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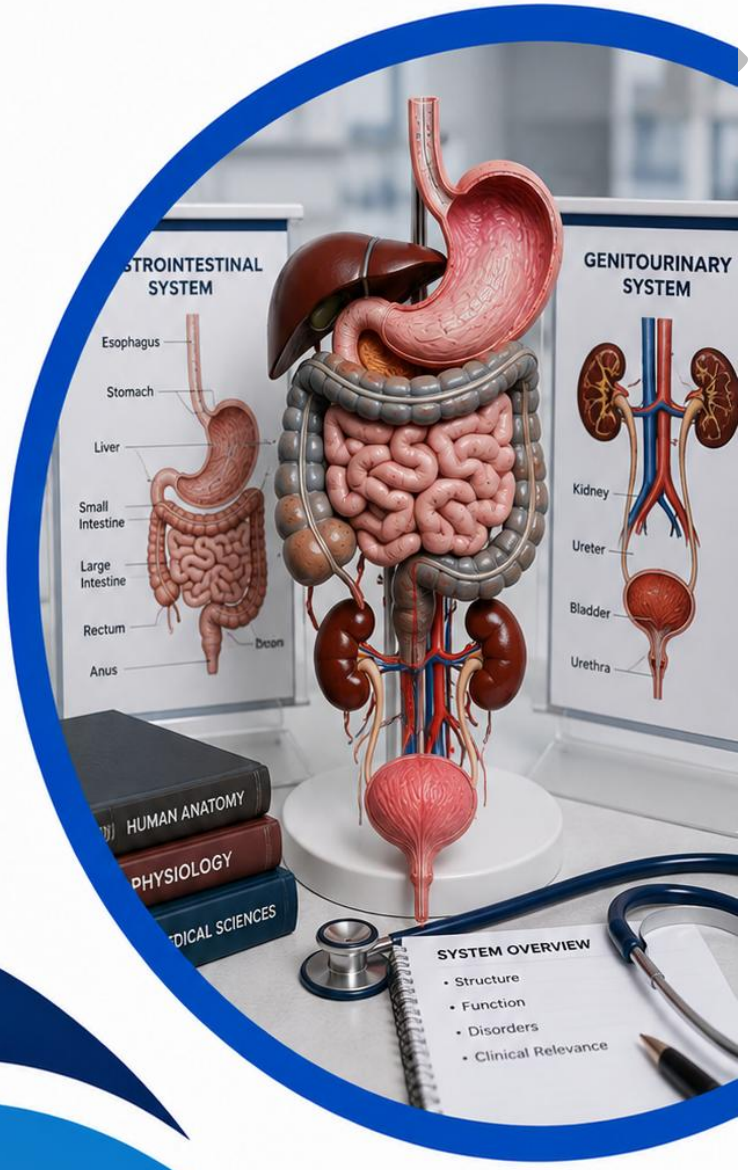
PAE 505

GASTROINTESTINAL AND GENITOURINARY SYSTEM



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Course code: PAE 505

Course title: Gastrointestinal and Genitourinary System

Course credit load: 2 Units

Series 1: Diarrhoeal Disease, Fluid Physiology, and Dehydration

- **Part 1.1: Diarrhoea in Children: Definition, Classification, and Aetiology**
  - Sub Part 1.1.1: Definition and mechanisms of diarrhoea
  - Sub Part 1.1.2: Classification of diarrhoea
  - Sub Part 1.1.3: Epidemiology and aetiology of diarrhoea
- **Part 1.2: Assessment, Complications, and Management of Diarrhoea**
  - Sub Part 1.2.1: Clinical assessment of a child with diarrhoea
  - Sub Part 1.2.2: Complications of diarrhoeal disease
  - Sub Part 1.2.3: General management and prevention of diarrhoea
- **Part 1.3: Vomiting and Fluid and Electrolyte Physiology in Children**
  - Sub Part 1.3.1: Causes and evaluation of vomiting in children
  - Sub Part 1.3.2: Complications and management of vomiting
  - Sub Part 1.3.3: Physiology of fluid and electrolyte distribution in children
- **Part 1.4: Dehydration and Oral Rehydration Therapy**
  - Sub Part 1.4.1: Types and severity of dehydration
  - Sub Part 1.4.2: Clinical assessment of hydration status
  - Sub Part 1.4.3: Oral rehydration therapy

Introduction

Picture an eighteen month old brought to a busy primary health centre by her mother, who reports three days of watery stool, poor feeding, and a child who now seems unusually quiet and less playful than usual. Findings such as these are among the most common reasons a child is brought to a clinician anywhere in the world, and **diarrhoeal disease** remains one of the leading causes of childhood illness and death globally, disproportionately affecting children in low resource settings such as much of Nigeria. This Series opens the PAE 505 course by building a

systematic understanding of diarrhoea, vomiting, and the fluid and electrolyte physiology that underlies the assessment and management of a dehydrated child.

The aim of this Series is to equip you with the knowledge required to classify and assess a child with diarrhoea or vomiting, to understand the basic physiology of fluid and electrolyte distribution in children, and to assess and manage dehydration, including through oral rehydration therapy.

This Series begins with the **definition, classification, and aetiology of diarrhoea**, before turning to its **assessment, complications, and management**. Next, we examine **vomiting** in children alongside the **physiology of fluid and electrolyte distribution**, before concluding with **dehydration** and **oral rehydration therapy**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 1.1:** describe the epidemiology and aetiology of diarrhoea,
- **SLO 1.2:** classify diarrhoea and explain the mechanisms underlying watery diarrhoea,
- **SLO 1.3:** describe the assessment of a child with diarrhoea and list its complications,
- **SLO 1.4:** discuss the association between diarrhoea and malnutrition, and strategies for prevention,
- **SLO 1.5:** describe the causes and evaluation of a child with vomiting,
- **SLO 1.6:** describe the physiology of fluid and electrolyte distribution in children,
- **SLO 1.7:** describe the types and severity of dehydration and its clinical assessment, and
- **SLO 1.8:** describe oral rehydration therapy, its mechanism of action, and the composition of oral rehydration solution.

1.1 Diarrhoea in Children: Definition, Classification, and Aetiology

1.1.1 definition and mechanisms of diarrhoea

Two mechanisms produce the same watery symptom

Diarrhoea is defined as the passage of three or more loose or watery stools in a 24 hour period, or a clear increase in stool frequency and looseness from what is normal for the individual child. Two broad mechanisms underlie watery diarrhoea: **osmotic diarrhoea**, in which poorly absorbed solutes remain within the bowel lumen and draw water into it, and **secretory**

diarrhoea, in which the intestinal mucosa actively secretes excess fluid and electrolytes into the lumen, often in response to a bacterial toxin.

A useful clinical distinction is that osmotic diarrhoea typically **improves with fasting**, since it depends on the ongoing presence of unabsorbed solute in the gut, while secretory diarrhoea **continues even when the child is fasted**, since the underlying secretory process is largely independent of oral intake, a distinction that can help narrow the differential diagnosis in a child with persistent diarrhoea.

