Chapter 6:

GASTROINTESTINAL DISORDERS

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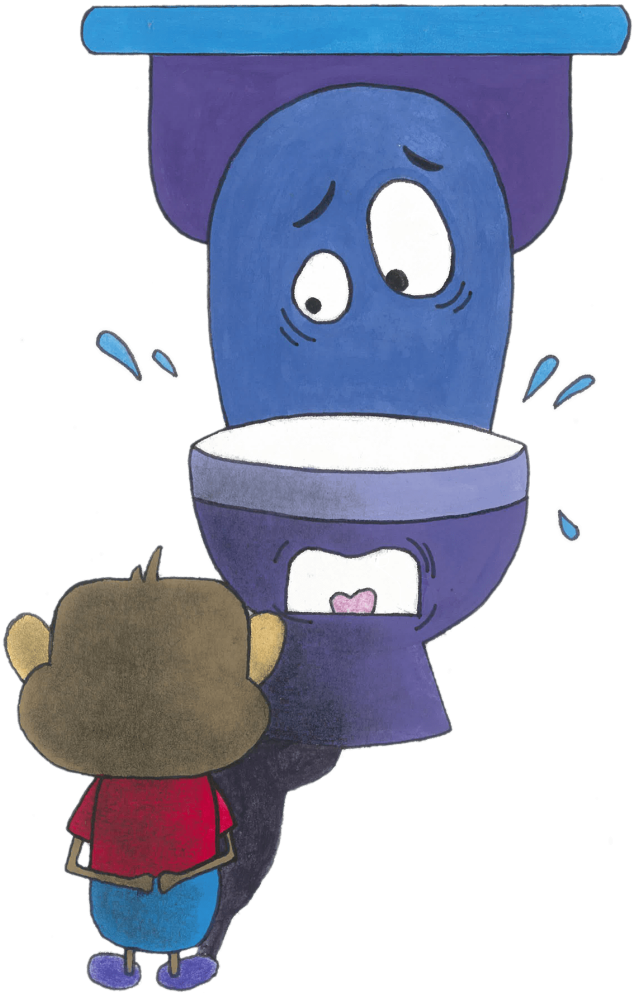

This chapter covers the following topics:

- Diarrhoea
- Hepatitis
- Portal hypertension
- Gastroesophageal reflux
- Constipation
- Jaundice
- Hepatosplenomegaly
- Gastrointestinal bleeding
- Functional abdominal pain (FAP)

DIARRHOEA

Diarrhoea is defined by the WHO as ≥3 watery stools in 24 hours. Almost every child will have diarrhoea at least once and approximately 1 in 3 hospital admissions in South Africa are due to diarrhoea. It remains a major cause of morbidity and mortality worldwide in children <5 years old. It is the second most common cause of death (after HIV) in South African children of that age group.

Classification, Pathophysiology and Aetiology

In diarrhoea, the excretion of water and electrolytes exceed net absorption. Diarrhoea may be classified according to pathophysiology or duration.

Pathophysiological Classification

Diarrhoea may be classified as:

- Osmotic diarrhoea:
    - It occurs when a large number of osmotically active particles are present in the lumen, leading to the passive flow of fluid into the bowel lumen.
    - Causes include laxatives, lactulose, lactose, and other food intolerances/allergies.
- Secretory diarrhoea:
    - An activated pathway (due to the release of a toxin by a pathogen that invaded the intestinal mucosa) or inherent abnormalities of enterocytes cause excessive amounts of fluid to be secreted.
    - Diarrhoea results when absorptive mechanisms become overwhelmed i.e. secretion exceeds absorption.
    - Infective causes of secretory diarrhoea include:
        - Viruses e.g. rotavirus, norovirus, calicivirus, Norwalk virus
        - Bacteria e.g. *Campylobacter jejuni*, *Salmonella* sp, *E. coli*, *C. difficile*, Shigella sp, *Yersinia enterocolitica*
        - Parasites e.g. *Cryptosporidium* sp*, Giardia lamblia*

Classification According to Duration

Diarrhoea may be considered:

- Acute:
    - There is a sudden onset of increased frequency of stools which lasts no longer than 14 days.
    - It is most commonly caused by a virus and laboratory investigation is not necessary to diagnose acute diarrhoea.
- Persistent diarrhoea:
    - It is an episode of diarrhoea of presumed infectious aetiology that begins acutely and lasts ≥14 days.
- Chronic diarrhoea:
    - It is a diarrhoeal episode that lasts ≥14 days and which most commonly has a non-infectious cause e.g. lactose intolerance, food allergies.

