may vomit repeatedly. In this case, give ORS more slowly until the vomiting subsides.

- Children receiving NG or oral therapy should be reassessed at least every hour. If the signs of dehydration do not improve after 3 hours, the child must be taken immediately to the nearest facility where intravenous therapy is available. Otherwise, if rehydration is progressing satisfactorily, the child should be reassessed after 6 hours and a decision on further treatment made as described above for those given IV therapy.

- If neither NG nor oral therapy is possible, the child should be taken immediately to the nearest facility where IV or NG therapy is available.

Further reading


5.12.B Post-infectious prolonged or persistent diarrhoea

<table>
<thead>
<tr>
<th>BOX 5.12.B.1 Minimum standards</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Low-osmolality ORS and ReSoMal in severely malnourished children.</td>
</tr>
<tr>
<td>■ Ringer-lactate or Hartmann’s solution with potassium: oral and IV</td>
</tr>
<tr>
<td>■ Antibiotics: amoxicillin, gentamicin.</td>
</tr>
<tr>
<td>■ Vitamin A and zinc.</td>
</tr>
<tr>
<td>■ Electrolyte and mineral mix.</td>
</tr>
<tr>
<td>■ Folic acid.</td>
</tr>
</tbody>
</table>

Introduction

Epidemiology

- Diarrhoeal episodes that start acutely and last for 7–14 days are usually labelled as prolonged diarrhoea, and may be associated with greater morbidity and more severe nutritional consequences.

- Persistent diarrhoea is commonly defined as diarrhoea that starts acutely, but lasts for more than 14 days and is associated with growth faltering.

- Most cases are thus post-infectious in origin, and other disorders such as inflammatory bowel disease and coeliac disease are therefore excluded.

- Around 4–20% of all episodes of diarrhoea in resource-limited countries become prolonged, with associated case-fatality rates that may exceed 50% in severe cases.

- In parts of sub-Saharan Africa, the association of persistent diarrhoea with HIV infection is often the terminal event.

Risk factors for prolonged and persistent diarrhoea

Appropriate case management of acute diarrhoea is key to the prevention of prolonged episodes.

Specific pathogens: although some studies have identified an association between persistent diarrhoea and infections with organisms such as entero-aggregative E. coli or Cryptosporidium, this is by no means pathognomonic, nor is there a particular pattern of small bowel microbial colonisation or overgrowth seen in most cases. In HIV-endemic parts of Africa an association of persistent diarrhoea with cryptosporidiosis is well recognised, but may represent a manifestation of immunodeficiency. Evidence from Bangladesh does suggest that recurrent bouts of infection with bacterial pathogens such as Shigella lead to prolongation of the duration of successive diarrhoeal episodes, and thus there is a link between prolonged and persistent diarrhoea as an epidemiological continuum.

Malnutrition: persistent diarrhoea is commonly seen in association with significant malnutrition, and the relationship may be bidirectional. It is widely recognised that diarrhoeal episodes, especially if invasive, may become prolonged in malnourished children. The recent evidence of micronutrient deficiencies, especially of zinc and vitamin A in malnourished children with persistent diarrhoea, may indicate impaired immunological mechanisms for clearing infections, as well as ineffective mucosal repair mechanisms.

Dietary risk factors: although many children with persistent diarrhoea are lactose-intolerant, there is no role of specific dietary allergies in inducing and perpetuating enteropathy of malnutrition or post-infectious prolonged diarrhoea. Several studies have highlighted the high risk of prolonged diarrhoea with lactation failure and early introduction of artificial feeds in resource-limited countries.

Inappropriate management of acute diarrhoea: the association of prolongation of diarrhoea with food deprivation and inappropriately prolonged administration of parenteral fluids is well recognised. Unnecessary food withdrawal, and replacement of luminal nutrients, especially breast milk, with non-nutritive agents is prolonging the mucosal injury after diarrhoea. In particular, blanket administration of antibiotics and any administration of antimotility agents must be avoided. Optimal management of acute diarrhoea episodes with ORS, zinc and appropriate diets is a key factor in reducing the risk of recurrence and prolongation of diarrhoeal episodes.

Principles of management of persistent diarrhoea

In general, the management of persistent diarrhoea in malnourished children (see Figure 5.12.B.1) represents a blend of the principles of management of acute diarrhoea and malnutrition (see Section 5.12.A and Section 5.10.B).
Associated malnutrition may be quite severe in affected children, necessitating appropriate and rapid nutritional rehabilitation, sometimes in hospital. Given the chronicity of the disorder, prolonged hospitalisation may be quite problematic in resource-limited countries, and whenever possible the importance of ambulatory or home-based therapy must be emphasised.

The following represent the basic principles of management of persistent diarrhoea, and a suggested therapeutic approach is shown in Figure 5.12.B.1.

**Rapid resuscitation and stabilisation**
- Most children with persistent diarrhoea and associated malnutrition are not severely dehydrated, and oral rehydration is adequate.
- However, acute exacerbations and associated vomiting may require brief periods of intravenous rehydration with Ringer-lactate solution.
- Acute electrolyte imbalance such as hypokalaemia and severe acidosis may require correction (see Section 5.6.A).
- Associated systemic infections (bacteraemia, pneumonia and urinary tract infections) are well recognised in severely malnourished children with persistent diarrhoea, and are a frequent cause of early mortality. These must be screened for at admission. In severely ill children requiring hospitalisation, it may be best to cover with intravenous antibiotics at admission (usually ampicillin, IV 25 mg/kg three times daily up to a maximum of 4 grams/day, and gentamicin, IV 7.5 mg/kg once daily) while awaiting cultures. In other instances with suspected severe pneumonia, oral amoxicillin will suffice.
- It should be emphasised that there is little role for oral antibiotics in persistent diarrhoea, as in most cases the original bacterial infection that triggered the prolonged diarrhoea has disappeared by the time the child presents.

**Oral rehydration therapy**
This is the preferred mode of rehydration and replacement of ongoing losses. Although in general the standard low-osmolality WHO oral rehydration solution (containing 75 mmol/litre of Na+) is adequate, some evidence indicates that the hypo-osmolar rehydration fluid ReSoMal (containing 45 mmol/litre of Na+) as well as cereal-based oral rehydration fluids may be advantageous in severely malnourished children. In general, replacing each stool with about 50–100 mL of ORS or ReSoMal is safe.

**Enteral feeding and diet selection**
- Most children with persistent diarrhoea are not lactose intolerant, although administration of a lactose load exceeding 5 grams/kg/day is associated with higher rates of stooling and treatment failure. In general, Persistent diarrhoea
(diarrhoea >14 days with growth slowing)

Assessment, resuscitation and stabilisation
Oral rehydration
Treat electrolyte imbalance
Screen and treat associated secondary infections

Continued breastfeeding
Milk or yoghurt/cereal (usually rice) diet
Supplement with zinc and vitamin A

**RECOVERY**
Sustained feeding at home
Follow-up growth monitoring

**FAILURE TO RECOVER**
Continued diarrhoea and poor weight gain
Comminuted chicken diet or elemental feed

**Continued diarrhoea and poor weight gain**
Re-investigate to exclude intractable diarrhoea of infancy
IV feeding/fluids as a last resort

**FIGURE 5.12.B.1** Management of persistent diarrhoea in malnourished children.
therefore, withdrawal of milk and replacement with specialised (and expensive) lactose-free formulations is unnecessary.

