

Gastroenterology

Vomiting

CAUSES

Gastrointestinal

1. Reflux: 'normal' possetting:
 - significant GOR
 - hiatus hernia.
2. Infective: viral gastroenteritis:
 - bacterial infection: see Box 9.1
 - toxin food poisoning.
3. Immunological: coeliac disease:
 - cow's milk intolerance
 - other specific food allergies (e.g. fish, strawberries).
4. Inflammatory: appendicitis:
 - mesenteric adenitis.
5. Obstructive: pyloric stenosis:
 - intussusception
 - volvulus
 - strangulated hernia

Box 9.1 Infective causes of diarrhoea and vomiting

Viruses

Rotavirus
Adenovirus
Coronavirus
Astrovirus
Calicivirus
Parvovirus
Echovirus
Following oral polio vaccine

Bacteria

Enteroinvasive *E. coli*
Campylobacter jejuni
Salmonellosis (esp. *S. typhimurium*)
S. typhi and *S. paratyphi*
Shigella (usually *Sh. sonnei*)
Vibrio cholera
Yersinia enterocolitica

Protozoa

Giardia lamblia
Cryptosporidium
Entamoeba histolytica
Malaria

Bacterial toxins (usually 'food poisoning')

Enterotoxigenic *E. coli*
Staph. aureus
Bacillus cereus
Clostridium

- inguinal
- umbilical
- epigastric
- Hirschsprung's disease
- tumour
- post-operative ileus.

Systemic

1. Infective: any febrile illness:
 - UTI in infants
 - following paroxysms of whooping cough
 - acute hepatitis.
2. Neurological:
 - increased ICP:
 - trauma
 - meningitis or abscess
 - tumour
 - migraine.
3. Metabolic: diabetic ketoacidosis:
 - Reye syndrome
 - many inborn errors of metabolism.
4. Ingestion: drugs (even at therapeutic doses):
 - poisons
 - post GA.

ASSESSMENT

History

A dietary/feeding history is paramount:

- Has vomiting been a problem since birth?
- Does vomiting only occur soon after food?
- Has vomiting only been a problem since weaning or the recent introduction of a new food into the diet?
- Is there diarrhoea?
- Do other family members have vomiting or diarrhoea?
- Has the child or a family member been abroad (within the last year)?
- Is the vomit green (Fig. 9.1)?

Constipation occurs in many febrile illnesses but is also a feature of obstruction. **Steatorrhoea** (smelly stools, difficult to flush) suggests malabsorption. **Abdominal pain** is not specific for a surgical cause of vomiting.

Pain also occurs in:

- gastroenteritis
- peptic ulceration and oesophagitis
- renal tract infection (ask about dysuria, haematuria, enuresis)
- hepatitis (travel abroad or recent contacts)
- migraine (family history, or history of recurrent abdominal pain)

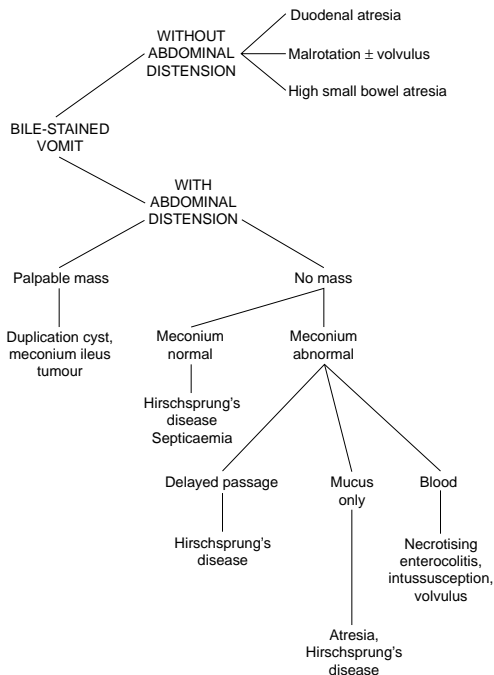


Fig. 9.1 Green vomit in an infant suggests mechanical obstruction until proved otherwise. In duodenal obstruction, stools may not be pale because of a Y-termination of the common bile duct (10%).

- diabetic ketoacidosis (polydipsia, polyuria, weight loss)
- iron ingestion (ask about drugs in the house or at the grandparents').

Examination

A full examination is necessary in all children. Look for fever (infection), anaemia (acute or chronic blood loss or chronic malabsorption), jaundice (pyloric stenosis, hepatitis).

1. Abdominal distension may occur in:

- chronic constipation
- gastroenteritis

- coeliac disease
- obstruction
- ileus.

Only in the latter case are bowel sounds diminished.

2. Tenderness is more likely to be the result of a surgical cause the more localised it is, the more reproducible it is, and the further from the umbilicus it is.
3. Don't forget the hernial orifices and genitalia.
4. Rectal examination to look for:
 - fissures exacerbating constipation
 - lax sphincter and loaded rectum of chronic constipation. Faecal 'rocks' may also be felt per abdomen, especially on the left side
 - tight sphincter and empty rectum of Hirschsprung's disease.
5. Whenever possible, examine the stools for blood, diarrhoea or steatorrhoea. Test the urine for blood or protein: if positive, urgent microscopy is required.
6. If an abdominal cause seems unlikely, look at the ears, throat and fundi.
7. Measure the height and weight of the child for evidence of a chronic problem.