Fill in the blank: Osmotic diarrhoea typically improves with _____, unlike secretory diarrhoea.

1.1.2 classification of diarrhoea

Diarrhoea is classified by **duration** into **acute diarrhoea**, lasting less than 14 days, the most common presentation and usually infectious in origin, **persistent diarrhoea**, lasting 14 days or more, and **chronic diarrhoea**, a term sometimes used for diarrhoea lasting beyond one month, often reflecting an underlying non-infectious cause such as malabsorption. Diarrhoea is also classified by **character** into watery diarrhoea, the most common type, and **dysentery**, diarrhoea containing visible blood, which suggests invasive bacterial infection such as **Shigella** and requires different management.

This dual classification, by both duration and character, is clinically important because it directly influences the likely causative organism, the necessary investigations, and the appropriate treatment approach, meaning the classification should always be actively established early in the assessment of any child presenting with diarrhoea.

Fill in the blank: Diarrhoea lasting 14 days or more is classified as _____ diarrhoea.

1.1.3 epidemiology and aetiology of diarrhoea

Diarrhoeal disease remains one of the leading causes of morbidity and mortality in children under five years of age globally, particularly in low and middle income countries, where poor access to **safe drinking water**, inadequate **sanitation**, and limited **hygiene practices** all contribute substantially to transmission. The most common infectious causes of acute watery diarrhoea in children include **rotavirus**, the leading viral cause, particularly in unvaccinated infants, together with bacterial causes such as **enterotoxigenic Escherichia coli** and, in dysentery, **Shigella** and **Campylobacter** species.

Non-infectious causes, while less common, include **dietary factors**, certain **medications** such as antibiotics disrupting normal gut flora, and, in persistent or chronic diarrhoea, underlying

conditions such as **malabsorption syndromes**, which should be considered when diarrhoea fails to resolve within the expected timeframe for an infectious cause.

Fill in the blank: _____ is the leading viral cause of acute watery diarrhoea in unvaccinated infants.

Table 1.1: Classification of diarrhoea by duration and character.

Classification	Definition	Typical implication
Acute diarrhoea	Lasting less than 14 days	Usually infectious, most commonly viral
Persistent diarrhoea	Lasting 14 days or more	May reflect ongoing infection or early malabsorption
Chronic diarrhoea	Lasting beyond one month	Often reflects a non-infectious underlying cause
Dysentery	Diarrhoea containing visible blood	Suggests invasive bacterial infection

Pilot questions 1.1

1. Define diarrhoea and distinguish osmotic from secretory diarrhoea. (Linked to SLO 1.1)
2. Explain why osmotic diarrhoea characteristically improves with fasting. (Linked to SLO 1.1)
3. Distinguish acute, persistent, and chronic diarrhoea by duration. (Linked to SLO 1.2)
4. What does the presence of visible blood in stool suggest, and what is this presentation called? (Linked to SLO 1.2)
5. Name the leading viral cause of acute watery diarrhoea in children. (Linked to SLO 1.1)

1.2 Assessment, Complications, and Management of Diarrhoea

1.2.1 clinical assessment of a child with diarrhoea

What the history and examination must establish

Assessment of a child with diarrhoea should establish the **duration**, **frequency**, and **character** of stools, including the presence of blood or mucus, together with associated symptoms such as **fever**, **vomiting**, and **poor oral intake**. A focused history should also explore potential sources of infection, including **recent travel**, **contact with other unwell children**, and the **home water**

and sanitation situation, since this context both supports the likely diagnosis and informs subsequent prevention advice.

Examination must always include a careful assessment of **hydration status**, discussed in detail later in this Series, together with assessment of the child's general condition, **nutritional status**, and **abdominal examination** to identify any signs of an acute abdomen or significant distension that might suggest an alternative or coexisting diagnosis requiring urgent attention.

Fill in the blank: Assessment of a child with diarrhoea must always include a careful assessment of _____ status.

1.2.2 complications of diarrhoeal disease

The most immediate and important complication of diarrhoea is **dehydration**, arising from ongoing fluid and electrolyte losses in the stool, and, if severe and untreated, progressing to **hypovolaemic shock**. Associated **electrolyte disturbances**, particularly **hyponatraemia** or **hypernatraemia** and **hypokalaemia**, may also develop, particularly with large volume losses or inappropriate fluid replacement, and require specific correction alongside general rehydration.

Beyond the acute episode, **persistent or recurrent diarrhoea** can significantly impair a child's **nutritional status**, contributing to **malnutrition** through reduced nutrient absorption, reduced appetite, and increased metabolic demand, which in turn increases susceptibility to further infections, creating a harmful cycle that is particularly important to recognise and interrupt in resource limited settings.

Fill in the blank: Severe, untreated dehydration from diarrhoea can progress to hypovolaemic _____.

1.2.3 general management and prevention of diarrhoea

Management of diarrhoea centres on **appropriate fluid replacement**, most commonly through **oral rehydration therapy**, discussed in detail later in this Series, together with **continued feeding**, since withholding food is not recommended and may worsen nutritional status without improving stool output. **Zinc supplementation** is also recommended in children with acute diarrhoea, since it has been shown to reduce the duration and severity of the episode and the risk of subsequent episodes over the following months.

Prevention strategies include promotion of **exclusive breastfeeding** in infancy, improved access to **safe drinking water** and **sanitation**, good **hand hygiene**, and **rotavirus vaccination**, all of which have been shown to substantially reduce the burden of diarrhoeal disease in children when implemented consistently at a population level.

Fill in the blank: _____ supplementation reduces the duration and severity of an episode of acute diarrhoea.

Box 1.2: Key complications of diarrhoeal disease to actively assess for.

Key complications of diarrhoea
Dehydration: from ongoing fluid loss, ranging from mild to severe with hypovolaemic shock.
Electrolyte disturbance: hyponatraemia, hypernatraemia, or hypokalaemia from fluid and electrolyte losses.
Malnutrition: from reduced absorption, reduced appetite, and increased metabolic demand.
Secondary infection: increased susceptibility due to impaired nutritional and immune status.
Prolonged or recurrent illness: particularly where underlying malnutrition is not addressed.

Pilot questions 1.2

1. List the key features to establish when taking a history from a child with diarrhoea. (Linked to SLO 1.3)
2. List the major complications of diarrhoeal disease. (Linked to SLO 1.3)
3. Explain the relationship between diarrhoea and malnutrition. (Linked to SLO 1.4)
4. Describe two components of general management of a child with acute diarrhoea. (Linked to SLO 1.4)
5. List two strategies for the prevention of diarrhoeal disease at a population level. (Linked to SLO 1.4)

1.3 Vomiting and Fluid and Electrolyte Physiology in Children

1.3.1 causes and evaluation of vomiting in children

Distinguishing benign from concerning vomiting

Vomiting in children has a broad differential diagnosis that varies considerably by age, ranging from benign, self-limiting causes such as **gastroenteritis** and **overfeeding** in infants, to more concerning causes such as **intestinal obstruction**, **raised intracranial pressure**, and, in infants, **pyloric stenosis**, which classically presents with progressively worsening, **non-bilious, projectile vomiting** in the first weeks of life.