- Alternative strategies for reducing the lactose load in malnourished children with persistent diarrhoea include reducing the overall amount of milk intake, addition of lactose-free milk to cereals, and replacement of milk with fermented milk products such as yoghurt. These measures have now been extensively evaluated in successive studies of the management of persistent diarrhoea in South Asia, and found to be extremely effective.

- It is rare to find persistent diarrhoea in breastfed infants, and it must be emphasised that breastfeeding must not be stopped under any circumstances.

- Rarely, when dietary intolerance precludes the administration of cow’s-milk-based formulations or milk, it may be necessary to administer specialised milk-free diets such as a comminuted or blenderised chicken-based diet or an elemental formulation. However, the latter may be almost unaffordable in most resource-limited countries. A choice of enteral diets and formulations is given in Table 5.12.B.2. It must be emphasised that this is extremely rare, and most infants will recover with the approach outlined above.

- The usual energy density of any diet used for the therapy of persistent diarrhoea should be around 1 kcal/gram, aiming to provide an energy intake of at least 110 kcal/kg/day and a protein intake of 2–3 grams/kg/day (in meals given six times daily). Nasogastric feeding may be required during the first 2–3 days of care, particularly while infection is being treated.

- There should be at least 3 successive days of increasing weight before a response can be verified.

- Dietary failure is shown by an increase in stool frequency (> 10 watery stools/day) or a failure to establish a daily weight gain within 7 days.

- In selected circumstances when adequate intake of energy-dense food is problematic, the addition of amylase to the diet through germination techniques which increase the endogenous amylase content of foods may be helpful. The ready-to-use therapeutic foods (RUTFs) can be used in moderate amounts in children with severe malnutrition and persistent diarrhoea, and the diets below also offer a suitable alternative.

### TABLE 5.12.B.2 Suggested composition of selected diets in children with persistent diarrhoea

<table>
<thead>
<tr>
<th>Component</th>
<th>Khitchri (rice-lentils) (per 100 grams)</th>
<th>Home made version of F-75 diet (WHO) (per 1000 mL)</th>
<th>Comminuted chicken (per 100 grams)</th>
<th>Semi-elemental diet (per 100 mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein</td>
<td>Mung lentils, 30 grams</td>
<td>Dried skimmed milk, 25 grams</td>
<td>Protein, 8 grams</td>
<td>Protein, 2.25 grams (hydrolysed)</td>
</tr>
<tr>
<td>Fat</td>
<td>Oil, 2 grams</td>
<td>Vegetable oil, 27 grams</td>
<td>Fat, 4 grams</td>
<td>Fat, 1.65 grams (medium-chain triglycerides)</td>
</tr>
<tr>
<td>Minerals and micronutrients</td>
<td>Salt (to taste)</td>
<td>Vitamin mix, 140 mg</td>
<td>Electrolytes (sodium, 0.4 mmol; potassium, 1.3 mmol; calcium, 0.2 mmol; phosphorus, 1.5 mmol)</td>
<td>Electrolytes (sodium, 1.9 mmol; potassium, 2.3 mmol; calcium, 1.8 mmol)</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>Rice, 60 grams</td>
<td>Cereal flour, 35 grams Sugar, 70 grams</td>
<td>Caloreen, 5 grams</td>
<td></td>
</tr>
</tbody>
</table>

**First diet: a starch-based reduced milk concentration (low-lactose) diet**

The diet should contain at least 70 kcal/100 grams, provide milk or yoghurt as a source of animal protein, but no more than 3.7 grams of lactose/kg body weight/day, and should provide at least 10% of calories as protein. The following example provides 83 kcal/100 grams, 3.7 grams of lactose/kg body weight/day and 11% of calories as protein:

- full-fat dried milk: 11 grams (or whole liquid milk: 85 mL)
- rice: 15 grams
- vegetable oil: 3.5 grams
- cane sugar: 3 grams
- water to make up to 200 mL.

Of the children who do not improve on this first diet, more than 50% will improve when given the second diet, from which the milk has been totally removed and starch (cereals) partly replaced with glucose or sucrose.

**Second diet: a no-milk (lactose-free) diet with reduced cereal (starch)**

The second diet should contain at least 70 kcal/100 grams, and provide at least 10% of calories as protein (egg or chicken). The following example provides 75 kcal/100 grams:

- whole egg: 64 grams
- rice: 3 grams
- vegetable oil: 4 grams
- glucose: 3 grams
- water to make up to 200 mL.

Finely ground, cooked chicken (12 grams) can be used in place of egg to give a diet that provides 70 kcal/100 grams.

Of the children who do not improve on the first diet, more than 50% will improve when given the second diet, from which milk has been totally removed and starch (cereals) partly replaced with glucose or sucrose.

**Micronutrient supplementation**

Most malnourished children with persistent diarrhoea have associated deficiencies of micronutrients, including zinc, iron and vitamin A. This may be a consequence of poor intake and continued enteral losses. It is therefore important to ensure that all children with persistent diarrhoea and malnutrition receive an initial dose of vitamin A orally, or if that is not possible by deep intramuscular injection (< 6 months of age, 50,000 units; 6–12 months, 100,000 units; > 1 year, 200,000 units). They should also receive a daily intake of the following for the next 2 weeks:

- a multivitamin supplement

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folic acid, 250 micrograms/kg on day 1, then 75 micrograms/kg/day.
- zinc, 3–5 mg/kg/day.
- copper, 0.3 mg/kg/day.
- magnesium, 0.2 mmol/kg/day.

Although the association of significant anaemia with persistent diarrhoea is well recognised, iron replacement therapy should not be initiated until recovery from diarrhoea has started (ferrous sulphate 18 mg/kg/day, or 6 mg/kg/day of elemental iron in two divided doses).

Follow-up and nutritional rehabilitation

Given the high rates of relapse in most children with persistent diarrhoea, it is important to address the underlying risk factors and institute preventive measures. These include appropriate feeding (breastfeeding, complementary feeding) and close attention to environmental hygiene and sanitation. This poses a considerable challenge in communities deprived of basic necessities such as clean water and sewage disposal.

By the time they return home, children should be receiving a diet that provides at least 110 kcal/kg/day (including milk and fresh fruit and well-cooked vegetables).

5.12.C Constipation

**BOX 5.12.C.1 Minimum standards**
- Movicol – osmotic laxative – softens
- Lactulose – osmotic laxative – softens
- Docusate sodium – softener and weak stimulant
- Senna or sodium picosulphate-stimulant laxative
- Glycerine suppositories – lubricant and rectal stimulant
- Small volume of phosphate enema (e.g. fleet enema)
- Sodium citrate enema (e.g. Micralax enema).

**Introduction**

**Definition**

Constipation is defined as difficulty with, delay in or pain on defecation.

**Normal defecation patterns**

- Breastfed babies average three stools per day and formula-fed babies two stools per day. However, the range of normal stool frequency in breastfed babies is very wide, from one stool every few days to a stool with every feed.
- Children average one stool per day after 3 years, but the normal range is from once on alternate days to three times daily.

**Pathophysiology**

Most children with constipation have no underlying medical cause. An episode of constipation can be triggered by inadequate food or fluid intake, an intercurrent illness, or excessive intake of cow’s milk.

**Constipation cycle**

The child passes a hard painful stool. On subsequent occasions they try to withhold the stool in order to avoid experiencing pain (faecal holding). The stool remains in the rectum, becoming harder still, and so causing even more pain when it is eventually passed.