Investigations

If the cause of vomiting is still not clear, consider:

- urine microscopy and culture
- FBC
- U & E for evidence of dehydration, hyponatraemia or hypokalaemic alkalosis (pyloric stenosis)
- blood sugar
- blood cultures if febrile
- stool specimens for both viral and bacterial culture
- erect and supine AXR if there is localised tenderness or distention and constipation. Air–fluid levels may be seen in:
 - gastroenteritis
 - malabsorption
 - obstruction
 - ileus
- in an infant between 2 and 10 weeks, consider a **test feed** to look for pyloric stenosis, even if the vomiting is not projectile.

Look for visible peristalsis and **feel** (from the left side, palpate the right upper quadrant immediately lateral to the rectus sheath) for a pea-sized pyloric mass. Often this mass is felt best immediately after a vomit when the muscle is in spasm.

NB. One negative test feed or normal biochemistry does **not** exclude this diagnosis. Ultrasound examination, in experienced hands, may be very helpful in making the diagnosis.

MANAGEMENT

Treat the cause if possible. However, two common and non-specific presentations which the junior doctor frequently has to manage are:

1. **Chronic vomiting in a baby** presenting to casualty or out-patients. Exclude cleft palate, Pierre Robin sequence and neurological abnormality.
 - If the infant is well, growing along a centile, and the vomits are small, reassure the parents and explain that reflux will improve with time. If vomiting is still a problem at follow-up, try:
 - Carobel 1 scoop to 100 ml milk
 - infant Gaviscon 1 dose ($\frac{1}{2}$ dual sachet) < 4.5 kg; 2 doses (1 dual sachet) > 4.5 kg with feeds
 - earlier introduction of solids and propping upright for 1 hour after feeds.
 - If the infant is falling away from the centiles, despite adequate intake, and no cause can be found, start Carobel and add infant Gaviscon to every feed but if the child has not regained the centile after 6 weeks of treatment, seek the advice of a more senior colleague or arrange admission. A barium swallow or oesophageal pH monitoring may elucidate a cause:
 - oesophagitis secondary to severe reflux
 - true sliding hiatus hernia
 - small tracheo-oesophageal fistula
 - oesophageal stricture
 - achalasia
 - vascular ring or mediastinal mass causing compression.
2. **Acute diarrhoea and vomiting.** Few children with gastroenteritis require hospital admission. If the child is < 10% dehydrated, stop all foods and drinks and give a water/dextrose/electrolyte mixture (e.g. Dextrolyte or Dioralyte) orally and frequently using volumes calculated as on p. 22. If there is no vomiting after 4 hours of rehydration with this therapy, restart normal diet but continue to give supplementary drink of Dioralyte with each loose motion in a volume of 10 ml/kg. Reintroduction of milk (or a light diet in an older child) should be based on cessation of vomiting, not diarrhoea.

Advise the parents that:

 - loose stools may continue for 2 weeks
 - the child is infectious to others
 - scrupulous handwashing is essential while diarrhoea persists
 - a breastfeeding mother must express to maintain her milk supply while the infant is restricted to the electrolyte/dextrose mixture.

If the child is $\geq 10\%$ dehydrated, admit for rehydration and measure electrolytes. Intravenous rehydration is essential in the very ill and severely dehydrated child (see Ch. 3) but should otherwise be reserved for children who have failed a 6-hour trial of water/dextrose/electrolyte mixture.

Diarrhoea

It is difficult to define an absolute threshold of normality; breast-fed infants have looser and more frequent stools, sometimes after every feed.

A. ACUTE DIARRHOEA

Whether or not there is vomiting, the commonest cause is gastroenteritis (Box 9.1). Fever may accompany gastroenteritis.

1. Gastrointestinal bleeding

Blood in the stools strongly suggests one of:

- *Campylobacter jejuni*
- shigella
- amoebae
- intussusception (3 months–3 years)
- HUS
- Meckel's diverticulum
- ulcerative colitis.

Small haematemeses, relatively common after vomiting, are presumably due to a small Mallory–Weiss tear or gastritis. Check Hb and clotting but no further investigation.

2. Fluid replacement

For the supportive treatment of gastroenteritis, see the sections on vomiting (p. 145) and on fluid balance in Ch. 3 (p. 22).

3. Specific chemotherapy

Kaolin or antispasmodics should be discouraged and antibiotics are only indicated as follows:

- erythromycin or ciprofloxacin for prolonged *C. jejuni* infection
- ciprofloxacin or trimethoprim for *Salmonella* bacteraemia or severe *Shigella*
- metronidazole for *Giardia lamblia* or *Entamoeba histolytica*
- oral vancomycin for *Clostridium difficile*.

4. Prevention

Advise the parents of measures to reduce cross-infection in the home. If the child is admitted, barrier nurse in an isolation cubicle.

5. Complications

- Recurrence of diarrhoea: warn the parents that loose stools may persist for 2 weeks.
- Diarrhoea persisting beyond 2 weeks:
 - transient lactose intolerance can occur after gastroenteritis. Stool is positive with 'Clinitest' tablets (i.e. reducing substances). Try a lactose-free milk (e.g. Pregestimil or Wysoy). There may be a generalised disaccharidase deficiency, in which case Pregestimil is better
 - transient protein intolerance. Normal stool pH and no reducing substances. Try a hydrolysed casein milk (Pregestimil) or soya-based milk (e.g. Wysoy or Formula S). Soya-protein intolerance may co-exist with cow's milk intolerance.

Transient intolerances tend only to last for a few weeks and normal diet should be resumed after this period.