Evaluation should establish the **character** of the vomiting, particularly whether it is **bilious**, which strongly suggests intestinal obstruction and requires urgent evaluation, the **timing** in relation to feeds, and any associated symptoms such as **fever**, **abdominal pain**, **headache**, or

neurological symptoms, which may point towards an underlying cause beyond the gastrointestinal tract itself.

Fill in the blank: Bilious vomiting strongly suggests intestinal _____ and requires urgent evaluation.

1.3.2 complications and management of vomiting

Like diarrhoea, persistent or severe vomiting can lead to significant **dehydration** and **electrolyte disturbance**, particularly **hypochloraemic, hypokalaemic metabolic alkalosis** in conditions such as pyloric stenosis, where repeated loss of hydrochloric acid rich gastric contents produces this characteristic pattern. Assessment should therefore always include the same careful attention to hydration status as for diarrhoea.

Management depends entirely on the underlying cause, ranging from **simple observation and small, frequent feeds** for mild, self-limiting vomiting, to **fluid resuscitation** and **specific treatment of the underlying cause** for more significant presentations, and any child with bilious vomiting, signs of obstruction, or associated neurological symptoms requires urgent further evaluation and, frequently, surgical or specialist input.

Fill in the blank: Pyloric stenosis classically causes a hypochloraemic, hypokalaemic metabolic _____.

1.3.3 physiology of fluid and electrolyte distribution in children

Total body water makes up a greater proportion of body weight in children, particularly infants, than in adults, and is distributed between the **intracellular fluid compartment**, comprising roughly two thirds of total body water, and the **extracellular fluid compartment**, comprising the remaining third and further divided into the **intravascular** and **interstitial** spaces. This higher proportion of body water, combined with a relatively greater **surface area to body weight ratio** and higher **metabolic rate**, means infants and young children lose fluid more rapidly relative to their size and can become dehydrated considerably faster than an older child or adult in a comparable illness.

Sodium is the principal extracellular cation and the main determinant of extracellular fluid volume, while **potassium** is the principal intracellular cation, and maintaining the normal distribution and concentration of these electrolytes across cell membranes is essential for normal cellular function, nerve conduction, and muscle contraction, making a solid understanding of this physiology essential before approaching the assessment and management of dehydration.

Fill in the blank: Sodium is the principal _____ cation and the main determinant of extracellular fluid volume.

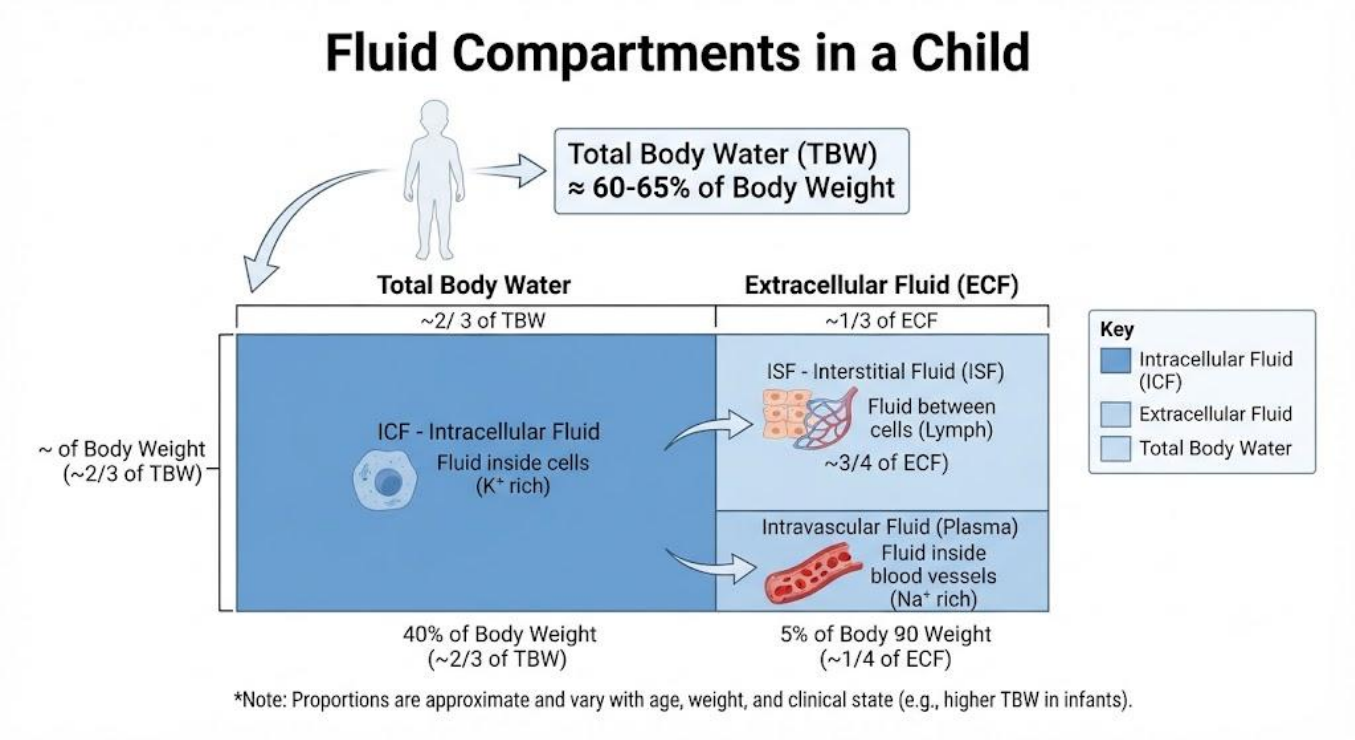

Figure 1.3: Fluid compartments and their approximate proportion of total body water in a child.

Pilot questions 1.3

1. List two benign and two concerning causes of vomiting in children. (Linked to SLO 1.5)
2. Describe the classic presentation of pyloric stenosis. (Linked to SLO 1.5)
3. Explain the electrolyte disturbance classically associated with pyloric stenosis. (Linked to SLO 1.5)
4. Describe the distribution of total body water between the intracellular and extracellular compartments. (Linked to SLO 1.6)
5. Explain why infants become dehydrated more rapidly than older children for a comparable illness. (Linked to SLO 1.6)

1.4 Dehydration and Oral Rehydration Therapy

1.4.1 types and severity of dehydration

Why the sodium level matters as much as the volume lost

Dehydration can be classified by the **type of fluid lost** relative to sodium concentration into **isonatraemic**, the most common type, in which water and sodium are lost in roughly proportionate amounts, **hyponatraemic**, in which relatively more sodium than water is lost, and **hypernatraemic**, in which relatively more water than sodium is lost, each with distinct clinical features and specific considerations for safe fluid replacement, particularly regarding the **rate** of correction in hypernatraemic dehydration.