If this cycle is allowed to continue, eventually the rectum may become enlarged, resulting in a ‘megacolon’. The child by this stage has lost the normal urge to defecate, and the large rectal mass of stool holds open the anal sphincter, which leads to soiling with liquid faeces. This is involuntary and should not be confused with encopresis, which occurs when the child voluntarily passes normal stools in unacceptable places.

**Further reading**


**Diagnosis**

Diagnosis can usually be made by taking a good history.

- On examination of the abdomen, faecal masses may be palpable. These are often in the left and right iliac fossae, but sometimes suprapubically. On inspection of the anus, anal tags and fissure may be seen in chronic constipation.
- On rectal examination, hard impacted faeces may be felt. Rectal examination is usually not necessary. If there is an anal fissure, rectal examination should be done with topical lignocaine jelly (1%) and terminated if it is too painful.
- Abdominal X-ray is not a useful examination for diagnosis of constipation.

**Pathological causes**

The vast majority of constipation is idiopathic, but there are a few uncommon causes that are important not to miss.
**Hirschsprung’s disease**
Suspect this when there is infancy-onset constipation and a delay of more than 48 hours in passing meconium at birth. In more advanced cases there will be abdominal distension and sometimes failure to thrive and vomiting. There may be alternating constipation and diarrhoea and surprisingly little soiling for the degree of constipation. On rectal examination an explosive gush of faeces occurs when the examining finger is withdrawn.

**Anal lesions that cause pain or create an obstruction**
These include anal fissures, perianal skin infections and (rarely) congenital anterior anus and anal stenosis. One cause of painful anal lesions is sexual abuse, a rare but important cause which should not be missed.

**Endocrine conditions**
Hypothyroidism, renal tubular acidosis, diabetes insipidus and hypercalcaemia can be associated with constipation. There should be a high level of suspicion for a metabolic or endocrine cause if constipation and failure to thrive coexist.

**Neurogenic constipation**
Spinal cord lesions involving sensation in the rectum will cause neurogenic constipation. These can be excluded by a normal neurological and spinal examination.

**Management of idiopathic constipation**
Parental understanding of the aetiology and sequence of events in developing chronic constipation is crucial to successful physical and psychological management (see Figure 5.12.C.1). Each and every element of this flow diagram should be addressed and treated if management is to be completely successful.

**Explanation**
A careful and thorough explanation of the problem should be given to the parent and child. Emphasise that soiling is not deliberate, and that the child needs support, not condemnation. Assess the need for psychological as well as physical treatment.

**Evacuation of hard impacted faeces**
1. To soften and lubricate the retained faeces, initially give a softener. This could be a macrogol such as Movicol or another softening laxative such as docusate sodium. The dose will vary according to age.
2. Alongside the softener, in order to expel the retained mass, give a stimulant laxative (e.g. sodium picosulphate).
3. If sodium picosulphate is not available, a large dose of sevina can be tried, but may need to be used for longer.
4. Only if the above fails give suppositories (glycerine) once daily (infant, 1 gram; child < 12 years, 2 grams; child > 12 years, 4 grams).
5. If the oral and suppository methods are unsuccessful, if excessive abdominal pain develops and/or there is vomiting, stimulant enemas will be required. Phosphate enemas should not be used in children under 2 years of age. For children aged 2–10 years give 60 mL (half a phosphate enema) and for those over 10 years of age give 120 mL (full enema). If phosphate enemas are not available, a small-volume sodium citrate enema (micro-enema) can be used. However, the use of enemas can add to the child’s fear of defecation. The child should never be forcibly held down to receive an enema. Give enemas once a day in the morning. Most children need two or three enemas to clear a faecal mass.
6. If these measures fail, the child should undergo manual evacuation of faecal mass under general anaesthetic, but only if this is available and can be done safely.

**Maintenance laxatives to keep the stool soft, defecation pain-free and overcome faecal holding**
- Softening agents such as Movicol or docusate sodium to keep the stool soft.
- Stimulant laxatives, usually senna or sodium picosulphate, to expel the soft stool. The aim is to produce

---

**FIGURE 5.12.C.1** Sequence of events in faecal soiling.
loose stools initially and then subsequently reduce the dose to produce at least one soft stool per day. Often large doses will be required initially to overcome the child’s faecal holding.

**Behaviour changes**
- Encourage increased fluid intake and a high-roughage diet (fruit, vegetables and cereals).
- Give positive praise and encouragement for regular toileting, and for passage of stool into the toilet.

**Length of treatment**
Children are likely to require several months of stimulant laxatives until their fear of defecation resolves, and often months to years of continuous or intermittent treatment with softening laxatives. A general rule of thumb is that the child will need laxatives for the same length of time that they were constipated before treatment started.

## 5.12.D Inflammatory bowel disease

### BOX 5.12.D.1 Minimum standards
- Aminosalicylates.
- Prednisolone.
- Methylprednisolone.
- Corticosteroid enemas.
- Blood transfusion.
- Polymeric diet.
- Metronidazole.

### Introduction
Inflammatory bowel disease (IBD) is uncommon in children in resource-limited countries, where abdominal tuberculosis is more common. However, in the UK about 18% of children with IBD are non-white, of whom most are of either Indian or Caribbean origin. Although IBD may present in younger children, the mean age in the UK is approximately 12 years. Crohn’s disease is more than twice as common as ulcerative colitis. A family history is common.

### Diagnosis
- Clinical symptoms of ulcerative colitis are almost invariably bloody diarrhoea with predefecation abdominal pain and tenesmus. Crohn’s disease may have a wide variety of symptoms, especially extra-intestinal ones. Iron-deficiency anaemia is common in both.

### Investigations
- The interval between onset of symptoms and diagnosis is often over 6 months in Crohn’s disease, and may be 2–3 months in ulcerative colitis. Denial of symptoms is common, especially in adolescents.
- Growth parameters and investigations are a guide to the severity and duration of disease and the nutritional state of the child.
- Examination of the mouth and anus is essential.
- Stool examination is essential to exclude bacterial and parasitological causes of diarrhoea, especially before corticosteroids are prescribed.
- Normal investigations: acute-phase reactants (erythrocyte sedimentation rate or C-reactive protein), haemoglobin, platelet count, albumin; do not exclude ulcerative colitis, but normal blood tests would be very unusual in Crohn’s disease.
- Children with ulcerative colitis often have little or no weight loss or growth failure.
- Children with Crohn’s disease and severe involvement of the colon may present similarly to those with ulcerative colitis, but generally have larger haematological changes.