B. CHRONIC DIARRHOEA (Box 9.2)

Distinguish between **faecal overflow** (see p. 157) and **true diarrhoea** by abdominal and rectal examination. A history of very frequent watery stools or soiling are both clues to spurious diarrhoea and abdominal and rectal examination looking for a megarectum full of faeces may be useful in distinguishing between this and primary diarrhoea.

Inflammatory bowel disease is rare, even in older children and adolescents.

If growth is normal, **toddler diarrhoea** and **post-gastroenteritis/giardiasis** should be distinguishable.

This leaves children with **malabsorption**, essentially coeliac disease, versus 'the rest'.

COELIAC DISEASE (gluten-sensitive enteropathy)

History

Anorexia, abdominal pain, vomiting and frequent, smelly, pale stools. Onset of symptoms after introduction of solids (usually 4–6 months; wheat, barley, rye and oats all contain gluten; corn and rice are non-toxic).

Examination

- Anaemia.
- Short stature.
- Muscle wasting (buttocks most obvious).
- Distended abdomen.
- Dermatitis herpetiformis (itchy vesicular rash, genitalia or buttocks, usually > 4 years).

Box 9.2 Causes of chronic diarrhoea

Non-specific diarrhoea	Malabsorption	Inflammatory bowel disease
Normal growth Well child	Abnormal growth Anaemia Rickets Muscle wasting/hypotonia	Abdominal symptoms Bloody diarrhoea
1. <i>Irritable bowel syndrome</i> = 'toddler diarrhoea' Onset 6 months–2 years 'Peas and carrots' stools Rarely persists into school age Exaggerated gastrocolic reflex	1. <i>Mucosal abnormality</i> Post gastroenteritis Cow's milk or soya milk protein intolerance Carbohydrate intolerance Coeliac disease Giardiasis Post cytotoxic drugs	1. <i>Crohn's disease</i> 2. <i>Ulcerative colitis</i> 3. Chronic <i>Campylobacter</i> infection or dysentery
2. <i>Post gastroenteritis</i> See above	2. <i>Pancreatic abnormality</i> CF	
3. <i>Giardiasis</i> Positive stool microscopy or duodenal aspirate	3. <i>Structural abnormality</i> Congenital lymphangiectasia Blind loops Malrotation Tumours (especially lymphoma)	
	4. <i>Immunodeficiency</i> Hypogammaglobulinaemia Severe combined immunodeficiency syndrome	
	5. <i>Other inborn errors of absorption</i> Acrodermatitis enteropathica (zinc deficiency) Congenital chloridorrhoea	

Investigation

- Full blood count for evidence of anaemia.
- Total IgA level with endomysial or tissue transglutaminase antibody titre. Approximately 5% of people with coeliac disease are IgA deficient and a severe deficiency may produce falsely negative antibodies, hence the importance of measuring the total IgA level as well as the antibody titre.
- Small bowel biopsy. The definitive test is now carried out in most centres by endoscopic duodenal biopsies obtained usually under general anaesthesia. This should be deferred only if the child is very ill or clotting is abnormal.

Differential diagnosis of abnormal small bowel biopsy (Box 9.3).

The characteristic histological features in coeliac disease are:

- varying degrees of villous atrophy
- increased intraepithelial lymphocytes
- crypt hyperplasia.

Box 9.3 Causes of an abnormal jejunal biopsy

Villous atrophy

Coeliac disease
 Temporary gluten intolerance
 Cow's milk protein intolerance
 Soya protein intolerance
 Gastroenteritis
 Giardiasis
 Severe combined immunodeficiency
 Cytotoxic chemotherapy

Other abnormalities

Agammaglobulinaemia (no plasma cells)
 Lymphangiectasis (dilated mucosal lymphatics)
 Abetalipoproteinaemia (distention of epithelial cells by fat)
 Disaccharidase deficiency (absent on histochemical staining)

Giardia may be seen on microscopy (or organisms cultured) in gastroenteritis or blind-loop syndrome.

The differential diagnoses of abnormalities not in this characteristic pattern are given in Box 9.3. It is no longer recommended that three jejunal biopsies are obtained on and off the gluten-free diet in order to confirm the diagnosis. It is now accepted that a single abnormal biopsy at the time of diagnosis is adequate except in the following circumstances:

- age under 2 years when there may be a chance of transient gluten intolerance of infancy (although this is probably a rare phenomenon)
- when there is any doubt at all about the original diagnosis (this may be because the histological picture was not classical or because the clinical presentation is slightly unusual or the serological evidence is not strong)
- when the response to diet is not as expected.

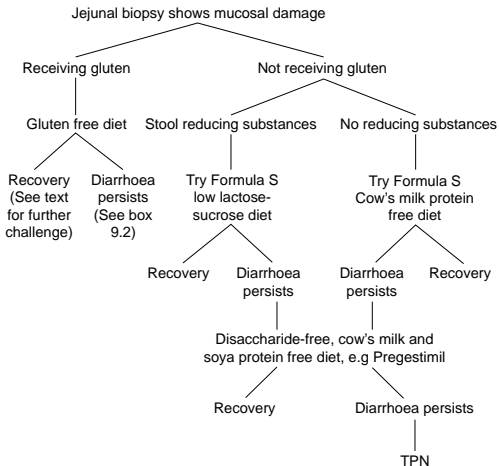
Management

Abnormal biopsy: follow the scheme in Fig. 9.2.

Normal biopsy: other investigations are necessary.