Clinical Features

The clinical presentation and course of diarrhoea depends on the aetiology of the diarrhoea and the host (see an image of a child threatened by severe diarrhoea here). The patient may present with:

- Features of dehydration:
    - Lethargy
    - Depressed level of consciousness
    - Sunken anterior fontanelle
    - Dry mucous membranes and no tears
    - Sunken eyes
    - Reduced skin turgor
    - Delayed capillary refill (>2 s)
- Complications of dehydration:
    - Electrolyte disturbances (hyponatraemia, hypokalaemia and metabolic acidosis)
    - Acute renal failure
    - Paralytic ileus
    - Convulsions
    - Cerebral damage
- FTT and malnutrition (diagnosed based on anthropometry measurements, reduced muscle/fat mass, peripheral oedema)

- Perianal erythema

Table 6.1: Recognising and Classifying Dehydration

Hydration	No dehydration	Some dehydration (5% dry)	Severe dehydration (≥10% dry)	Shock (an important danger sign)
Level of consciousness	Normal	Normal	Lethargic	Not responding
Eyes	Normal	Sunken	Very sunken	Dull
Mucous membranes	Moist	Dry	Very dry	Varies
Thirst	Drinks normally	Thirsty	Extremely thirsty	Does not want to drink
Skin	Normal skin turgor	Decreased skin turgor	Very decreased skin turgor	Mottled and cold
Anterior fontanelle	Normal	Sunken	Very sunken	-
Weight loss	None	<5%	5-10%	Varies
Pulse rate	Normal	Normal	Fast	Fast/slow
Pulse volume	Normal	Normal	Thready	Thready/impalpable
Respiration	Normal	Deep	Fast and deep	Irregular and difficult

Note: The most accurate measure of dehydration is loss of weight. One must remember that shock may occur even in the absence of other signs/symptoms of dehydration.

Complications

Diarrhoea may be complicated by:

- Electrolyte abnormalities:
    - Hypernatremia and hyponatraemia (common in secretory diarrhoea and the malnourished) – causes irritability and seizures
    - Hypokalaemia – causes hypotonia, paralytic ileus, bradycardia and respiratory failure in prolonged or recurrent episodes
    - Hypocalcaemia (may occur in secretory diarrhoea)
    - Hypomagnesaemia (may occur in secretory diarrhoea)

- Hypoglycaemia (due to catecholamine release and may develop in the malnourished, septic or hypothermic patient)
- Metabolic acidosis (caused by loss of bases in stool, poor tissue perfusion and decreased renal hydrogen clearance)
- Renal failure (in severe dehydration)
- Haemolytic uraemic syndrome – characterised by haemolytic anaemia, acute kidney injury (uraemia) and thrombocytopaenia
- Rhabdomyolysis
- Shock
- Death

Investigations

No routine investigations are required, as most children will recover spontaneously within a few days, regardless of the underlying causative organism. However, a urine dipstick and finger-prick glucose should be done on all children admitted to hospital. Investigations may include:

- Stool culture (always done for):
    - Children with bloody diarrhoea (send cultures for *C. difficile, Campylobacter* sp, *Shigella* sp and *E.coli*)
- Electrolytes, RFTs and ABG (always done for):
    - Severely dehydrated children
    - Moderately dehydrated children with an unusual clinical picture
    - Malnourished children
    - Children requiring IV fluids for rehydration
    - Children with complications
- ABG

Management

Primary Prevention

Diarrhoea may be prevented by:

- Ensuring a clean water supply
- Encouraging good sanitation and hygiene
- Giving vitamin A prophylaxis

- Encouraging breastfeeding
- Giving supplemental zinc
- Vaccinating against rotavirus

Secondary Prevention

This involves the prevention of dehydration and its complications. Acute diarrhoea is usually self-limiting. However, one must always assess the patient for danger signs (not drinking, lethargy, intractable vomiting, convulsions and bloody stools) and give supportive management.

Supportive management includes:

- Oral rehydration therapy (ORT; give as soon as possible)
- Zinc supplementation (10-20 mg daily for 10-14 days). See also the rehydration formula recommended by UNICEF here.

Tertiary Prevention

One's aim here is to prevent death from dehydration and complications. Thus, tertiary prevention involves appropriately managing the complications that the child has and appropriately managing dehydration and shock (refer to general fluid management in the *Feeds and Fluid Management* chapter).

HEPATITIS

Hepatitis is inflammation of the liver which can result in the damage and destruction of hepatocytes.

Aetiology

Common causes of hepatitis include viruses, autoimmune liver disease, and drugs and toxins.

Viral Hepatitis

In viral hepatitis, the damage to the liver is not due to the viral invasion but the immune response to the virus. In neonates the virus is usually vertically transmitted from the mother in the perinatal period. Viral causes of hepatitis include:

- Hepatitis viruses:

    - A (HAV) – transmitted via the faecal-oral transmission; the degree of hepatic injury depends on the host's immune response
    - B (HBV) – transmitted via bodily fluids e.g. blood, semen
    - C (HCV) – transmitted through contact with infected blood
    - D (HDV) – not common in children as it requires HBV co-infectivity for replication
    - E (HEV) – transmitted via the faecal-oral route
- CMV
- VZV
- HSV (especially in infants)
- Enterovirus
- Rubella
- Adenovirus
- Parvovirus

Autoimmune Liver Disease

They include:

- Autoimmune hepatitis
- Sclerosing cholangitis
- Kawasaki disease
- Graft-versus-host disease
- Immunodeficiencies

Drugs and Toxins

- Medication-induced hepatitis
- Paracetamol toxicity
- *Amanita phalloides* (a poisonous wild mushroom)