### TABLE 5.12.D.1 Comparison between Crohn’s disease and ulcerative colitis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Ulcerative colitis</th>
<th>Crohn’s disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathology</strong></td>
<td>Mucosal disease</td>
<td>Transmural disease, skin lesions, strictures, fistulae</td>
</tr>
<tr>
<td><strong>Site</strong></td>
<td>Recto-colonic (rectum always involved). In children over 70% have a pancolitis</td>
<td>Panenteric disease is common in children: small bowel and colon, 50%; colon, 35%; ileum, 6%; upper gastrointestinal tract, 50%</td>
</tr>
<tr>
<td><strong>Common presenting symptoms</strong></td>
<td>Diarrhoea mixed with blood/mucus Pain (lower abdominal) Often no or little weight loss</td>
<td>Pain in the right iliac fossa Diarrhoea with or without blood Growth failure and weight loss Peri-anal and oral disease</td>
</tr>
<tr>
<td><strong>Extra-intestinal features</strong></td>
<td>Uncommon</td>
<td>Common</td>
</tr>
</tbody>
</table>

**General investigations**
- **Stool**
  - Blood, mucus.
  - Microscopy for *Entamoeba histolytica*, *Schistosoma*, *Trichuris trichiura*, *Giardia lamblia*.
  - Culture for bacteria.
International Maternal & Child Health Care

**Full blood count**
- Haemoglobin level decreased.
- White blood cell count increased.
- Platelet count increased.

**Acute-phase reactants**
- Erythrocyte sedimentation rate raised.
- C-reactive protein raised.

**Chemical pathology**
- Electrolytes (if diarrhoea severe).
- Ferritin (may be spuriously raised – acute-phase reactant).
- Albumin level low.

**Specific investigations**
Specific investigations depend on the availability of paediatric gastrointestinal facilities.
- Sigmoidoscopy is essential.
- Flexible endoscopy of the lower and upper gastrointestinal tract should ideally be undertaken.
- Barium enema (double contrast) is required in colitis only if colonoscopy is not available.
- Normal macroscopic appearance of the lower or upper gut does not exclude IBD. **Histology is essential.**
- ‘Indeterminate colitis’ is a term used to describe patients whose histology is not typical of ulcerative colitis or Crohn’s disease. They are usually treated initially as having ulcerative colitis.

**TABLE 5.12.D.2 Specific investigations for Crohn’s disease and ulcerative colitis**

<table>
<thead>
<tr>
<th>Feature</th>
<th>Ulcerative colitis</th>
<th>Crohn’s disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endoscopy*</td>
<td>Proctoscopy</td>
<td>Lower gut</td>
</tr>
<tr>
<td></td>
<td>Sigmoidoscopy</td>
<td>Upper gut*</td>
</tr>
<tr>
<td></td>
<td>Colonoscopy</td>
<td></td>
</tr>
<tr>
<td>Radiological studies</td>
<td>Barium enema†</td>
<td>Barium meal and follow-through</td>
</tr>
<tr>
<td></td>
<td>(double contrast)</td>
<td></td>
</tr>
<tr>
<td>White blood cell scan</td>
<td>Screening</td>
<td>Screening</td>
</tr>
<tr>
<td>(technetium labelled)‡</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Depending on availability.
† Only required if colonoscopy is unavailable.
‡ Only available in well-resourced countries.

**Management of ulcerative colitis**
- Initial management depends on severity.
- Follow-up: parents and older children should be taught so that they understand how to recognise and treat any relapse promptly.

**Management of active colitis (see Table 5.12.D.3)**
- **Mild disease:** less than four motions per day, intermittent blood, normal acute-phase reactants, no toxicity:
  - Aminosalicylates.
  - Mesalazine (1 g rectally) or corticosteroid (20 mg) enema until the bleeding stops, and then given alternate nights for 1 week.
- **Moderate disease:** four to six motions per day, moderate blood, slight toxicity, anaemia and raised acute-phase reactants:
  - As above plus oral steroids immediately. If there is a poor response, treat as for severe disease.
- **Severe disease:** more than six bloody motions per day, nocturnal stools, toxicity, fever, anaemia and hypoalbuminaemia:
  - Intravenous pulse methylprednisolone or hydrocortisone dose for 3–5 days.
  - Antibiotics (e.g. metronidazole) (benefit is not proven).
  - Intravenous fluids and correction of electrolyte deficits.
  - Blood transfusion if required.
  - Intravenous cyclosporine (500 micrograms–1 mg/kg aged 3–18 years) or oral cyclosporine (2 mg/kg twice daily maximum 5 mg/kg aged 2–18 years) may be of value if there is no response to intravenous corticosteroids.
- **Toxic dilation:** if there is no response to intensive therapy by 12–24 hours, perform colectomy.

**Relapse**
Prompt commencement of rectal mesalazine or a corticosteroid enema is essential. If there is no response, give a course of oral corticosteroids.

**Maintenance**
- Aminosalicylic acid preparations are generally given lifelong. Mesalazine 10 mg/kg 2–3 times daily 5–12 years, 2 G once daily 12–18 years.
- If relapses occur when corticosteroids are reduced, give azathioprine for up to 3–5 years.

**TABLE 5.12.D.3 Drug dosages for ulcerative colitis**

<table>
<thead>
<tr>
<th>Corticosteroids</th>
<th>Prednisolone: 2 mg/kg/day (maximum 40 mg) for 3 weeks, then reduce by 5 mg/week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Methylprednisolone: IV 1–1.5 mg/kg/day (maximum 60 mg)</td>
</tr>
<tr>
<td>Hydrocortisone:</td>
<td>IV 4 mg/kg 6-hourly</td>
</tr>
<tr>
<td>Prednisolone enema or foam (20 mg in 100 mL):</td>
<td>50–100 mL at night</td>
</tr>
<tr>
<td>Aminosalicylates</td>
<td>Sulphasalazine: (tablets 10 mg and 50 mg)</td>
</tr>
<tr>
<td></td>
<td>10 mg/kg 4- to 6-hourly for acute episodes. Decrease the dose by half for maintenance as soon as possible. Urine and tears will turn orange. Report sore throat</td>
</tr>
<tr>
<td>Mesalazine: (oral tablets 500 mg)</td>
<td>Under 40 kg body weight aged 5–12 years give 10 mg/kg 2–3 times daily; over 40 kg body weight aged 12–18 years, give 2 G once daily</td>
</tr>
<tr>
<td>Mesalazine: rectal</td>
<td>Aged 12–18 years, 1 gram daily</td>
</tr>
<tr>
<td>Metronidazole: (orally)</td>
<td>7.5 mg/kg three times daily</td>
</tr>
<tr>
<td>Azathioprine: (orally)</td>
<td>1.5–2.5 mg/kg once daily</td>
</tr>
</tbody>
</table>
Regular monitoring of the blood count (every 1 to 2 months) is important.

**Indicators for colectomy**
- Toxic megacolon (see above), intractable disease and growth failure.
- The risk of cancer relates to the extent of disease and its duration. Good maintenance therapy is important for prevention. Two-yearly colonoscopy should be considered in those with pancolitis for 10 years after the commencement of disease.
- Colectomy and ileostomy would be the usual operation in resource-limited settings, and are curative symptomatically, but the patient then has the ileostomy for life.

**Management of Crohn’s disease**
- The key to management is to maintain growth and nutrition and control symptoms.
- Most children will have recurrent relapses.
- Many will require surgery at some stage.
- Nutritional treatment and support are essential.

**Polymeric diet**
A polymeric diet can be any liquid nutritional preparation that is nutritionally complete. Examples would include PaediaSure/Ensure (Abbott Nutrition), Modulen IBD/Resource Junior (Nestle) and Alicalm/Fortini (Nutricia). Polymeric diet is effective in producing 70% remission in small bowel disease and 50% remission in colonic disease. The advantages over corticosteroids are the positive effect on growth and lack of side effects. The diet is given for 6 weeks, usually orally, during which time no other food is given (but the child can drink water), and then the normal diet is re-introduced.

Maintenance therapy with polymeric diet can also be used.