Failure to thrive

- Distinguish from acute weight loss, usually due to dehydration.
- The cause of FTT is chronic and more often there is inadequate weight gain rather than actual loss. Hence, the child 'falls away' on a centile chart. Serial weights over 3 months are more reliable than single absolute measurements.

**Notes:**

1. The very ill child may need immediate resuscitation with plasma/blood transfusion and TPN until well enough for a trial of diet.
2. Introduce each new preparation by a graded increase in concentration. Diarrhoea may persist for several weeks. Try each stage for at least 1 week. Ideally, recovery should be proved by repeat biopsy.
3. Reintroduce potential provoking nutrients in the order lactose, protein, (soya/cow's milk) gluten.

Fig. 9.2 Trial of diets for chronic diarrhoea. Specific vitamin and mineral supplements may also be required.

- A common pitfall is that birthweight is an unreliable predictor of future weight and 'physiological down regulation' is common in the first 12 months of life.
- In the older child, decreased height velocity is a more sensitive indicator of FTT than weight.

CAUSES

Almost any chronic paediatric condition can result in FTT but all causes act via one or more of the mechanisms in Box 9.4. In over half the cases, the cause is poor dietary intake.

Box 9.4 Causes of failure to thrive*

Inadequate diet offered

Too little offered

Not offered often enough

Offered but deficient in calories, protein or vitamins

Inadequate intake

The 'fussy eater'

Anorexia through an organic cause, e.g. coeliac disease

Cardiovascular, respiratory or neurological disease may render intake difficult despite good appetite

Vomiting

Diet is taken but not absorbed.

Severe reflux has the same consequence and may additionally cause oesophagitis

Chronic vomiting or reflux will lead to loss of appetite also

Malabsorption

Diet is taken in sufficient quantities but not absorbed: specific mucosal or exocrine problem

Increased requirements

Cardiac failure, respiratory failure and thyrotoxicosis result in increased basal energy expenditure

Decreased utilisation

Many dysmorphic children fail to thrive even with adequate intake. Endocrine FTT is due to inadequate utilisation of diet

*Several may occur simultaneously, e.g. CF.

HISTORY

- Detailed history of the onset of FTT and growth prior to introduction of solids.
- Past medical history.
- Family history of short stature, CF, coeliac disease, etc. Social circumstances: who feeds the child?
- Pregnancy history (congenital infection), gestation and birth-weight.
- Detailed dietary history, both offered and taken. The mean milk intake of thriving babies is > 150 ml/kg (2.5 oz/lb) per 24 hours during the first 6 months, with feeds at 3–6-hour intervals.

The diet of older children should be scrutinised by a dietitian for calories, protein, vitamins, iron and Ca^{2+} intake.

- Developmental milestones: motor delay is common.
- Ask specifically about:
 - vomiting
 - diarrhoea

- abdominal pain
- shortness of breath, chronic cough (recurrent aspiration, CF)
- tiredness/cyanosis/sweating on feeding (cardiac failure)
- urinary frequency or excessive thirst (UTI, diabetes mellitus or diabetes insipidus).

EXAMINATION

- Measure height, weight and OFC and plot on an appropriate centile chart using the child's decimal age.
- Height of parents and siblings (see Ch. 5, p. 50).
- Complete physical examination, including mouth, BP and fundoscopy.
- Stool inspection and urinalysis.

The features in Box 9.5 suggest the child is constitutionally small. Serial weights should continue for 6 months but no other action is necessary.

If the child is failing to thrive, but is well, simple dietary advice may suffice. However, if at follow-up 6 weeks later there has been no 'catch-up', admit for a **trial of feeding** (Fig. 9.3).

A successful trial effectively excludes an organic cause for FTT and often requires hospital admission; 4 weeks may be needed to demonstrate the improvement effectively. If the trial is unsuccessful, the period of inpatient observation may direct the nature of further investigations (see Fig. 9.2 and Table 9.1).

Box 9.5 The constitutionally small child

1. Asymptomatic
2. Low birthweight for gestational age
3. Proportionally small (height, weight and OFC centiles similar)
Asymmetrical growth patterns suggest particular causes:
 - (a) OFC centile > height > weight suggests third trimester IUGR (usually followed by 'catch up' growth if diet is adequate) or nutritional FTT
 - (b) OFC centile = weight > height suggests endocrine FTT
4. Normal height and weight velocities (i.e. growing parallel to but below the third centile)
5. Small parents

If not, look for evidence of:

- congenital infection
 - fetal alcohol syndrome
 - chromosomal abnormality
 - dysmorphic features
 - skeletal dysplasia
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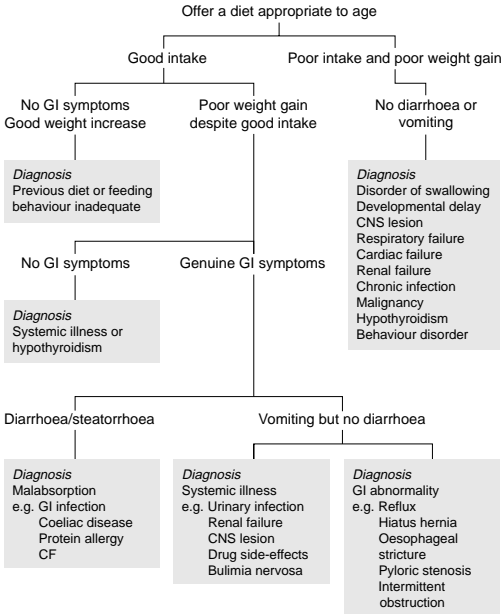


Fig. 9.3 Trial of feeding for failure to thrive.