Dehydration is also classified by **severity**, generally described as **mild**, **moderate**, or **severe**, based on the estimated percentage of body weight lost as fluid, with severe dehydration representing a medical emergency requiring urgent intravenous fluid resuscitation rather than oral rehydration alone.

Fill in the blank: In hypernatraemic dehydration, relatively more _____ than sodium is lost.

1.4.2 clinical assessment of hydration status

Clinical assessment of hydration status relies on a combination of history and examination findings, including **general appearance and alertness**, **skin turgor**, **capillary refill time**, **mucous membrane moisture**, presence or absence of **tears**, and **eye appearance**, since sunken eyes are a recognised sign of at least some dehydration. **Sunken fontanelle** in an infant, **reduced urine output**, and, in more severe cases, **cold extremities** and **weak or absent peripheral pulses** indicate progressively more severe dehydration.

No single sign reliably determines the degree of dehydration in isolation, so clinical assessment relies on considering the **combination** of findings together, and structured assessment tools, such as those used within the **Integrated Management of Childhood Illness** approach, provide a systematic way of combining these findings to classify a child as having no dehydration, some dehydration, or severe dehydration, guiding the appropriate management pathway.

Fill in the blank: Assessment of hydration status relies on considering the _____ of clinical findings together, not any single sign alone.

1.4.3 oral rehydration therapy

Oral rehydration therapy, abbreviated ORT, is the cornerstone of managing mild to moderate dehydration from diarrhoea, exploiting the physiological principle that **glucose-coupled sodium absorption** remains intact in the small intestine even during acute diarrhoeal illness, allowing water to be absorbed alongside sodium and glucose when given in the correct proportions, even though the bowel's usual absorptive capacity is otherwise impaired.

Oral rehydration solution, or ORS, has a specific, evidence-based composition of **glucose**, **sodium**, **potassium**, and **citrate** in carefully balanced concentrations, and the current **low-osmolarity formulation** has been shown to reduce stool output, vomiting, and the need for unplanned intravenous fluids compared with the original, higher-osmolarity formulation, making it the preferred formulation recommended for routine use in managing acute diarrhoea in children.

Fill in the blank: ORT exploits the physiological principle of glucose-coupled _____ absorption in the small intestine.

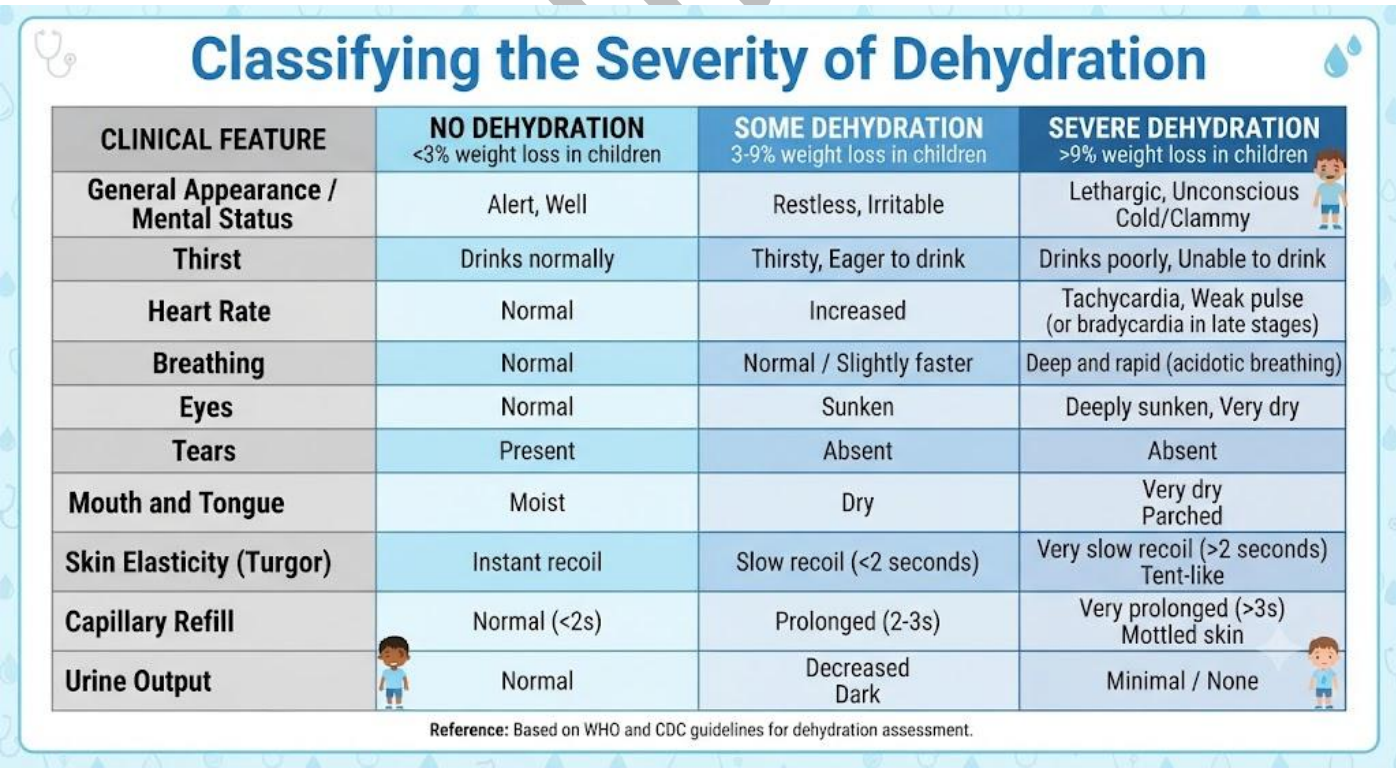

Figure 1.4: Clinical features used to classify the severity of dehydration.

Pilot questions 1.4

1. Distinguish isonatraemic, hyponatraemic, and hypernatraemic dehydration. (Linked to SLO 1.7)
2. List five clinical signs used to assess hydration status. (Linked to SLO 1.7)
3. Why should no single clinical sign be relied upon alone to assess dehydration severity? (Linked to SLO 1.7)
4. Explain the physiological principle underlying oral rehydration therapy. (Linked to SLO 1.8)
5. Describe the advantage of low-osmolarity ORS over the original, higher-osmolarity formulation. (Linked to SLO 1.8)

Series Summary

This Series introduced **diarrhoeal disease** in children, beginning with its definition, classification, and aetiology, before turning to its **assessment, complications, and management**, including the important relationship between diarrhoea and malnutrition.

We then examined **vomiting** in children alongside the underlying **physiology of fluid and electrolyte distribution**, before concluding with the classification and assessment of **dehydration** and the principles of **oral rehydration therapy**. Having worked through this Series, you should now be able to assess and manage a child with diarrhoea, vomiting, or dehydration. The next Series turns to **electrolyte and acid-base disorders in paediatric fluid therapy**.