**Drug therapy**
- Prednisolone 2 mg/kg/day (maximum 40 mg/day) is effective in small and large bowel disease. Continue this dose for 3 weeks, then reduce it by 5 mg/week and then stop. If required to maintain remission, alternate-day therapy may have fewer side effects.
- Mesalazine but not sulphasalazine can be effective for maintaining remission in ileal as well as colonic disease (dose is aged 5–12 years 10–15 mg/kg orally 2–3 times daily, aged 12–18 years 2 G once daily).
- Azathioprine is effective in long-term maintenance and has steroid-sparing effects. It may be useful for healing perianal fistulae. It takes many months to act, and it should be continued for at least 4 years. Blood counts should be undertaken every 1–2 months.
- Metronidazole may be effective in controlling perianal disease and fistulae. It may also reduce small bowel overgrowth. Ciprofloxacin is an alternative.
- Infliximab is a very expensive monoclonal antibody that inhibits tumour necrosis factor alpha (TNFα). It is used in severe Crohn’s disease that is not responding to conventional treatment. It is administered IV at intervals. Because of its immunosuppressive effects there are real dangers from infection, especially latent TB. Other side effects include anaphylaxis, lymphoma and possibly demyelinating disorders.

**Surgery**
Indications for surgery include failure of medical therapy, intestinal obstruction and growth failure. Strictureplasty may be an effective method of avoiding excision of bowel when strictures are present.

**Follow-up and support for IBD**
Patients and their families require long-term understanding and support. Psychological therapy may be helpful in some cases.

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**5.12.E Upper gastroenterological disorders**

**Introduction**
Upper gastrointestinal disorders are not common complaints in the population presenting to hospitals in resource-limited countries. It may be that symptoms are under-reported or overlooked because of more common problems, such as gastroenteritis, persistent diarrhoea, intestinal helminths and malnutrition. However, certain life-threatening conditions do occur, including obstruction of the oesophagus due to a foreign body, strictures due to caustic soda poisoning, haematemesis due to peptic ulcer or portal hypertension, and volvulus due to malrotation.

In well-resourced communities, particularly where facilities for upper gastrointestinal paediatric endoscopy are available, similar symptoms to those that occur in well-resourced countries present. These include recurrent abdominal pain, epigastric and substernal pain, recurrent/persistent vomiting, dyspepsia and water-brash/heartburn.

**Gastro-oesophageal reflux**

<table>
<thead>
<tr>
<th>TABLE 5.12.E.1 Gastro-oesophageal reflux</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
</tr>
<tr>
<td>Vomiting (regurgitation)</td>
</tr>
<tr>
<td>Water-brash/heartburn</td>
</tr>
<tr>
<td>Nausea</td>
</tr>
<tr>
<td>Epigastric/substernal pain</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
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<tr>
<td></td>
</tr>
</tbody>
</table>

*Particularly in children with cerebral palsy.
Gastro-oesophageal reflux (GOR) is a normal physiological condition in infants, children and adults. If GOR is associated with complications (as below) then it is termed gastro-oesophageal reflux disease (GORD). GORD is common in children with cerebral palsy.

**Note:** Sandifer–Sutcliffe syndrome is dystonic posturing associated with GOR.

### Diagnosis

Often no investigations are needed and a diagnosis can be made by taking a good clinical history. The following investigations are helpful if they are needed and available:

- **Barium swallow:** This is often the only diagnostic facility available in resource-limited countries. It is a much less sensitive method for diagnosing reflux than pH monitoring, but will detect associated or other conditions such as oesophageal stricture, hiatus hernia, diaphragmatic hernia and malrotation.
- **Endoscopy and biopsy** (particularly looking for oesophagitis).
- **pH monitoring:** this grades the frequency and duration of exposure of the lower oesophagus to acid (pH < 4.0).

### Management

- Simple GOR in the thriving child: reassurance is all that is needed.
- Excessive regurgitation causing failure to thrive in an infant, or mild symptoms of oesophagitis: treatment by thickening feeds with Carobel (Cow & Gate) or an alginate preparation (e.g. Gaviscon) can be tried.
- Moderate to severe GOR with oesophagitis: H₂-receptor antagonists, such as ranitidine (2–4 mg/kg twice daily, maximum 150 mg twice daily) or the proton pump inhibitor, omeprazole (700 micrograms to 3 mg/kg once daily) should be given. Motility stimulants such as domperidone (200–400 micrograms/kg every 4–8 hours) may be effective, particularly in children with cerebral palsy. However, proof of their efficacy is lacking.
- Surgery: Nissen fundoplication would be considered if, despite medical management, there was severe oesophagitis, failure to thrive or aspiration pneumonia. It is sometimes required in children with cerebral palsy and GOR.

### Helicobacter pylori

*Helicobacter pylori* is a ubiquitous bacterium that commonly infects the stomach (especially the antrum) in children in resource-limited countries from an early age. Child-to-child transmission is important. In developed countries up to 40–60% of adults are infected, probably mainly during childhood. Conditions associated with *H. pylori* include the following:

- **Chronic gastritis:** often asymptomatic; not a major cause of abdominal pain in children.
- **Duodenal ulcer:** *H. pylori* has a strong association with duodenal ulcer and must be eradicated to ensure healing.

**Diagnosis**

Testing for *H. pylori* should only be undertaken if the child has symptoms of ulcer dyspepsia. Diagnostic tests (outlined below) are rarely available as routine in resource-limited countries.

- **Serology:** this is good for epidemiological studies, but has reduced sensitivity in children under 7 years of age.
- **Urea breath test (13C-UBT):** this is sensitive and specific, especially in children over 6 years of age.
- **Faecal antigen testing:** this is sensitive and specific in both children and adults.
- **Endoscopy:** histological demonstration and culture of *H. pylori*.

### Management

Selection of optimal antibacterial agents is difficult because of the development of resistance.

**Suggested regimen**

1. **Omeprazole**
   - Aged < 2 years: 700 micrograms/kg to 3 mg/kg once daily up to a maximum of 20 mg.
   - Aged > 2 years, body weight 10–20 kg: give 10 mg once daily up to a maximum 20 mg once daily.
   - Aged > 2 years, body weight > 20 kg: give 20 mg once daily up to a maximum of 40 mg once daily.
2. **plus antibiotics,** such as amoxicillin (< 1 year, 62.5 mg; 1–4 years, 125 mg; 5–12 years, 250 mg; > 12 years, 250–500 mg, three times daily)
3. **plus clarithromycin (7.5 mg/kg twice daily) or metronidazole (7.5 mg/kg three times daily).**

Treatment should be continued for 1–2 weeks. Strict compliance in order to avoid the development of resistance is imperative.

### Duodenal ulcer

Duodenal ulcers are uncommon in children, but can be life-threatening due to haematemesis, melaena and perforation. There is often a family history. Common symptoms include epigastric pain that typically:

- is worsened by fasting
- is improved by eating or antacids
- causes nocturnal waking.

**Diagnosis**

- **Endoscopy, including biopsy for *H. pylori,* is the optimal method.
- **Barium swallow:** this is less sensitive for diagnosing acute ulceration and better at detecting scarring.