Gastrointestinal bleeding

In over 50% of cases in childhood, no specific cause is found.

A cause should be sought more vigorously in either the newborn (vitamin K deficiency) or an older child (portal hypertension and oesophageal varices), or if the bleeding is severe.

Haematemesis is more common than melaena, and if a small haematemesis occurs after previous bloodless vomiting, the cause is probably a small Mallory–Weiss tear. In the newborn, haematemesis may be swallowed maternal blood, and in an older child, the result of a nosebleed.

Table 9.1 Investigation of failure to thrive

	Investigation	Probable diagnosis
Trial of normal diet in hospital for 2–4 weeks	Daily weights	Deprivation
	Inspect stools	
<i>Stools</i>	Microscopy	Cysts of <i>Giardia</i> or <i>Amoeba</i> . Pathogenic <i>E. coli</i> , <i>Shigella</i> , <i>Salmonella</i>
	Reducing substances pH < 5.5	Disaccharidase deficiency
	Chymotrypsin or Pancreatic elastase	Pancreatic disorder
<i>Urine</i>	Culture	UTI
	Glycosuria	Diabetes mellitus
	Proteinuria/haematuria	Renal failure
	Reducing substances other than glucose (Clinitest positive, Clinistix negative)	Galactosaemia
	Osmolality < 200 mosmol/l	Diabetes insipidus
	Specific gravity < 1.005	
	Aminoacid and organic acid chromatography Vanil mandelic acid	Amino or organic aciduria Neuroblastoma
<i>Sweat</i>	Sweat chloride > 60 mmol/l (40–60 mmol/l = equivocal result). Must have at least 100 mg of sweat. If doubt exists send blood for genotype (NB : sweat test negative CF does occur).	CF
<i>Blood</i>	Hb, WBC, platelets and film	Anaemia
	Folate (serum and RBC)	Schwachmann's syndrome
		Leukaemia
	Iron/ferritin/transferrin/TIBC	Iron deficiency
	Electrolytes	Chronic vomiting
		Diabetes insipidus
		Chloridorrhoea
	HCO ₃ ⁻	Renal tubular disorder
	Creatinine	Renal failure
	Ca ²⁺ , PO ₄ ³⁻ , alkaline phosphatase	Rickets
Conjugated bilirubin, liver function tests, albumin	Biliary or liver disease	
Clotting studies	Dietary deficiency, malabsorption or liver disease	

Table 9.1 (contd)

	Investigation	Probable diagnosis
	Thyroid function	Hypo/hyperthyroidism
	GH	Hypopituitarism
	Cholesterol	Abetalipoproteinaemia
	Immunoglobulins	Hypogammaglobulinaemia
	CF genotype	CF
	Hypoproteinaemia	Dietary deficiency, liver disease, nephrotic or protein losing enteropathy
<i>Small bowel biopsy</i>	See p. 148	See Box 9.3
<i>Radiology</i>	CXR	Aspiration pneumonia, CF Cardiac failure
	Erect and supine AXR Consider barium meal and follow through	Malrotation Blind loop syndrome Crohn's disease Peptic ulcer Constipation Cystinosis
	Left hand and wrist	Bone age

Fresh blood p.r. is most commonly due to an anal fissure or bacterial enteritis. If not, consider:

- clotting abnormality
- intussusception (3 months–3 years)
- volvulus
- dietary protein allergy
- inflammatory bowel disease
- haemangioma or telangiectasia
- sexual abuse or foreign body.

Melaena suggests bleeding from stomach or small bowel:

- clotting abnormality
- peptic ulceration or gastritis
- oesophageal or gastric varices
- Meckel's diverticulum.

In all cases:

- resuscitate first, if necessary. Give plasma 20 ml/kg if shocked. If the shock is severe and obviously due to haemorrhage, give blood, type-specific if possible, but in very severe haemorrhagic shock use Group O Rhesus negative
- look for other bleeding sites, signs of portal hypertension, skin haemangioma
- initial investigations:
 - crossmatch blood

- Hb (initial Hb may not reflect serious blood loss)
- platelet count
- clotting screen
- evidence of chronic liver disease (see Table 9.3)
- involve a surgeon early. Proctoscopy for small rectal bleeds
- all children with head injuries or receiving intensive care should be given ranitidine 1 mg/kg 6–8-hourly as a 3–5 min bolus injection.

Bleeding varices

In addition to the above:

- low threshold for insertion of central venous catheter. Keep CVP 1–2 cm H₂O relative to sternal angle. Avoid over-transfusion
- pass an NGT and aspirate frequently
- treat hepatic encephalopathy (see management of acute liver failure, p. 162)
- octreotide 25–50 µg per hour (or 1 µg/kg/hour) as an infusion
- ranitidine i.v. as above together with oral sucralfate
- passage of a Sengstaken–Blakemore tube requires expert assistance
- discuss with Regional Children’s Liver Unit.

Abdominal pain

A. ACUTE ABDOMINAL PAIN (Box 9.6)

The single most important question is: ‘Does this child require emergency surgery?’ This is a clinical decision but pointers to a surgical cause are:

- signs of peritonism (fever, localised tenderness, including rectally, guarding, rigidity and absent bowel sounds). Appendicitis is the commonest cause but is rare < 2 years. Anorexia is usual and vomiting very common. The younger the child, the more vague the signs of appendicitis
- signs of obstruction (vomiting, abdominal distension, high-pitched bowel sounds and constipation – the rectum may or may not be empty)
- GI bleeding.