Clinical Features

They vary as they depend on the aetiology and the child's age of the child (infant vs child vs adolescent). Some children may be asymptomatic, while others may present with:

- Flu-like symptoms

- Malaise
- Jaundice
- Fever
- Nausea and vomiting
- Anorexia or poor feeding
- Abdominal discomfort
- Diarrhoea
- Dark urine and clay-coloured stools
- Tender hepatomegaly

Acute HBV infection is usually symptomatic, while chronic infection is often asymptomatic (children with the latter infection will grow and develop normally). Chronic infection may be associated with polyarteritis nodosa and glomerulonephritis, and can progress to cirrhosis and hepatocellular carcinoma. Most children with chronic HCV infection are asymptomatic, but can develop cirrhosis and hepatocellular carcinoma (especially if there is a hepatitis B co-infection).
Some children may present in acute liver failure i.e. fulminant liver failure without pre-existing liver disease (an uncommon medical emergency). Causes include infection with HAV, HBV, Reye's syndrome, drugs (e.g. paracetamol, anti-TB drugs) and toxins (e.g. traditional medication).

Investigations

One may order:

- Blood tests
    - Liver enzyme levels (ALT, AST, ALP, GGT)
    - LFTs (INR, albumin, etc.)
    - Antibody and PCR studies for suspected viral hepatitis: these results must be interpreted to differentiate between acute and chronic infection
    - FBC
    - Serum glucose
- Imaging (USS of the liver)

Pathophysiology, Diagnosis and Management

Table 6.2: Pathophysiology, Diagnosis and Management of Hepatitis A and B

	HAV	HBV
Pathophysiology	It is transmitted by the faecal-oral route and has an incubation period of 15-50 days. The child is most infectious 1-2 weeks before symptom onset. The child may be asymptomatic (young children) or may have a prodrome of nausea, anorexia and malaise. S/he may go on to develop jaundice, dark urine and tender hepatomegaly. Chronic infection does not develop and fulminant infection is very rare.	Children may be infected by horizontal, vertical or parenteral transmission. HBV has an incubation period of 2-6 months and a similar prodrome and clinical features to HAV (but the disease is more insidious and there is a longer prodrome). 90% of neonates and 30-40% of infected children become chronic carriers – asymptomatic in the beginning and then develop chronic hepatitis, cirrhosis and HCC.
Diagnosis	Current/recent HAV infection is diagnosed if the child is HAV IgM positive (can remain positive for 4-6 months). Previous infection can be diagnosed if the child is HAV IgG positive.	Acute HBV infection is diagnosed if the child is HBV IgM, HBsAg and HBeAg positive. Chronic HBV is also diagnosed using the above tests and may be: ● High-risk (HBeAg-positive. HBeAb-positive, HBV IgG-positive) ● Low-risk (HBeAg-negative. HBeAb-positive, HBV IgG-positive).
Management	The local authorities should be notified (HAV is a notifiable disease). Management includes: ● Giving supportive care (avoid liver toxic drugs and ensure adequate hydration)	Treatment of chronic HBV infection includes α-interferon, pegylated interferon and nucleotide/-sides (entecavir, tenofovir or lamivudine). Acute infection

	● Following an appropriate diet (high in calories and low in protein) ● Encouraging good hand hygiene and in-hospital isolation for 1 week after the onset of jaundice (to prevent transmission) ● Managing household contacts The child should be admitted if s/he has any danger signs (prolonged vomiting, dehydration, persistent fever, hypoglycaemia, confusion, intercurrent infection or raised INR).	is usually self-limiting and the patient should just isolate.
Prevention	Two doses of the HAV vaccine (0.5 mL IMI) should be administered. The HAV vaccine can also be used as post- or pre-exposure prophylaxis. Otherwise one can give a single dose of pooled human Ig (0.04 mL/kg IM).	HBV Ig may be given to: ● Non-immune children (2 mL IMI given 1-7 days after exposure) ● Children who have had a high-risk exposure (same as above plus a second dose 1 month later) ● Infants born to HBsAg-positive mothers (0.5 mL IMI within 12 hours of birth) The HBV vaccine is part of the routine vaccination programme in SA.

Prevention of other causes of hepatitis:

- HCV:
    - HCV is acquired during infancy and is most likely to clear spontaneously.
    - Interferon-α or ribavirin should be considered in children with chronic infection.
- Autoimmune hepatitis (give prednisone).

Note: See the *Infectious Diseases* chapter for management of other viral causes.

PORTAL HYPERTENSION

It is defined if the child has a portal pressure >10 mmHg or hepatic venous pressure gradient >4 mmHg. Chronic liver disease results in increased vascular resistance or blood volume within the portal venous system and may be complicated by portal hypertension.

Aetiology

Portal hypertension may be due to a pre-, intra- or post-hepatic cause.