**Management**

- Unless facilities to diagnose *H. pylori* are available, all children should be treated for eradication of presumed *H. pylori*.
- Give H₂ antagonists or a proton pump inhibitor for 6–8 weeks: ranitidine 2–4 mg/kg twice daily (maximum 150 mg twice daily); omeprazole 10 mg for 10–20 kg (can increase to 20 mg) and 20 mg for > 20 kg once daily (can increase to 40 mg).
5.12.F Gastrointestinal bleeding

**BOX 5.12.F.1 Minimum standards**
- Full blood count.
- Stool examination, microscopy and culture, parasite identification.
- Ultrasound.
- Barium X-ray studies.
- Gastroscopy.
- Colonoscopy.

**Introduction**
- The causes of bleeding from the gastrointestinal tract are many, and relate to the age of the child. A good history and clinical examination are essential and will indicate specific investigations.
- In haematemesis, it is important to exclude swallowed blood due to disorders of the nose and mouth.
- In children the commonest cause of fresh rectal bleeding is an anal fissure.
- Melena has to be differentiated from dark stools associated with medication (e.g. iron preparations) and colouring from foods.
- A large bleed from the upper gastrointestinal tract may present as red blood at the anus because of rapid transit.

<table>
<thead>
<tr>
<th>Site of bleeding</th>
<th>Clinical features/further information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper gut</td>
<td></td>
</tr>
<tr>
<td>Poisoning with or treatment with salicylates</td>
<td>‘Coffee-ground’ vomit</td>
</tr>
<tr>
<td>Mallory–Weiss syndrome</td>
<td></td>
</tr>
<tr>
<td>Oesophagitis, gastro-oesophageal reflux</td>
<td>See Section 5.12.E</td>
</tr>
<tr>
<td>Portal hypertension, oesophageal varices</td>
<td>See Section 5.7.B (liver disease)</td>
</tr>
<tr>
<td></td>
<td>See Section 6.3.C.c (schistosomiasis)</td>
</tr>
<tr>
<td>Midgut</td>
<td></td>
</tr>
<tr>
<td>Intussusception, volvulus</td>
<td>Infants (see Section 3.4 and 5.19)</td>
</tr>
<tr>
<td>Meckel’s diverticulum</td>
<td>Often symptomless</td>
</tr>
<tr>
<td>Colorectal</td>
<td></td>
</tr>
<tr>
<td>Infection (e.g. shigellosis, amoebiasis)</td>
<td>See Section 5.12.A and Section 6.3.B</td>
</tr>
<tr>
<td>Inflammatory bowel disease</td>
<td>Abdominal pain, diarrhoea, weight loss</td>
</tr>
<tr>
<td></td>
<td>See Section 5.12.D</td>
</tr>
<tr>
<td>Milk protein intolerance</td>
<td>See Section 5.12.G</td>
</tr>
<tr>
<td>Polyps (single, multiple, Peutz–Jeghers syndrome)</td>
<td>Blood separate from normal stool</td>
</tr>
<tr>
<td>Anus</td>
<td></td>
</tr>
<tr>
<td>Fissure</td>
<td>Infants, constipation, tags (see Section 5.12.C)</td>
</tr>
<tr>
<td>Crohn’s disease</td>
<td>See Section 5.12.D</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td></td>
</tr>
<tr>
<td>Necrotising enterocolitis (see Section 3.4), Henoch–Schoénlein purpura (see Section 5.13), AIDS (see Section 6.2.D)</td>
<td>Any coagulation or blood malignancy disorder (see Section 5.11.D and Section 5.14)</td>
</tr>
</tbody>
</table>

**Investigations**
The investigations chosen will depend on the suspected site of bleeding and the clinical features.
- See appropriate sections as indicated in the tables above.
- It is important to consider the following:
  - Stool:
    - Direct observation: blood, mucus.
    - Microscopy: Cryptosporidium, Salmonella, E. coli, Shigella, Campylobacter, ova, cysts and parasites.
    - Faecal occult blood.
  - Full blood count, grouping and cross-matching.
  - Serum ferritin and iron levels.
  - Isotope scan: diagnosis of Meckel’s diverticulum (30% false negative).
  - Barium studies: diagnosis of malrotation.
  - Ultrasound: diagnosis of intussusception.
  - Upper endoscopy: diagnosis and treatment of oesophageal, gastric and/or duodenal bleeding.
  - Colonoscopy: diagnosis and treatment of colitis and/or polyps.
TABLE 5.12.F.2 Features of gastrointestinal bleeding

<table>
<thead>
<tr>
<th>History/examination</th>
<th>Looking for:</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td></td>
</tr>
<tr>
<td>Acute/chronic, amount of blood</td>
<td>Severity</td>
</tr>
<tr>
<td>Endemic area</td>
<td>Schistosomiasis</td>
</tr>
<tr>
<td>Haematemesis</td>
<td>Upper gastrointestinal disorder</td>
</tr>
<tr>
<td>Nose and mouth lesions</td>
<td>Swallowed blood</td>
</tr>
<tr>
<td>Site of any pain</td>
<td>Upper or lower gastrointestinal tract</td>
</tr>
<tr>
<td>Stool:</td>
<td></td>
</tr>
<tr>
<td>Hard/loose</td>
<td>Constipation/diarrhoea</td>
</tr>
<tr>
<td>Blood mixed in stool</td>
<td>Inflammation/infection</td>
</tr>
<tr>
<td>Blood around or separate</td>
<td>Anal fissure/polyp</td>
</tr>
<tr>
<td>History/examination</td>
<td>Looking for:</td>
</tr>
<tr>
<td>Inflammatory bowel disease, family history</td>
<td>See Section 5.12.D</td>
</tr>
<tr>
<td>Bleeding tendency</td>
<td>Clotting disorder, malignancy</td>
</tr>
<tr>
<td>Examination</td>
<td></td>
</tr>
<tr>
<td>Nose and mouth lesions</td>
<td>Swallowed blood</td>
</tr>
<tr>
<td>Pallor, capillary refill, blood pressure</td>
<td>Anaemia, shock</td>
</tr>
<tr>
<td>Palpeches, telangiectasia</td>
<td>Thrombocytopenia, hereditary telangiectasia</td>
</tr>
<tr>
<td>Abdomen</td>
<td>Tenderness, hepatosplenomegaly</td>
</tr>
<tr>
<td>Anus</td>
<td>Fissure, tags, infection</td>
</tr>
</tbody>
</table>

5.12.G Malabsorption, including coeliac disease

Malabsorption

Malabsorption is an abnormality in absorption of food nutrients from the gastrointestinal tract.

Common causes of malabsorption and resultant failure to thrive in resource-limited countries include recurrent respiratory infection, persistent diarrhoea and HIV infection. None of these require bowel investigation. The main emphasis is on nutritional rehabilitation which regenerates the small bowel atrophy and the immune system (see management of persistent diarrhoea and severe malnutrition in Sections 5.12.B and 5.10.B, respectively). Only a limited response to nutritional support is expected in HIV infection, depending generally on the stage of disease and the response to antiretroviral (ARV) drugs.

Types of malabsorption

- **Selective**: as seen in lactose intolerance.
- **Partial**: as observed in Crohn’s disease and HIV infection.
- **Total**: as seen in coeliac disease.

Pathophysiology

The gastrointestinal tract functions to digest and absorb nutrients (fat, carbohydrate, protein and fibre), micronutrients (vitamins and trace minerals), water and electrolytes. This is dependent on the proper processing of food by mechanical (chewing and gastric churning) and enzymatic (gastric, pancreatic, biliary or intestinal) means. The final products of digestion are then absorbed through the intestinal epithelial cells.