Glossary of Terms

- **Osmotic diarrhoea:** diarrhoea caused by poorly absorbed solutes drawing water into the bowel lumen, characteristically improving with fasting.
- **Secretory diarrhoea:** diarrhoea caused by active intestinal secretion of fluid and electrolytes, continuing despite fasting.
- **Dysentery:** diarrhoea containing visible blood, suggestive of invasive bacterial infection.
- **Rotavirus:** the leading viral cause of acute watery diarrhoea in unvaccinated infants.
- **Pyloric stenosis:** a condition causing progressively worsening, non-bilious, projectile vomiting in early infancy.
- **Bilious vomiting:** vomiting containing bile, strongly suggestive of intestinal obstruction.
- **Intracellular fluid:** the fluid compartment within cells, comprising roughly two thirds of total body water.
- **Extracellular fluid:** the fluid compartment outside cells, comprising intravascular and interstitial fluid.
- **Isonatraemic dehydration:** dehydration in which water and sodium are lost in roughly proportionate amounts.
- **Hypernatraemic dehydration:** dehydration in which relatively more water than sodium is lost.
- **Oral rehydration therapy:** the use of oral rehydration solution to correct mild to moderate dehydration, exploiting glucose-coupled sodium absorption.
- **Oral rehydration solution:** a specific balanced solution of glucose, sodium, potassium, and citrate used in oral rehydration therapy.

Concept Check Questions [Series 1]

Objective questions

1. Osmotic diarrhoea typically improves with _____, unlike secretory diarrhoea.
2. Diarrhoea lasting 14 days or more is classified as _____ diarrhoea.
3. Bilious vomiting strongly suggests intestinal _____.
4. Pyloric stenosis classically causes a hypochloraemic, hypokalaemic metabolic _____.
5. ORT exploits the physiological principle of glucose-coupled _____ absorption in the small intestine.

Short-answer questions

6. Distinguish osmotic from secretory diarrhoea.
7. List the major complications of diarrhoeal disease.
8. Describe the classic presentation of pyloric stenosis.
9. List five clinical signs used to assess hydration status in a child.
10. Describe the composition of oral rehydration solution.

Long-answer questions

11. Discuss the classification and aetiology of diarrhoea in children. (Linked to SLO 1.1, SLO 1.2)
12. Discuss the assessment, complications, and management of a child with diarrhoea. (Linked to SLO 1.3, SLO 1.4)
13. Describe the causes and evaluation of a child presenting with vomiting. (Linked to SLO 1.5)
14. Discuss the physiology of fluid and electrolyte distribution in children. (Linked to SLO 1.6)
15. Discuss the classification of dehydration and the principles of oral rehydration therapy. (Linked to SLO 1.7, SLO 1.8)

Answers to Concept Check Questions [Series 1]

Objective questions

1. Fasting.
2. Persistent.

3. Obstruction.
4. Alkalosis.
5. Sodium.

Short-answer questions

6. Osmotic diarrhoea results from poorly absorbed solutes drawing water into the bowel and improves with fasting, while secretory diarrhoea results from active fluid secretion and continues despite fasting.
7. Dehydration, electrolyte disturbance, and malnutrition are the major complications of diarrhoeal disease.
8. Progressively worsening, non-bilious, projectile vomiting in the first weeks of life.
9. General appearance, skin turgor, capillary refill time, mucous membrane moisture, and eye appearance.
10. Glucose, sodium, potassium, and citrate in carefully balanced concentrations.

Long-answer questions

11. **Basic response:** Diarrhoea is classified by duration into acute, persistent, and chronic, and by character into watery diarrhoea and dysentery, with common causes including rotavirus and enterotoxigenic Escherichia coli.

Value-added response: This classification directly influences the likely causative organism and appropriate management, with dysentery in particular suggesting invasive bacterial infection requiring different treatment.

12. **Basic response:** Assessment establishes duration, frequency, and character of stools alongside hydration status, and management centres on oral rehydration, continued feeding, and zinc supplementation.

Value-added response: Persistent or recurrent diarrhoea can significantly impair nutritional status, creating a harmful cycle with increased susceptibility to further infection, making prevention through breastfeeding, sanitation, and vaccination particularly important.

13. **Basic response:** Vomiting evaluation establishes the character, timing, and associated symptoms, with bilious vomiting strongly suggesting intestinal obstruction requiring urgent evaluation.

Value-added response: Pyloric stenosis classically causes non-bilious, projectile vomiting with a characteristic hypochloraemic, hypokalaemic metabolic alkalosis from loss of gastric acid.

14. **Basic response:** Total body water is distributed between the intracellular compartment, roughly two thirds of the total, and the extracellular compartment, further divided into intravascular and interstitial spaces.

Value-added response: Infants have a higher proportion of body water and greater surface area to weight ratio, meaning they lose fluid more rapidly relative to their size and become dehydrated faster than older children.

15. **Basic response:** Dehydration is classified by sodium balance into isonatraemic, hyponatraemic, and hypernatraemic, and by severity into mild, moderate, and severe.

Value-added response: Oral rehydration therapy exploits intact glucose-coupled sodium absorption even during diarrhoeal illness, and low-osmolarity ORS reduces stool output, vomiting, and the need for intravenous fluids compared with the original formulation.

Answers to Pilot Questions [Series 1]

Part 1.1

1. Diarrhoea is the passage of three or more loose or watery stools in 24 hours; osmotic diarrhoea results from unabsorbed solutes, secretory diarrhoea from active fluid secretion.
2. Osmotic diarrhoea depends on the ongoing presence of unabsorbed solute in the gut, which fasting removes.
3. Acute diarrhoea lasts less than 14 days, persistent diarrhoea 14 days or more, and chronic diarrhoea beyond one month.
4. Visible blood suggests invasive bacterial infection, and this presentation is called dysentery.
5. Rotavirus is the leading viral cause of acute watery diarrhoea in unvaccinated infants.

Part 1.2

1. Duration, frequency, character of stools, associated fever or vomiting, and sanitation context.
2. Dehydration, electrolyte disturbance, and malnutrition are the major complications.
3. Diarrhoea impairs nutrient absorption and appetite while increasing metabolic demand, contributing to malnutrition, which in turn increases susceptibility to further infection.
4. Oral rehydration therapy and continued feeding are key components of management.

5. Improved sanitation and access to safe drinking water, and rotavirus vaccination, are effective prevention strategies.

Part 1.3

1. Benign causes include gastroenteritis and overfeeding; concerning causes include intestinal obstruction and raised intracranial pressure.
2. Pyloric stenosis presents with progressively worsening, non-bilious, projectile vomiting in early infancy.
3. Pyloric stenosis causes a hypochloraemic, hypokalaemic metabolic alkalosis from loss of gastric acid.
4. Roughly two thirds of total body water is intracellular, and one third is extracellular, divided into intravascular and interstitial spaces.
5. Infants have a higher proportion of body water and greater surface area to weight ratio, causing faster relative fluid loss.