- Pre-hepatic causes:
    - Portal or splenic vein obstruction
    - Congenital portal vein stenosis
    - Extrinsic compression of the portal vein
- Intrahepatic causes:
    - Biliary atresia
    - Cystic fibrosis
    - Autoimmune hepatitis
    - Choledochal cyst
    - Cirrhosis
    - Schistosomiasis
    - Congenital hepatic fibrosis
    - Veno-occlusive disease
    - Granulomatous diseases e.g. sarcoidosis, TB
- Post-hepatic causes:
    - Budd-Chiari syndrome
    - Inferior vena cava thrombosis
    - Congenital malformation of inferior vena cava
    - Constrictive pericarditis or right heart failure

Clinical Features

Portal hypertension should be suspected in any child with significant GI bleeding (acute variceal haemorrhage is the most serious complication) or unexplained

splenomegaly. The child may also present with signs of chronic liver disease such as:

- Ascites (see also image here)
- Periumbilical vascular collaterals
- Manifestations of hypersplenism e.g. bruising from vitamin K deficiency (leading to a prolonged INR)
- FTT

Patients with cirrhosis may present with hepatic decompensation and encephalopathy. Pre-hepatic causes may not cause jaundice.

Investigations

One must look for the underlying aetiology by performing:

1. Blood tests:
    - FBC
    - Liver enzyme levels
2. Imaging:
    - Doppler USS e.g. portal vein and splenic vein thrombosis
    - CT angiography (not routinely done but may be ordered depending on the clinical picture)
    - Liver biopsy (not routinely done but may be ordered depending on the clinical picture)
    - Endoscopy (to look for oesophageal varices)

Management

The aim is to treat the underlying aetiology and complications (especially if the child is bleeding). An early referral should be made to a hepatologist or gastroenterologist after the patient has been stabilised.

One must avoid morbidity and mortality after a bleeding episode. Management of the patient with an acute variceal haemorrhage may include:

- Resuscitation (ABCs):
    - The patient may need to be given fluids, FFP or a RBC transfusion (restrict fluids to 70% of maintenance). Vitamin K should also be administered.

    - Actively bleeding veins should be tamponaded with a Sengstaken-Blakemore tube.
- Monitoring vitals, urine output, haemoglobin and level of consciousness.
- Keeping the patient nil per os and inserting an NGT (avoid if ongoing variceal bleeding is suspected).
- Administering empiric, broad-spectrum antibiotics, octreotide and omeprazole as needed.
- Performing surgery:
    - One may perform endoscopic variceal ligation or injection sclerotherapy (the former is the preferred method).

GASTROESOPHAGEAL REFLUX (GOR)

It is the involuntary passage of gastric contents into the oesophagus and is a normal physiological process. Episodes occur in the distal oesophagus, last <3 mins and are asymptomatic. Secretions contain food, drink, saliva, and gastric, pancreatic and biliary secretions. See also an image related to Reflux Gastro-Oesophagien here.

Pathophysiology

GOR is usually due to transient relaxation of the lower oesophageal sphincter (LOS). Less commonly it may be due to low LOS tone (chalasia). When the refluxed material passes into the mouth, this is termed regurgitation. Regurgitation is common in infancy (present in 60% of infants at 3 months old; resolved in 90% of infants by 1 year old). GOR and regurgitation are often not pathological, but complications may arise in a few children.

Clinical Features and Complications

GOR disease (GORD) is diagnosed when there are complications of GOR. There are no clinical features that are diagnostic of GORD but the following clinical features suggest the diagnosis:

- Oesophagitis:
    - Peptic (reflux) oesophagitis causes pain, food refusal, irritability, posturing and, less frequently, haematemesis and iron-deficiency anaemia

- Dental erosions
- Respiratory disease/complications:
    - Stridor/laryngitis
    - Recurrent wheezing
    - Hoarseness
    - Chronic cough
    - Aspiration pneumonia
    - Bronchiectasis
    - Asthma exacerbations
- FTT and poor weight gain
- Oesophageal strictures (in children with long-standing GORD)
- Athetoid movements and posturing:
    - They are associated with GORD (Sandifer syndrome) and may be confused with seizures, especially in children with brain damage.
    - Children with cerebral palsy may have severe GORD that is resistant to treatment and are more prone to oesophageal strictures following erosive oesophagitis.
- Sinusitis and otitis media (this association has not been well-established)

Investigations

One may order the following investigations:

- Barium swallow:
    - It is performed if oesophageal, stomach or proximal bowel structural abnormalities are suspected (e.g. malrotation, hiatal hernia, oesophageal stricture) as these disorders may present similarly to GORD.
- 24-hour oesophageal pH-metry:
    - It is performed when the diagnosis of GOR is uncertain or to assess the effect of therapy, as it provides a quantitative measure of acid reflux.
- Upper GI endoscopy:
    - It is performed to look for features of reflux oesophagitis and to exclude eosinophilic oesophagitis, infectious causes and structural causes.

- Nuclear medicine “milk scan”/nuclear scintigraphy:
    - It allows one to quantify the volume, frequency and height of the reflux.
- Endoscopy and biopsy:
    - They are performed to identify and grade oesophagitis, and exclude eosinophilic oesophagitis.

One must, therefore, exclude other causes of chronic respiratory disease that may mimic GORD and assess the patient for signs of raised intracranial pressure, GI obstruction (e.g. projectile vomiting, abdominal distension) and urinary tract infection.

Management

GOR does not require treatment, however GORD does.