Malabsorption constitutes the pathological breakdown of the normal physiological sequence of digestion (i.e. intraluminal process), absorption (i.e. mucosal process) and transport (post-mucosal events) of nutrients.

Clinical features

Symptoms can be intestinal or extra-intestinal, and include the following:

- diarrhoea/statorrhoea: watery, diurnal and nocturnal, bulky, frequent stools
- bloating
- flatulence
- abdominal discomfort/cramping abdominal pain
- growth retardation
- weight loss
- failure to thrive
- delayed puberty
- swelling or oedema from loss of protein
- anaemia (vitamin B12, folic acid and iron deficiency)
- fatigue
- weakness
- muscle cramp
- osteomalacia and osteoporosis
- bleeding tendencies.

Diagnosis

Investigation is guided by symptoms and signs. Since a range of different conditions can produce malabsorption, it is necessary to look for each of these specifically. Tests are also needed to detect the systemic effects of deficiency of the malabsorbed nutrients (e.g. anaemia with vitamin B12 malabsorption).

Investigations may include the following:

- full blood count and blood film
- C-reactive protein and erythrocyte sedimentation rate
- serum albumin
- serum iron, ferritin and total iron-binding capacity (TIBC)
- serum folic acid
- serum cholesterol or triglyceride
- serum calcium, phosphate and alkaline phosphatase
- prothrombin time and activated partial thromboplastin time
- blood chemistry (electrolytes, glucose, HCO3–, urea and creatinine)
- serum zinc levels
- stool studies, including cultures.
Section 5.12

Serological studies
The following specific tests are carried out to determine the underlying cause:
- IgA anti-transglutaminase antibodies
- IgA anti-endomysial antibodies

Radiological studies
- Barium meal and follow-through,
- Barium enema,
- CT of the abdomen.

Specialised tests (if available)
- Biopsy of small bowel,
- Colonoscopy can be helpful in colonic and ileal disease,
- Endoscopic retrograde cholangiopancreatography (ERCP) will show pancreatic and biliary structural abnormalities,
- Glucose hydrogen breath test for bacterial overgrowth.

Management
Treatment is directed largely towards management of the underlying cause. In severe nutritional deficiency, hospital admission may be required for total parenteral nutrition (TPN). Subsequently, advice and support from a dietitian is vital.

Coeliac disease

Coeliac disease is an autoimmune disorder of the small intestine in genetically predisposed people of all ages from middle infancy onwards. It is caused by a reaction to gliadin, a gluten protein found in wheat and similar cereals. Therefore it is common among populations whose diet contains substantial amounts of wheat. Apart from people of European origin, in whom it commonly manifests, it is also frequently seen in North Africa, the Middle East, and the north of the Indian subcontinent where wheat is a staple diet. Other populations at increased risk for coeliac disease include children with Down’s syndrome and Turner syndrome, type 1 diabetes and autoimmune thyroid disease, including both hyperthyroidism and hypothyroidism.

Pathophysiology
Upon exposure to gliadin, the enzyme tissue transglutaminase (tTG) modifies the immune system to cross-react with the small-bowel villous lining, causing an inflammatory reaction. This leads to villous atrophy, which interferes with the absorption of nutrients, minerals and the fat-soluble vitamins A, D, E and K.

Coeliac disease appears to be polyfactorial. Almost all people with coeliac disease have either the HLA-DQ2 or the HLA-DQ8 allele. However, additional factors are needed for coeliac disease to manifest besides the HLA risk alleles. Furthermore, around 5% of those people who do develop coeliac disease may not have typical HLA-DQ2 or HLA-DQ8 alleles.

TABLE 5.12.G.1 Common causes of malabsorption

<table>
<thead>
<tr>
<th>Common causes of malabsorption</th>
<th>Common causes of malabsorption</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Due to infective agents</strong></td>
<td>Intestinal tuberculosis</td>
</tr>
<tr>
<td></td>
<td>HIV-related malabsorption</td>
</tr>
<tr>
<td></td>
<td>Tropical sprue</td>
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<tr>
<td></td>
<td>Traveller’s diarrhoea</td>
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<tr>
<td></td>
<td>Parasites, such as Giardia lamblia, fish tapeworm, roundworm, hookworm (Ancylostoma duodenale and Necator americanus)</td>
</tr>
<tr>
<td><strong>Due to structural defects</strong></td>
<td>Blind loops</td>
</tr>
<tr>
<td></td>
<td>Inflammatory bowel diseases (e.g. Crohn’s disease)</td>
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<td></td>
<td>Intestinal hurry from surgical procedures (e.g. post-gastrectomy, gastro-jejunostomy)</td>
</tr>
<tr>
<td></td>
<td>Fistulae, diverticulae and strictures</td>
</tr>
<tr>
<td></td>
<td>Short bowel syndrome</td>
</tr>
<tr>
<td><strong>Due to mucosal abnormality</strong></td>
<td>Coeliac disease</td>
</tr>
<tr>
<td></td>
<td>Cow’s milk intolerance</td>
</tr>
<tr>
<td></td>
<td>Soya milk intolerance</td>
</tr>
<tr>
<td><strong>Due to enzyme deficiencies</strong></td>
<td>Lactose intolerance (constitutional, secondary or rarely congenital)</td>
</tr>
<tr>
<td></td>
<td>Sucrose intolerance</td>
</tr>
<tr>
<td><strong>Due to digestive failure</strong></td>
<td>Pancreatic insufficiencies</td>
</tr>
<tr>
<td></td>
<td>Cystic fibrosis</td>
</tr>
<tr>
<td></td>
<td>Chronic pancreatitis</td>
</tr>
<tr>
<td></td>
<td>Bile salt malabsorption</td>
</tr>
<tr>
<td></td>
<td>Terminal ileal disease</td>
</tr>
<tr>
<td></td>
<td>Obstructive jaundice</td>
</tr>
<tr>
<td></td>
<td>Bacterial overgrowth</td>
</tr>
<tr>
<td><strong>Due to systemic diseases</strong></td>
<td>Coeliac disease</td>
</tr>
<tr>
<td></td>
<td>Hypothyroidism and hyperthyroidism</td>
</tr>
<tr>
<td></td>
<td>Addison’s disease</td>
</tr>
<tr>
<td></td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td></td>
<td>Hyperparathyroidism and hypoparathyroidism</td>
</tr>
<tr>
<td></td>
<td>Malnutrition</td>
</tr>
</tbody>
</table>

Serological studies
The following specific tests are carried out to determine the underlying cause:
- Lactose hydrogen breath test for lactose intolerance.
- Magnetic resonance cholangiopancreatography (MRCP).


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Clinical features
Clinical features may range from severe to almost nonexistent. Severe coeliac disease in young children leads to the characteristic symptoms of pale, loose and greasy stools (steatorrhoea) with weight loss or failure to gain weight. Adolescents and older children with milder coeliac disease may have symptoms that are much more subtle and occur in other organs rather than in the bowel itself.

TABLE 5.12.G.2 Clinical features of coeliac disease

<table>
<thead>
<tr>
<th>Under 2 years of age</th>
<th>Over 2 years of age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vomiting</td>
<td>Delayed puberty</td>
</tr>
<tr>
<td>Abdominal distension</td>
<td>Iron-resistant anaemia</td>
</tr>
<tr>
<td>Irritability</td>
<td>Rickets/osteomalacia</td>
</tr>
<tr>
<td>Anorexia</td>
<td>Behaviour problems</td>
</tr>
<tr>
<td>Growth failure</td>
<td>With or without the gut disorders that occur in younger children</td>
</tr>
</tbody>
</table>

Diagnosis
The diagnosis of coeliac disease is based on two types of testing.