Part 1.4

1. Isonatraemic dehydration involves proportionate water and sodium loss, hyponatraemic involves relatively more sodium loss, and hypernatraemic involves relatively more water loss.
2. General appearance, skin turgor, capillary refill time, mucous membrane moisture, and eye appearance are among the signs used.
3. No single sign reliably determines severity alone, so findings must be considered together for an accurate classification.
4. ORT relies on intact glucose-coupled sodium absorption in the small intestine, allowing water absorption despite ongoing diarrhoea.
5. Low-osmolarity ORS reduces stool output, vomiting, and the need for unplanned intravenous fluids compared with the original formulation.

References and Attributions [Series 1]

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- Guarino, A. et al. (2014). European Society for Paediatric Gastroenterology, Hepatology, and Nutrition Guidelines for the Management of Acute Gastroenteritis in Children. Journal of Pediatric Gastroenterology and Nutrition, 59(1), 132 to 152.

Figures, Plates, Tables, and Boxes

- Table 1.1: Classification of diarrhoea by duration and character. AI-generated using the prompt: "Create a clean reference table titled Classification of Diarrhoea, listing classification, definition, and typical implication. Simple clinical reference table style with outside border."
- Box 1.2: Key complications of diarrhoeal disease to actively assess for. AI-generated using the prompt: "Create a simple single-column reference box titled Key Complications of Diarrhoeal Disease. Clean clinical reference box style."
- Figure 1.3: Fluid compartments and their approximate proportion of total body water in a child. AI-generated using the prompt: "Create a professional medical diagram titled Fluid Compartments in a Child, showing intracellular and extracellular fluid with intravascular and interstitial subdivisions and approximate proportions. Clean clinical educational diagram style."
- Figure 1.4: Clinical features used to classify the severity of dehydration. AI-generated using the prompt: "Create a professional medical reference chart titled Classifying the Severity of Dehydration, listing clinical features across no, some, and severe dehydration columns. Clean clinical educational diagram style."

Series 2: Electrolyte and Acid-Base Disorders in Paediatric Fluid Therapy

- **Part 2.1: Fluid Types and Principles of Fluid Therapy**
  - Sub Part 2.1.1: Fluid types used in paediatrics
  - Sub Part 2.1.2: Calculating maintenance fluid requirements
  - Sub Part 2.1.3: Deficit and ongoing loss replacement
- **Part 2.2: Sodium and Potassium Disorders in Children**
  - Sub Part 2.2.1: Hyponatraemia
  - Sub Part 2.2.2: Hypernatraemia
  - Sub Part 2.2.3: Hypokalaemia and hyperkalaemia
- **Part 2.3: Acid-Base Balance and Disorders**
  - Sub Part 2.3.1: Physiology of acid-base balance
  - Sub Part 2.3.2: Metabolic and respiratory acidosis
  - Sub Part 2.3.3: Alkalosis and correction of acidosis
- **Part 2.4: Differentiating Dehydration from Other Fluid Disorders**
  - Sub Part 2.4.1: Distinguishing dehydration from fluid overload
  - Sub Part 2.4.2: Syndrome of inappropriate antidiuretic hormone secretion and diabetes insipidus
  - Sub Part 2.4.3: A structured approach to the child with a fluid disorder

Introduction

In the last Series, we explored diarrhoeal disease, vomiting, and the basic assessment and management of dehydration. We now build on that foundation by examining the fluids used to treat a dehydrated or unwell child, and the electrolyte and acid-base disturbances that so often accompany fluid loss. Picture a two year old admitted with severe diarrhoea whose blood results return showing a low sodium and a low bicarbonate. Understanding what these results mean, and how to correct them safely, is the focus of this Series.

The aim of this Series is to equip you with the knowledge required to select appropriate intravenous fluids, apply the principles of paediatric fluid therapy, and recognise and manage the common electrolyte and acid-base disorders encountered in unwell children.

This Series begins with the **types of fluids** used in paediatrics and the **principles of fluid therapy**, before turning to **sodium and potassium disorders**. Next, we examine **acid-base balance and its disorders**, before concluding by **differentiating dehydration** from other disorders of fluid balance.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 1.1:** list the types of intravenous fluids used in paediatrics and their uses,
- **SLO 1.2:** describe the principles of fluid therapy in paediatrics, including maintenance, deficit, and ongoing losses,
- **SLO 1.3:** describe the causes, clinical features, and management of hyponatraemia and hypernatraemia,
- **SLO 1.4:** describe the causes, clinical features, and management of hypokalaemia and hyperkalaemia,
- **SLO 1.5:** describe the physiology of acid-base balance in children,
- **SLO 1.6:** differentiate metabolic and respiratory acidosis and alkalosis,
- **SLO 1.7:** describe the principles of correcting base deficit and acidosis in children, and
- **SLO 1.8:** differentiate dehydration from other disorders of fluid balance in paediatrics.

2.1 Fluid Types and Principles of Fluid Therapy

2.1.1 fluid types used in paediatrics

Crystalloids remain the mainstay of paediatric fluid therapy

Intravenous fluids used in paediatric practice are broadly divided into **crystalloids**, solutions of small molecules such as electrolytes and glucose that distribute freely across the vascular membrane, and **colloids**, larger molecules that remain within the intravascular space for longer, though colloids are used far less commonly in routine paediatric fluid therapy. Commonly used crystalloids include **0.9 percent sodium chloride**, or normal saline, **Ringer's lactate**, a balanced solution containing sodium, potassium, calcium, chloride, and lactate, and various **dextrose-containing solutions**, such as 5 percent dextrose in 0.45 percent or 0.9 percent saline.

The choice of fluid depends on the clinical indication: **isotonic crystalloids**, such as normal saline or Ringer's lactate, are generally preferred for **fluid resuscitation** and replacement of ongoing losses, since they remain within the extracellular space and effectively restore

circulating volume, while **maintenance fluids** for a child who is not significantly dehydrated but requires ongoing intravenous fluid typically include a lower sodium concentration alongside dextrose to meet basal glucose requirements.

Fill in the blank: Isotonic crystalloids such as normal saline are generally preferred for fluid _____.

2.1.2 calculating maintenance fluid requirements

Maintenance fluid requirements in children are commonly calculated using the **Holliday-Segar formula**, based on body weight: **100 millilitres per kilogram** for the first 10 kilograms of body weight, **50 millilitres per kilogram** for the next 10 kilograms, and **20 millilitres per kilogram** for each kilogram above 20 kilograms, giving a total daily maintenance volume that can then be divided by 24 to give an hourly rate.

This formula reflects normal insensible losses and urine output for a healthy child at rest, and requires **adjustment** in specific clinical circumstances, such as **fever**, which increases insensible losses and therefore fluid requirements, or conditions associated with **fluid retention**, such as heart failure or acute kidney injury, where maintenance volumes must be reduced to avoid fluid overload.

Fill in the blank: The Holliday-Segar formula calculates maintenance fluid requirements based on a child's _____.

2.1.3 deficit and ongoing loss replacement

In addition to maintenance requirements, a dehydrated child requires replacement of the **existing fluid deficit**, estimated from the assessed percentage dehydration multiplied by body weight, and of **ongoing losses**, such as continuing diarrhoea or vomiting, which should be measured or estimated and replaced in addition to maintenance and deficit replacement, since failing to account for ongoing losses can result in a child who appears to be receiving adequate fluid but remains persistently dehydrated.