Investigations

Consider:

- urine microscopy
- urinalysis for glycosuria, proteinuria, haematuria
- FBC and WBC differential. Sickling test in African and Afro-Caribbean children



Box 9.6 Causes of acute abdominal pain

Require early surgical referral	Abdominal cause but does not require immediate surgical referral	Systemic cause
Appendicitis	Gastroenteritis*	Any febrile illness but especially ENT infection Lower lobe pneumonia Abdominal migraine* Diabetic ketoacidosis Sexual abuse* Sickle causes Porphyria Lead poisoning HSP
Peritonitis	Infantile colic*	
Intussusception	Ingestion	
Volvulus	Constipation*	
Strangulated hernia	Peptic ulcer (including <i>Helicobacter pylori</i> associated	
Obstruction	gastritis/ulceration)*	
Trauma	Pancreatitis, including mumps	
GI bleeding	Cholecystitis/cholangitis	
	UTI*, nephrotic syndrome	
	Urinary calculus*	
	Hepatitis	
	Dysmenorrhoea*	

*More common organic causes of recurrent abdominal pain

- plain AXR (erect and supine).

B. RECURRENT ABDOMINAL PAIN

All of the causes of acute abdominal pain may recur but some are more likely than others (see Box 9.6, asterisks). However, at least 90% of children with recurrent abdominal pain do **not** have an organic cause. Functional abdominal pain ('periodic syndrome', 'abdominal migraine') is a positive clinical diagnosis and **not** a diagnosis of last resort after multiple investigations.

Clues are:

- child is otherwise healthy and thriving
- good appetite and no diarrhoea or vomiting
- episodes are relatively short and may coincide with environmental triggers
- pain is periumbilical; there are no abnormal physical signs
- family history of recurrent abdominal pain or migraine.

If still in doubt, urine microscopy, FBC and ESR (or plasma viscosity), and plain AXR should suffice to reassure you and the parents.

Constipation, soiling and encopresis

While these are usually considered together, **encopresis** is a quite separate entity:

- no organic abnormality
- no constipation: therefore laxatives are not indicated
- normal faeces are voluntarily passed in an unacceptable place, including the child's pants.

There is a significant emotional disorder in the child or in the family and this must be recognised early.

Soiling (involuntary passage of faeces) may be caused by:

- 'developmental delay', i.e. the skill of toilet training has not yet been acquired
- a neurological abnormality, e.g. spina bifida
- chronic constipation and overflow of liquid faeces.

The management of **constipation** is in two stages.

First, exclude an organic cause (Box 9.7), and second, restore a normal bowel habit. Clues to an organic cause are:

- constipation since birth or delay in passage of meconium beyond 24 hours
- the more severe the constipation and the younger the child
- FTT or abnormal physical signs
- empty rectum or 'toothpaste sign' after p.r. exam (suggests Hirschsprung's disease).

MANAGEMENT OF CONSTIPATION WITHOUT AN ORGANIC CAUSE

Response to treatment is directly proportional to the confidence, enthusiasm and persistence of the paediatrician.

Box 9.7 Organic cause of chronic constipation

Gastrointestinal

Hirschsprung's disease (if necessary, exclude by rectal biopsy stained for ganglion cells and cholinesterase)

Anal stricture

Anal fissure

Partial intestinal obstruction

Systemic

Hypothyroidism

Hypercalcaemia

Lead poisoning (associated with pica: anaemia and basophilic stippling on blood film)

Renal tubular disorders (plasma HCO_3^- and urine pH)

Diabetes insipidus (urine osmolality; may need water deprivation test, see p. 29)

Sexual abuse

*Organic causes are more likely in infants, especially if onset is from birth.

ASSESSMENT

Detailed history of the onset of constipation (e.g. febrile illness or visit to a relative), whether unremitting or alternating with periods of normal bowel habit, the age and pattern of toilet training, and the ambience of the domestic lavatory. A dietary history including drinking habits may suggest a cause. A complete physical examination is essential, including rectal examination and assessment of anal tone. Look particularly for an anal fissure.

TREATMENT

- Explain that the problem is common, treatable and not the child's fault.
- Recommend increased fluids (especially fruit juice), brown bread instead of white, bran or high fibre breakfast cereals, cooking with vegetables and pulses.
- Start **regular** faecal softeners, e.g. lactulose 5 ml 12-hourly and increase by 5 ml 12-hourly every 4 days until the stools are soft.
- Recommend that the child sits on the toilet for at least 5 minutes after mealtimes.
- Advise that a diary be kept. See the child with the diary again in 2 weeks. Be encouraging, even if there has been no result.
- If there is no response, start regular laxatives, e.g. Senokot 2.5 ml 12-hourly which can be increased to 10 ml 12-hourly.
- If there is still no response after a further 4 weeks, start behaviour modification (star chart) with 'rewards' which are appropriate to the child's age, sex and family circumstances. Follow-up at 2-week intervals and do not allow your enthusiasm to wane. Remember that forceable use of suppositories or enemas in a resisting child is harrowing for all concerned and may hamper future treatments. If there is still no improvement, arrange an inpatient admission for nursing observation, dietary manipulation, behaviour assessment and intensive behaviour therapy employing nursing staff and the hospital school. This inpatient period may include more aggressive therapy using aperients such as Movicol. After this continue vigorous outpatient follow-up including home visits.