Serological blood tests
These are the first-line investigation and include the following:

- **IgA anti-tissue transglutaminase (tTG) antibodies:** this test is reported to have a high sensitivity (99%) and specificity (over 90%) for identifying coeliac disease. Therefore it should be done first. It is also an easier test to perform. An equivocal result on tTG testing should be followed by antibodies to endomysium.

- **IgA anti-endomysial antibodies:** this test has a sensitivity and specificity of 90% and 99%, respectively, for detecting coeliac disease.

It is important that the total serum IgA level is also checked, as coeliac patients with IgA deficiency may be unable to produce the antibodies on which these tests depend (‘false-negative’). In such patients, IgG antibodies against transglutaminase (IgG-1TG) or IgG anti-gliadin antibodies (IgG-AGA) may be helpful in reaching a diagnosis.

Dudeno-jejunal biopsies
Because of the implications of a diagnosis of coeliac disease, guidelines recommend that a positive serological blood test may still be followed by a dudeno-jejunal biopsy. Similarly, a negative serology may still be followed by a recommendation for a biopsy if clinical suspicion remains high. Tissue biopsy is still considered the gold standard in the diagnosis of coeliac disease.

For this purpose, biopsies can be obtained using metal capsules attached to a suction device. The capsule is swallowed and allowed to pass into the small intestine. After X-ray verification of its position, suction is applied to collect part of the intestinal wall inside the capsule. Commonly used capsule systems are the Watson capsule and the Crosby–Kugler capsule. This method has now been largely replaced by fibre-optic endoscopy, which carries a higher sensitivity and a lower frequency of errors.

Initially, take a gluten-free diet and perform a re-challenge with gluten-containing food over 2–6 weeks before repeating the investigations.

A histology compatible with coeliac disease on a gluten-containing diet, followed by a clinical improvement (i.e. gain in weight and height and resolution of symptoms) once the gluten is removed from the diet is often enough to establish the diagnosis. Most guidelines do not recommend a repeat biopsy unless there is no improvement in the symptoms on the gluten-free diet. In some cases a deliberate gluten challenge, followed by biopsy, may be conducted to confirm or refute the diagnosis. A normal biopsy and normal serology after the challenge indicates that the diagnosis may have been incorrect.

In resource-limited countries where facilities for biopsies may not exist, the same model can be used with serological tests. A positive serological test on a gluten-containing diet will revert to normal with clinical improvement once the patient is on a gluten-free diet.

TABLE 5.12.G.3 Investigations for malabsorption

<table>
<thead>
<tr>
<th>General</th>
<th>Specific</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full blood count plus film</td>
<td>Immunoglobulins IgA and IgG</td>
</tr>
<tr>
<td>Serum iron and ferritin</td>
<td>IgA anti-endomyosal antibodies</td>
</tr>
<tr>
<td>Folate (red blood cell)</td>
<td>IgA anti-gliadin (AGA)</td>
</tr>
<tr>
<td>Vitamin B12 levels</td>
<td>Small bowel biopsy:</td>
</tr>
<tr>
<td>Serum albumin</td>
<td>• Villous atrophy</td>
</tr>
<tr>
<td>Hydrogen breath test</td>
<td>• Hyperplasia of crypts</td>
</tr>
<tr>
<td>Serum calcium, phosphate and alkaline phosphatase</td>
<td>• Increased inflammatory cells</td>
</tr>
</tbody>
</table>

There are several ways in which these tests can be used to assist in diagnosing coeliac disease. However, all tests become invalid if the patient is already taking a gluten-free diet. Intestinal damage begins to heal within weeks of gluten being removed from the diet, and antibody levels decline over a period of months. In such cases it may be necessary to perform a re-challenge with gluten-containing food over 2–6 weeks before repeating the investigations.

A histology compatible with coeliac disease on a gluten-containing diet, followed by a clinical improvement (i.e. gain in weight and height and resolution of symptoms) once the gluten is removed from the diet is often enough to establish the diagnosis. Most guidelines do not recommend a repeat biopsy unless there is no improvement in the symptoms on the gluten-free diet. In some cases a deliberate gluten challenge, followed by biopsy, may be conducted to confirm or refute the diagnosis. A normal biopsy and normal serology after the challenge indicates that the diagnosis may have been incorrect.

In resource-limited countries where facilities for biopsies may not exist, the same model can be used with serological tests. A positive serological test on a gluten-containing diet will revert to normal with clinical improvement once the patient is on a gluten-free diet.

**FIGURE 5.12.G.2 Diagnosis of coeliac disease.**

Initial diagnosis
Based on clinical signs and symptoms and biopsy shows atrophic mucosa or increased tTG antibody levels

Gluten-free diet
Clinical remission (within weeks) and decreased tTG antibody levels

COELIAC DISEASE
Section 5.13

5.13 Rheumatology

Box 5.13.1 Minimum Standards
- Penicillin.
- Aspirin.
- Prednisolone.
- Haloperidol, diazepam and lorazepam.
- Anti-endocarditis measures.
- IV gamma globulin if at all possible.
- Non-steroidal anti-inflammatory drugs (NSAIDs).
- Sulphasalazine.
- Ocular steroids and mydriatics.
- Intra-articular steroids.
- Physiotherapy and family support.

Introduction
Making a diagnosis of a rheumatic disease in a child relies primarily on clinical skill and experience, as there are few diagnostic laboratory tests. Although these diseases are rare in children, symptoms that raise the possibility of rheumatic disease are common. Rheumatic symptoms may be relatively specific, such as joint swelling, or relatively non-specific, such as fever, lethargy, pallor, anorexia, failure to thrive, muscle weakness, musculoskeletal pain, rash, headache and abdominal pain. The interpretation of these clinical features requires a meticulous approach to characterising the nature of each feature and considering the overall pattern of all the clinical features in the individual patient. The aims of this section are to assist in the recognition of common patterns of clinical features, and to provide guidance for appropriate treatment and monitoring of rheumatic disease in children.

pGALS (paediatric Gait, Arms, Legs and Spine) is a simple quick approach to joint examination and helps to discern abnormal from normal joints; this is especially useful in the context of non-specific features such as limp or fever. pGALS includes incorporates a series of simple manoeuvres to assess all joints quickly (takes approximately 2–3 minutes). It has been validated in school-aged children (although can be performed in younger children) and has been shown to be effective when performed by non-specialists in detecting significant joint abnormalities in acute paediatric practice (including in Africa). The interpretation of pGALS requires knowledge of normal musculoskeletal development and the clinical context to facilitate a differential diagnosis (www.arthritisresearchuk.org/health-professionals-and-students/video-resources/pgals.aspx).

Rheumatic Fever
Rheumatic fever is an abnormal immune response to group A streptococcal infection in genetically susceptible individuals. It is most common between the ages of 6 and 16 years. Symptoms of acute rheumatic fever follow streptococcal pharyngitis after a latent period of approximately 3 weeks. The disease usually presents with joint pain, but may have an insidious onset, especially if carditis is the predominant feature. There is no definitive test, and diagnosis depends on recognition of clinical signs known as the Jones criteria.