Total fluid therapy in an unwell child is therefore generally considered as the sum of three components, **maintenance**, **deficit replacement**, and **ongoing losses**, each calculated separately and then combined into an overall fluid prescription, with the **rate and type** of fluid adjusted according to the clinical picture and any electrolyte abnormalities identified on laboratory testing.

Fill in the blank: Total paediatric fluid therapy is generally considered the sum of maintenance, deficit replacement, and _____ losses.

Table 2.1: Common intravenous fluids used in paediatric practice.

Fluid	Composition summary	Typical use
0.9% sodium chloride (normal saline)	Isotonic, sodium 154 mmol/L	Resuscitation and deficit replacement
Ringer's lactate	Balanced isotonic solution with lactate	Resuscitation, especially in theatre and emergency settings
5% dextrose in 0.45% saline	Half-isotonic saline with dextrose	Maintenance fluid in many children
5% dextrose in water	Free water with glucose, no electrolytes	Rarely used alone; risk of hyponatraemia

Pilot questions 2.1

- Distinguish crystalloids from colloids and name two commonly used crystalloids. (Linked to SLO 2.1)
- Which type of fluid is generally preferred for fluid resuscitation, and why? (Linked to SLO 2.1)
- State the Holliday-Segar formula for calculating maintenance fluid requirements. (Linked to SLO 2.2)
- Give one example of a clinical circumstance requiring adjustment of maintenance fluid volume. (Linked to SLO 2.2)
- List the three components that together make up total paediatric fluid therapy. (Linked to SLO 2.2)

2.2 Sodium and Potassium Disorders in Children

2.2.1 hyponatraemia

Correcting sodium too quickly can be dangerous

Hyponatraemia, defined as a serum sodium below 135 millimoles per litre, may result from **excess water intake or retention** relative to sodium, as occurs with inappropriate use of hypotonic intravenous fluids or the syndrome of inappropriate antidiuretic hormone secretion, or from **excess sodium loss**, as occurs with diarrhoea, vomiting, or certain kidney conditions. Clinical features range from **asymptomatic**, mild hyponatraemia to **lethargy**, **seizures**, and, in

severe or rapidly developing cases, **coma**, reflecting cerebral oedema from the osmotic shift of water into brain cells.

Management depends on the severity and rate of onset, but a critical principle is that **chronic hyponatraemia must be corrected slowly**, generally no faster than 8 to 10 millimoles per litre in 24 hours, since overly rapid correction risks **osmotic demyelination syndrome**, a severe and potentially permanent neurological injury, whereas acute, symptomatic hyponatraemia with seizures may require more urgent, carefully monitored correction with hypertonic saline.

Fill in the blank: Overly rapid correction of chronic hyponatraemia risks osmotic _____ syndrome.

2.2.2 hypernatraemia

Hypernatraemia, defined as a serum sodium above 145 millimoles per litre, most commonly results from **water loss in excess of sodium loss**, as occurs with hypernatraemic dehydration from diarrhoea, or, less commonly, from **excess sodium intake**, such as inappropriate preparation of infant formula or oral rehydration solution. Clinical features include **irritability**, a **high-pitched cry**, and, in severe cases, **seizures**, though seizures in hypernatraemia more typically occur during **correction** rather than at presentation.

As with hyponatraemia, **correction must proceed slowly**, since rapid lowering of serum sodium causes water to shift rapidly into brain cells that have adapted to the hypertonic environment, risking **cerebral oedema** and seizures; the sodium should generally be lowered no faster than 10 to 12 millimoles per litre over 24 hours, using an appropriately calculated, gradually administered fluid regimen.

Fill in the blank: In hypernatraemia, seizures more typically occur during _____ rather than at presentation.

2.2.3 hypokalaemia and hyperkalaemia

Hypokalaemia, a serum potassium below 3.5 millimoles per litre, commonly results from **gastrointestinal losses**, such as diarrhoea or vomiting, or from **diuretic use**, and can cause **muscle weakness**, **cardiac arrhythmias**, and characteristic **ECG changes** including flattened T waves and prominent U waves. Correction involves careful, monitored **potassium replacement**, generally added to intravenous fluids once adequate urine output is confirmed, since giving potassium to a child with poor renal function or oliguria risks dangerous **hyperkalaemia**.

Hyperkalaemia, a serum potassium above 5.5 millimoles per litre, is potentially more immediately dangerous than hypokalaemia, since it can cause life-threatening **cardiac**

arrhythmias, with characteristic ECG changes including **peaked T waves**, widened QRS complex, and, in severe cases, a sine wave pattern preceding cardiac arrest. Emergency management includes **calcium gluconate** to stabilise the cardiac membrane, together with measures to shift potassium intracellularly, such as **insulin with glucose** and **salbutamol nebulisation**, alongside identifying and treating the underlying cause.

Fill in the blank: Calcium gluconate is given in severe hyperkalaemia to stabilise the _____ membrane.

Box 2.2: Emergency management steps for severe, symptomatic hyperkalaemia.

Emergency management of severe hyperkalaemia
Calcium gluconate: stabilises the cardiac membrane, does not lower potassium.
Insulin with glucose: shifts potassium into cells, lowering serum levels temporarily.
Salbutamol nebulisation: also shifts potassium intracellularly as an adjunct measure.
Continuous ECG monitoring: to detect worsening arrhythmia during treatment.
Identify and treat the underlying cause: to achieve definitive correction.

Pilot questions 2.2

1. Define hyponatraemia and list two possible causes. (Linked to SLO 2.3)
2. Explain why chronic hyponatraemia must be corrected slowly. (Linked to SLO 2.3)
3. Define hypernatraemia and describe its typical clinical features. (Linked to SLO 2.3)
4. List two ECG changes seen in hypokalaemia. (Linked to SLO 2.4)
5. Describe the emergency management of severe hyperkalaemia. (Linked to SLO 2.4)

2.3 Acid-Base Balance and Disorders

2.3.1 physiology of acid-base balance

Three systems keep blood pH within a narrow range

Normal acid-base balance is maintained through the interaction of **buffer systems**, particularly the **bicarbonate buffer system**, **respiratory compensation**, through changes in the rate and depth of breathing that alter carbon dioxide elimination, and **renal compensation**, through the kidney's ability to excrete or retain hydrogen ions and bicarbonate, together keeping arterial blood pH within the tightly regulated normal range of approximately 7.35 to 7.45.

Acidosis refers to a process tending to lower blood pH, and **alkalosis** to a process tending to raise it, and each may be further classified as **metabolic**, arising from a primary change in bicarbonate concentration, or **respiratory**, arising from a primary change in carbon dioxide, with the non-affected system typically attempting **compensation** in the opposite direction to minimise the overall change in pH.

Fill in the blank: Normal arterial blood pH is tightly regulated within the range of approximately 7.35 to _____.