Management of Functional Regurgitation

It will include:

- Parental reassurance
- Advice regarding feeding technique (e.g. avoiding overfeeding, practising burping technique) and thickening feeds (reduces frequency of vomiting, not reflux)
- Changing feeds if milk protein sensitivity is suspected (a trial of extensively hydrolysed feed may be done)
- Positioning the infant in a prone position (regurgitate less often):
    - This should only be done in infants >1 year old who are no longer at risk of sudden infant death syndrome (SIDS).
    - Otherwise, placing the child on his/her side may provide some relief.
- Prescription of proton pump inhibitors (PPIs) e.g. omeprazole (0.7-1.4 mg/kg in the morning 20 mins before breakfast)

The child with GORD or severe reflux may need to have a Nissen fundoplication (if s/he does not respond to optimal medical treatment).

Management of GORD

Young infants with severe malnutrition or respiratory complications should be given = transpyloric (NGT) feeds and PPIs for acid-related complications. H_2-receptor antagonists may also help acutely (if the patient presents with gastritis), however

they should not be chronically used. Surgery may be performed if there is no response to optimal medical treatment

CONSTIPATION

Definitions

Constipation is the infrequent or irregular passage of unduly hard stools.
Faecal loading is the build-up of faeces due to ineffective or incomplete evacuation of stool. **Encopresis** is an apparently wilful passage of normal consistency stool into underclothes or other places. (Refer to child psychiatry.)
Soiling is involuntary leakage of small amounts of soft or watery stool secondary to faecal loading and rectal dysfunction.
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Aetiology

Causes may be grouped according to the age of the child.

- Neonate:
    - Intestinal obstruction e.g. atresias
    - Cystic fibrosis (meconium ileus, meconium plug)
    - Hirschsprung's disease
- Child in early infancy:
    - Misdiagnosis of normal, infrequent, breastfeeding stools
    - Hypothyroidism
    - Hirschsprung's disease
    - Dehydration
- Toddler:
    - "Toilet training" constipation
    - Transient constipation
    - Acute constipation
    - Cerebral palsy (unco-ordinated peristalsis and evacuation lead to constipation)
- School-going child:
    - Change in environment or lack of privacy
    - Side effects of medication

    - Abovementioned causes which have been inadequately or ineffectively managed
    - Lack of exercise or inactivity

Other causes of constipation include coeliac disease and drugs. See related image here.

Clinical Features

The child may have

- <3 bowel movements/week
- Hard, dry and difficult to pass stools
- Large stools which may obstruct the toilet
- Painful defecation (check for anal fissures, especially if there is blood in stool)
- Soiling of underwear or clothes (faecal incontinence)
- Abdominal pain
- Abdominal distension (mild)
- Weight loss
- Fever and/or vomiting

Investigations

They may include:

- Growth assessment
- Abdominal examination (a faecal mass may be palpated)
- Digital rectal examination (including inspection of the peri-anal area and rectum)
- Full neurological examination

Other tests that are not routinely done (but may be performed depending on the suspected aetiology) include:

- Barium enema X-ray
- Thyroid function tests and serum calcium levels (only done in resistant cases)
- Rectal biopsy (if Hirschprung's disease is suspected)
- Abdominal X-ray (not necessary to make the diagnosis)

Management

Immediate Management

The parents and child should be counselled on constipation and the importance of behavioural and dietary changes. The colon can then be cleared with repeated phosphate-containing enemas (for disimpaction) or a balanced electrolyte polyethylene glycol (PEG) solution.

Klean Prep® (15-25 mL/kg/hour) may also be given and an enema done (within the first hour of starting Klean Prep®. The solution should be continued until the rectum is clear and the abdomen is soft (~6-8 hours). However, the patient should be observed for aspiration.

Maintenance Therapy

It may need to be continued for months or years and may include

- Macrogol e.g. Movicol®
- Osmotic laxatives e.g. lactulose, sorbitol
- Stool lubricants e.g. liquid paraffin
- Glycerine suppositories and prune juice (for children <1yrs)
- Anaesthetic cream (for anal fissures)

Prevention

Constipation may be prevented with:

- Regular physical activity
- High-fibre diet (supplement with bulk laxatives)
- Good hydration (water or fruit juice)
- Regular toilet and daily bowel routine
- Star charts or a stool diary

JAUNDICE

Jaundice is the yellow discolouration of the skin and mucous membranes and is a sign of hyperbilirubinaemia.