Hepatosplenomegaly

The causes are legion but many are rare. The differential diagnosis can be narrowed by ascertaining whether there is enlargement of liver only, spleen only, or both (Table 9.2) and whether this is recent or chronic.

ACUTE LIVER FAILURE

Suggested by:



Table 9.2 Causes of hepatomegaly. Hyperinflation of the chest (asthma, bronchiolitis) may push the liver down but there is not true hepatomegaly

	Hepatomegaly	Hepatosplenomegaly	Splenomegaly
<i>Infection</i>	Viral hepatitis	Congenital infections Glandular fever	Subacute bacterial endocarditis Malaria
<i>Congestion</i>	Cardiac failure Biliary atresia	Portal hypertension due to a prehepatic cause <ul style="list-style-type: none"> ● cardiac failure ● pericarditis ● Budd–Chiari syndrome 	Portal hypertension <ul style="list-style-type: none"> ● idiopathic extrahepatic portal vein obstruction ● cirrhosis ● previous portal sepsis
<i>Haematological</i>	Haemolytic disease of the newborn	Thalassaemia	Sickle cell disease in a young child (later splenic atrophy) Spherocytosis
<i>Malignancy</i>	Neuroblastoma	Leukaemia Lymphoma	
<i>Metabolic</i>	Reye syndrome Galactosaemia Glycogen storage disorder Wilson's disease	Mucopolysaccharidoses	
<i>Inflammatory</i>	Early cirrhosis Juvenile chronic arthritis		

Table 9.3 Investigation of liver failure

	Abnormality	Diagnosis
<i>Urine</i>	Glycosuria	Haemochromatosis or CF associated with liver damage and diabetes
	Reducing substances	Galactosaemia
	Haematuria	Coagulopathy Hepatorenal syndrome
	Bilirubin but no urobilinogen	Biliary obstruction
Save urine for toxicology screen and chromatography for rare inborn errors of metabolism. Send for viral (CMV) and bacterial culture, and microscopy (infection may precipitate acute or chronic liver failure)		
<i>Blood</i>	1. FBC	
	Hb and iron binding	Iron deficiency anaemia due to chronic GI bleeding Iron overload in haemochromatosis Haemolytic anaemia due to hypersplenism
	WBC	Infection may precipitate acute or chronic liver failure
	Platelets	Low if DIC has supervened or there is hypersplenism
	2. U & E	
	Glucose	Hypoglycaemia commonly complicates liver failure and inborn errors of metabolism
	Electrolytes	Electrolyte abnormalities may precipitate liver failure or result from liver failure (usually hyponatraemia)
	Urea	May be low in severe liver failure (urea is synthesised in the liver) or high if ascites and vomiting cause hypovolaemia and prerenal failure
	3. Tests of hepatocellular function	
	NH ₄ ⁺	May be elevated in liver failure and is not specific for Reye syndrome Falsely elevated if sample is not fresh
	ALT or AST	Transaminases are elevated with liver cell damage
	4. Tests of synthetic function	
	Clotting studies	Both intrinsic and extrinsic paths are prolonged by deficiency of vitamin K dependent factors (II, VIII, IX, X) May occur rapidly
	Albumin	Synthesised in the liver but several days must elapse before levels fall significantly
	5. Tests of excretory function	
Conjugated bilirubin	May be elevated in hepatocellular or obstructive jaundice (see urine)	

Table 9.3 (contd)

	Abnormality	Diagnosis
	Unconjugated bilirubin	Only elevated in very severe liver failure or co-existent hypersplenism (haemolytic anaemia)
	Alkaline phosphatase	Note the wide and age-dependent normal range High levels suggest biliary obstruction
	Gamma-glutamyl transpeptidase	High levels suggest biliary obstruction
	6. Evidence of infection	
	Blood cultures	
	Hepatitis antigens and antibodies	See Box 9.9
	Also CMV and EBV	
	7. Metabolic and other disorders	
	Alpha-1-antitrypsin	Deficiency causes cirrhosis
	Plasma Cu ²⁺ and caeruloplasmin	↑Cu ²⁺ and ↓caeruloplasmin in Wilson's disease
	Autoimmune hepatitis	Immunoglobulins and auto-antibodies
	8. Evidence of toxic substance ingestion, especially paracetamol	
<i>Sweat</i>	Sweat test	CF leads to cirrhosis
<i>Radiology</i>	USS	Essential to exclude biliary obstruction and space-occupying lesion
	<i>Liver biopsy</i> See p. 165	

- alteration of consciousness level
- vomiting
- hypoglycaemia
- bleeding diathesis
- jaundice and electrolyte abnormalities
- enlarged and tender liver.