Pathophysiology, Classification and Aetiology

Unconjugated Hyperbilirubinaemia

It may be further subclassified as:

- Neonatal jaundice (jaundice which usually develops on the second or third day of life and which persists for <14 days from birth):
  - Physiological jaundice:
    - It usually develops on the second or third day of life.
    - The short lifespan of foetal RBCs leads to increased haemolysis in the neonate and, therefore, increased bilirubin production.
    - However, the immature liver is unable to process these large amounts of bilirubin (decreased bilirubin conjugation) resulting in unconjugated hyperbilirubinemia.
  - Breastfeeding jaundice:
    - It occurs in the first week of life in some infants and is the result of decreased milk intake.
    - The resulting dehydration leads to increased enterohepatic circulation of bilirubin.
  - Haemolytic disease of the neonate
  - Haemorrhage
  - Polycythaemia
- Prolonged neonatal jaundice (jaundice persisting >14 days from birth):
  - Breast milk jaundice:
    - It occurs in some neonates, in the second week of life or later and is thought to be due to a substance in breast milk that affects the infant liver's metabolism of bilirubin.
  - Isoimmunisation
  - Cephalohaematoma
  - Hypothyroidism
  - Sepsis
  - ABO incompatibility

Conjugated Hyperbilirubinaemia

It is diagnosed if the conjugated bilirubin level is >34mmol/L or >15% of the total bilirubin level. Causes include:

- Infection:
    - Viruses – HAV, HBV, CMV, rubella, HIV, HSV (TORCH infections)
    - Bacteria – syphilis, sepsis, UTI
    - Protozoa – toxoplasmosis
- Biliary pathology:
    - Biliary atresia
    - Choledochal cyst
    - Alagille's syndrome
- Metabolic/genetic disease:
    - α_1-antitrypsin deficiency
    - Galactosaemia
    - Wilson's disease
    - Cystic fibrosis
- Drugs/toxins e.g. total parenteral nutrition (TPN)
- Autoimmune:
    - Autoimmune hepatitis
    - Sclerosing cholangitis

Clinical Features

The child may present with:

- Yellow sclera, mucous membranes and/or skin
- Poor feeding
- Weight loss >10% in a neonate
- Lethargy

Jaundice which develops within the first 24 hours of life is likely to be pathological and requires further investigation. Jaundice which develops after day 3 of life also needs close monitoring and investigation.

Investigations

They should include:

- Transcutaneous bilirubin (TCB) level (in neonates)
- Conjugated and total serum bilirubin (TSB) levels (in older children and/or infants with mild jaundice)
- Other LFTs
- Maternal and neonatal blood type and rhesus (Rh) factor screen, and Coombs test
- Haemoglobin level and reticulocyte count
- CRP
- Cholesterol level
- Thyroid function test
- Tests for viral and/or parasitic infection, as needed e.g. urine dipstick, blood culture

Management

It is important to diagnose and manage jaundice appropriately as it can result in kernicterus if left untreated. Generally, management includes:

- Continuing breastfeeding
- Starting phototherapy and/or exchange transfusion in neonates (use phototherapy chart for guidance in neonates)
- Treating the cause (if pathological and reversible)

Management of Early-Onset Jaundice

Jaundice which develops within the first 24 hours of life is most likely due to haemolytic disease of the neonate (ABO or Rh incompatibility). Management, therefore, includes:

- Checking the mother's blood group:
    - If the mother is type O then ABO incompatibility is most likely.
    - Isoimmune-haemolytic disease may be treated with IV gamma globulin (0.5 g/kg over 2 hrs) if the TSB is increasing at a fast rate despite phototherapy or if it is <50 mmol/L below the exchange value.
    - Haemolytic disease of the neonate may be prevented by administering anti-D globulin to Rh-negative mothers within 72 hours of giving birth to a Rh-positive child.

- Performing three-hourly TSBs
- Starting phototherapy

The following tests should also be done:

- Direct Coombs test
- Hb or packed cell volume (to diagnose anaemia)
- Peripheral blood smear

Rarely, one may need to test for glucose-6-phosphate-dehydrogenase (G6PD) deficiency and do Hb electrophoresis.

Management of Late-Onset Jaundice

If the jaundice develops >24 hours after birth and the unconjugated bilirubin is above the normal limit, one must:

- Check the blood groups of the mother and child (exclude blood group incompatibility)
- Exclude sepsis or cephalohaematoma in a neonate
- Check that the neonate is sucking well and weigh baby (breastfeeding jaundice)
- Measure packed cell volume (the child may be polycythaemic i.e. have haematocrit (Hct) >70%)

HEPATOSPLENOMEGALY

It is enlargement of both the spleen and the liver.

Aetiology

Causes of hepatosplenomegaly include:

- Inflammation:
    - Infection – malaria, toxoplasmosis, infectious mononucleosis, HIV, rubella, congenital syphilis, HSV, CMV, schistosomiasis, etc. (TORCH infections)
    - Autoimmune disease
    - Drugs
    - Obstruction
- Infiltration:

    - Disseminated TB
    - Malnutrition
    - Septicaemia
    - Malignancy
    - Sarcoidosis
    - Reye's syndrome
    - Amyloidosis
- Congestion:
    - Biliary atresia
    - Cardiac failure (congestive or right-sided failure)
    - Constrictive pericarditis
    - Budd-Chiari syndrome
    - Cirrhosis
- Storage disorders:
    - Galactosaemia
    - Glycogen storage disease
    - Lipidosis
    - Uncontrolled diabetes
- Space-occupying lesions:
    - Abscess
    - Neoplasm (benign or malignant)
- Metabolic disease:
    - Wilson's disease
    - Gaucher's disease
    - Niemann-Pick disease
- Haematological disorders
    - Leukaemia
    - Lymphoma
    - Sickle cell anaemia
    - Thalassaemia
- Miscellaneous
    - Juvenile rheumatoid arthritis
    - Systemic lupus erythematosus (SLE)

Clinical Features

The child may present with:

- Fever
- Jaundice
- FTT
- Dyspnoea
- Vomiting
- GIT bleeding
- Pallor
- Petechiae, purpura or ecchymosis
- Lymphadenopathy
- Tender hepatosplenomegaly
- Ascites
- Raised JVP (if there is a cardiac cause for the hepatosplenomegaly)

Investigations

Investigations should only be performed as indicated. They may include:

- LFTs
- FBC
- Blood culture
- Mantoux test
- Imaging – USS, chest X-ray, CT scan
- Other tests e.g. α-fetoprotein, HBV antigen, PTT, INR, sweat chloride test, ceruloplasmin

Management

One must treat the cause. Thus, the patient should be referred to the relevant paediatric specialist depending on the cause or a hepatologist if the cause is unknown.

GASTROINTESTINAL (GI) BLEEDING

Upper GI bleeding (UGIB) is GI bleeding which is proximal to the ligament of Treitz (the junction of the duodenum and jejunum) i.e. oesophagus, stomach or duodenum. These patients often present with haematemesis and/or melena.

Lower GIGI bleeding (LGIB) is bleeding which is distal to the ligament of Treitz i.e. small bowel or colon. These patients usually present with haematochezia.

Aetiology

The causes of GI bleeding vary depending on the child's age.

Table 6.3: Causes of UGIB and LGIB

Age Group	Cause of UGIB	Cause of LGIB
Neonates	● Swallowed maternal blood i.e. not true bleeding ● Vitamin K deficiency/ haemorrhagic disease of the neonate ● Vascular malformations ● Stress gastritis or ulcers associated with critical illness ● Coagulopathy ● Cow's milk protein intolerance ● Gastric or duodenal ulcers ● GI duplication cyst	● Swallowed maternal blood (not true bleeding) ● Anorectal fissures ● Necrotising enterocolitis ● Malrotation with midgut volvulus ● Hirschsprung's disease with enterocolitis ● Coagulopathy ● Brisk UGIB ● Vascular malformations
Infants and toddlers	● Mallory-Weiss syndrome ● Oesophageal or GI foreign body ● Oesophagitis ● Peptic ulcers and gastritis	● Meckel's diverticulum ● Intussusception ● Anal fissures (especially around the time of the introduction of solid food or cow's milk) ● Milk- or soy protein-induced colitis (allergic colitis) ● Infectious colitis

	● Bleeding oesophageal varices or gastric varices ● Arterial bleeding (rare)	● Lymphonodular hyperplasia ● GI duplication cyst ● Coagulopathy ● Eosinophilic GI disease ● Infantile and very early-onset inflammatory bowel disease (IBD)
Pre-school going age		● Anal fissures ● Intussusception ● Meckel's diverticulum ● Other causes e.g. infectious colitis, haemolytic uraemic syndrome, IgA vasculitis, Henoch-Schonlein purpura, juvenile polyps, very early-onset IBD, solitary ulcer syndrome
School going age		● Anal fissures ● Juvenile polyps ● Infectious colitis (*Salmonella sp, Shigella sp, Campylobacter sp, E.coli, Clostridium difficile* are the most common) ● Inflammatory bowel disease ● Meckel's diverticulum ● Solitary rectal ulcer syndrome ● IgA vasculitis (Henoch-Schonlein purpura ● Haemorrhoids

Clinical Features

The child may present with:

- Haematemesis (vomiting of bright red blood or coffee ground-like material)
- Melena (passage of black and tar-like stools with a strong odour):
    - The colour and smell are due to the haemoglobin in the blood being altered by the digestive enzymes and intestinal bacteria.
    - It is important to note that black stools may also be caused by certain medications and foods.

- Haematochezia (passage of bright red/maroon-coloured blood or fresh clots per rectum):
    - It is usually due to LGIB but can be due to UGIB in cases of short intestinal transit time or massive UGIB.
- Occult GI bleeding (bleeding is not visible to the naked eye of the patient or physician):
    - Patients usually present with iron-deficiency anaemia or occult GI bleeding may be identified by testing the stool for occult blood.

Approach

When a patient presents with GI bleeding, one must ask the following questions

1. Is the patient haemodynamically stable or is resuscitation indicated?
2. Is it blood?
3. Is the blood from the upper GIT (dark red/black) or lower GIT (bright red)?
4. What are the most likely causes of the bleed?