Management

- Discuss with Regional Children's Liver Unit. Identify and treat the underlying cause (Table 9.3).
- Record baseline vital signs and Glasgow Coma Score (see Ch. 2).
- Avoid sedation. In the presence of liver failure, the dose of all drugs must be checked (see also Table 9.4).
- Minimise encephalopathy:
 - low protein diet; avoid TPN; *but*

Table 9.4 Drugs to be used with caution in liver disease

Drug	Problems in liver disease
Magnesium trisilicate	Sodium load aggravates ascites
Gaviscon	Sodium load aggravates ascites
Diuretics	Hypokalaemia precipitates coma
Beta-blockers	Reduce dose as decreased first pass metabolism
Anticoagulants	Clotting already prolonged
Aminophylline	Reduce dose
Aspirin	GI bleeding and Reye syndrome
Paracetamol	Reduce dose
Opiates	May precipitate coma
Anticonvulsants	Avoid valproate Dose of other anticonvulsant may need to be reduced
Chloramphenicol	Increased risk of bone marrow failure
Erythromycin	} Avoid: increased risk of hepatosplenomegaly
Isoniazid	
Rifampicin	} Reduce dose
Doxorubicin	
Methotrexate	

- maintain a normal blood glucose with i.v. dextrose if necessary
- purge the gut of blood and protein using oral lactulose (1 ml/kg 8-hourly)
- oral neomycin (15 mg/kg 6-hourly) to reduce gut bacteria.
- Correct any coagulopathy. Give vitamin K₁ (phytomenadione) 0.3 mg/kg i.v. slowly routinely and FFP 10 ml/kg if there is active bleeding or to cover an invasive procedure. Cross-matched blood should always be available if there has been haematemesis, malaena or known varices.
- Correct electrolyte imbalances, preferably by altering intake. Treat ascites by:
 - restricting fluid intake to $\frac{2}{3}$ maintenance requirement
 - restricting Na⁺ intake to 1 mmol/kg per 24 hours
 - giving oral spironolactone (1–2 mg/kg 12-hourly) or i.v. potassium canrenoate (1–2 mg/kg 12-hourly, contraindicated in hyponatraemia) to combat hyperaldosteronism; avoid all other diuretics
 - considering salt-poor albumin solution i.v. if serum albumin is < 25 g/l and response to the above is poor *but* do not expect to generate a negative fluid balance of more than 1% bodyweight in each 24-hour period. Avoid paracentesis unless infected ascites is suspected.
- Mannitol 1 g/kg i.v. may temporarily reduce ICP. If more aggressive management is deemed appropriate (ventilation, ICP monitoring, renal dialysis, exchange transfusion, charcoal

Box 9.8 Causes of childhood cirrhosis

Hepatic (commonest)

1. Wilson's disease
2. Chronic hepatitis (following hepatitis B or as an autoimmune disorder)
3. Alpha-1-antitrypsin deficiency
4. Drug induced (paracetamol overdose, chlorocarbon ingestion, isoniazid, methotrexate, halothane)

Post-hepatic

1. Biliary atresia and other anatomical anomalies of the biliary tree
2. Recurrent cholangitis
3. Inflammatory bowel disease
4. CF

Pre-hepatic (rarest)

Budd–Chiari syndrome

haemoperfusion), consider this early on and arrange transfer to a major centre.

CHRONIC LIVER FAILURE

Hepatomegaly or portal hypertension due to cirrhosis (Box 9.8) are suggested by:

- spider naevi
- palmar erythema
- jaundice, anaemia or Kayser–Fleischer ring
- purpura, haematemesis or malaena
- enlarged and hard liver with non-tender splenomegaly
- ascites and peripheral oedema.

A mixture of these features may be seen in acute on chronic liver failure and obviously there may be other signs of the underlying cause.

Management

Usually as an outpatient. The following may require attention:

- Treatment of the underlying cause.
- FTT:
 - high-energy, high-carbohydrate, low-protein diet
 - vitamin supplementation (particularly fat soluble vitamins A, D, E and K: use Ketovite liquid 5 ml per 24 hours and Ketovite tablets orally, one tablet 8-hourly, unless serum monitoring demonstrates inadequate levels in which case each vitamin can be supplemented individually.

- Coagulopathy: monitor PT as index of adequacy of vitamin K supplementation. Iron deficiency anaemia suggests chronic GI bleeding.
- Biochemical rickets: monitor Ca^{2+} , PO_4^{3-} and alkaline phosphatase as index of adequacy of vitamin D supplementation.
- Jaundice: monitor levels of unconjugated bilirubin. Cholestyramine may be necessary for pruritus but may exacerbate fat-soluble vitamin and folic acid deficiency.
- Monitor transaminase levels: look for causes of abrupt deterioration.
- Ascites (see p. 163).
- Haematemesis and melaena: advise the parents to seek urgent admission. Avoid aspirin and other non-steroidal anti-inflammatory analgesics (see Table 9.4). Endoscopy and oesophageal variceal banding should be undertaken by an expert.

Liver biopsy

Should be performed only by an expert and only when the result may aid management:

1. Wilson's disease
2. Chronic hepatitis
3. Conjugated jaundice without evidence of viral infection or anomaly of biliary tree
4. Unconjugated jaundice without haemolysis (Crigler–Najjar syndrome).

Platelet count and PT should be checked, blood crossmatched and available, and consent obtained. The histopathologist **must be consulted before** the procedure regarding the need for samples for:

- formalin fixation for light microscopy
- unfixed core for frozen section
- glutaraldehyde fixation for electron microscopy
- snap freezing for enzyme studies
- fresh specimen for chemical analyses.

Jaundice

NEONATAL

See Ch. 19.

THE OLDER CHILD (Box 9.9)

Mixed hyperbilirubinaemia is more common and the cause is usually infective. Hepatitis A is the commonest in the UK and rarely requires hospital admission. Elevated transaminases precede jaundice. No longer infectious once jaundice appears. Urine is dark. Stools may be pale.

Conjugated hyperbilirubinaemia (pale stools and dark urine) is rare in childhood and always requires investigation: early USS to demonstrate biliary obstruction. Look for evidence of chronic liver disease (see Box 9.8).