The patient should then be assessed based on:

- History and examination:
    - One should ask about:
        - This episode of bleeding – chronology of the bleeding episode, estimated blood loss, colour of blood and any associated symptoms e.g. abdominal pain, fever, weight loss and fatigue, recent use of NASIDs, etc.
        - Associated symptoms (paying attention to GI symptoms) – dyspepsia, heartburn, abdominal pain, dysphagia, and weight loss, poor feeding or irritability (in infants), history of jaundice, easy bruising, or change in stool colour (liver disease).
        - Possible causes: easy bruising/bleeding, personal or family history of liver, kidney, heart disease or coagulopathies, drug history, travel history, diet, etc.
    - On examination one should look for signs of shock and possible causes of bleeding. Thus, a full examination (including a rectal examination) must be done.
- Diagnostic studies; may include:

- Bloods – FBC, CRP, ESR, coagulation studies
- Stool MC&S – *C. difficile*, enteric pathogens, ova and parasites
- RFTs and/or LFTs (in cases where related causes are suspected)
- Plain radiographs (to identify for foreign bodies)
- Abdominal USS
- Endoscopy (if the patient has brisk or unexplained bleeding after a thorough examination, or if s/he is in shock)
- Angiography (if the source of the bleeding could not be found on endoscopy)
- Colonoscopy

Management

Emergency Management

A gastroenterologist and general surgeon should be immediately called for any patient with severe acute UGIB. The patient should then be resuscitated and stabilised:

- If the child is shocked or has orthostatic hypotension (i.e. had a severe GI bleed), s/he should be admitted to ICU for resuscitation and close observation.
- Two large-bore IV catheters should be inserted and fluid boluses given. A transfusion may be required (if Hb <8 g/dL).
- Surgical intervention may be required for uncontrollable bleeds.

Follow-up visits should be scheduled, especially for first-time bleeders.

Routine Management

Patients who have had a UGIB:

- The patient should be resuscitated (ABCs) and the cause treated.
- Pharmacological management includes:
  - Acid suppression in clinically significant UGIB (IV PPIs or H_2-receptor antagonists).
  - Temporising the difficult to control bleed (e.g. variceal bleeding) with somatostatin and octreotide.
- Surgical management:

- Sengstaken-Blakemore tube placement.
- Endoscopic treatment (within 24-48 hours of presentation) e.g. sclerotherapy, elastic ligature (also used for haemorrhoids), transjugular intrahepatic portosystemic shunt (TIPS) for variceal bleeds.
- Catheter tamponade (used if medical management fails, to stop a continuously bleeding vessel which has been identified using a catheter; only perform in theatre or the ICU setting).

The cause of the lower GI bleed should be identified and treated. See also a figure on Deployed Sengstaken-Blakemore Tube in the Patient with an UGIB here.

FUNCTIONAL ABDOMINAL PAIN (FAP)

These disorders are the most common causes of chronic (>2 months) abdominal pain in children and adolescents.

Pathophysiology

The pathophysiology is poorly understood but it is thought to involve an interplay between enteric and CNS regulatory factors. These disorders may be associated with:

- Visceral hyperalgesia:
  - Eating may be associated with onset of pain and the patient is, therefore, likely to skip meals in an attempt to avoid pain.
- Reduced pain threshold
- Referred pain following rectal distension
- Impaired gastric relaxation response to meals

Diagnosis, Classification and Clinical Features

The diagnosis is made in the child with chronic abdominal pain, no danger signs, normal examination and stool which is negative for occult blood. Certain recognisable patterns of symptoms may be used to classify the FAP:

- Functional dyspepsia
- Irritable bowel syndrome (a disorder of large intestine) – cramping, abdominal pain, bloating, gas, diarrhoea and/or constipation

- Abdominal migraine (most common in children) – abdominal pain, nausea, vomiting, family or personal history of migraines
- FAP not otherwise specified

Management

It should be managed in a primary care setting. The goal of treatment is a return to normal function rather than complete elimination of pain. However, a referral may be made if the pain cannot be managed at a primary care level.

Management is individualised and depends on the child's and family behaviours, triggers and symptoms. Regardless of the subtype of FAP, the management includes:

- Assuring the patient and family that a treatment will be initiated and the patient (and family, as they may also be affected by the condition) followed up on a regular basis.
- Patient education:
    - FAPDs are best treated using the biopsychosocial model of care. Before starting therapy, one must define the expectation of the patient and parents and be realistic about treatment aims.
    - One must reassure the patient and family by acknowledging that the pain is real and has affected important activities in the patient's life.
    - They must be informed that FAP is common and can be exacerbated or made to persist by environment and psychosocial factors e.g. stress, anxiety, social reinforcement. However, they are not life threatening.
    - They must also be told that management focuses on rehabilitation rather than treatment, and includes avoiding triggers and improving coping skills.
- Prescribing a return to structured activities of daily living (including school):
    - School absenteeism adds to family stress and can interfere with school performance), see also an image of active children in a classroom.
    - One must implement a plan ahead for pains at school, such as keeping the first back at school short, arranging for the child to go to the nurse's office until the pain stops and use the bathroom whenever necessary.

A letter should be written to make the school aware of the condition and plan.
    - The family must be given guidelines for when the pain is severe enough to warrant going home or missing school.
    - School-related stressors must be identified and dealt with.
- Behaviour modification (positive, well behaviours should be reinforced, and triggers and behaviours that cause pain should be stopped/avoided).
- Strategies to improve pain tolerance and coping:
    - Psychological treatments which improve coping should form part of the management of children and adolescents with FAP e.g. relaxation techniques, distraction, and cognitive behavioural therapy (CBT).
    - Psychological interventions appear to be more effective than pharmacological interventions.
- Symptomatic management.