2.3.2 metabolic and respiratory acidosis

Metabolic acidosis, a primary fall in bicarbonate, commonly results from **excess acid production**, as in diabetic ketoacidosis or severe sepsis with lactic acidosis, **loss of bicarbonate**, as in severe diarrhoea, or **impaired renal acid excretion**, as in acute or chronic kidney disease. The **anion gap**, calculated from serum sodium minus the sum of chloride and bicarbonate, helps distinguish causes, with a raised anion gap suggesting excess acid production and a normal anion gap suggesting bicarbonate loss or a renal cause.

Respiratory acidosis, a primary rise in carbon dioxide, results from **inadequate ventilation**, as in severe respiratory failure, airway obstruction, or central nervous system depression affecting the respiratory drive, and the kidney compensates over time by retaining bicarbonate, though this renal compensation takes hours to days to become fully effective, meaning acute respiratory acidosis is often only partially compensated at presentation.

Fill in the blank: A raised anion gap in metabolic acidosis suggests excess _____ production.

2.3.3 alkalosis and correction of acidosis

Metabolic alkalosis, a primary rise in bicarbonate, commonly results from **loss of gastric acid**, as in severe or prolonged vomiting, particularly in pyloric stenosis, or from **excessive diuretic use**, while **respiratory alkalosis**, a primary fall in carbon dioxide, results from **hyperventilation**, whether from anxiety, pain, fever, or, importantly, as a compensatory response to an underlying metabolic acidosis, which must always be considered before attributing hyperventilation to a primary respiratory cause.

Correction of acidosis in a child focuses primarily on treating the **underlying cause**, such as fluid resuscitation in shock or insulin therapy in diabetic ketoacidosis, rather than direct

bicarbonate administration in most cases, since correcting the underlying process typically resolves the acidosis, and inappropriate bicarbonate use can cause its own complications, including a paradoxical worsening of intracellular acidosis in some circumstances.

Fill in the blank: Correction of acidosis in a child focuses primarily on treating the underlying _____.

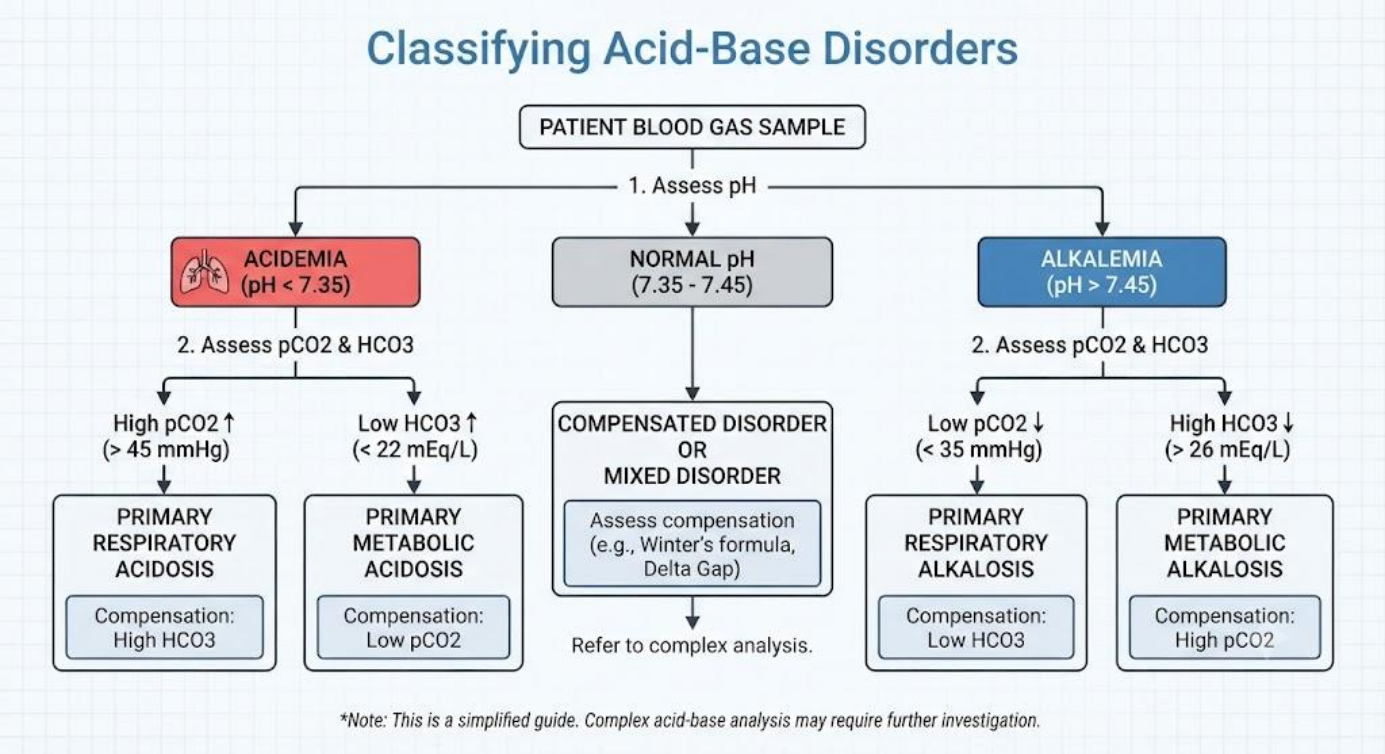

Figure 2.3: A simple approach to classifying acid-base disorders using pH, bicarbonate, and carbon dioxide.

Pilot questions 2.3

1. Describe the three systems that maintain normal acid-base balance. (Linked to SLO 2.5)
2. Distinguish metabolic from respiratory acidosis. (Linked to SLO 2.6)
3. Explain how the anion gap helps distinguish causes of metabolic acidosis. (Linked to SLO 2.6)
4. Describe two causes of metabolic alkalosis. (Linked to SLO 2.6)
5. Explain the primary approach to correcting acidosis in a child. (Linked to SLO 2.7)

2.4 Differentiating Dehydration from Other Fluid Disorders

2.4.1 distinguishing dehydration from fluid overload

Two opposite fluid states can share overlapping features

While much of this course focuses on **dehydration**, it is equally important to recognise **fluid overload**, in which excess fluid accumulates in the body, presenting with **oedema**, **weight gain**, **hepatomegaly**, and, in severe cases, **pulmonary oedema** with respiratory distress. Fluid overload commonly arises in the context of **excessive intravenous fluid administration**, particularly in children with underlying **cardiac**, **renal**, or **hepatic** disease who have a reduced capacity to handle a normal fluid load.

Distinguishing dehydration from fluid overload relies on careful clinical assessment, since some features, such as poor feeding or lethargy, can occur in both states, but the overall clinical picture, particularly the presence or absence of **oedema**, **weight trend**, and **respiratory findings**, generally allows the two to be clearly distinguished with careful examination.

Fill in the blank: Fluid overload commonly arises in children with underlying cardiac, renal, or _____ disease.

2.4.2 syndrome of inappropriate antidiuretic hormone secretion and diabetes insipidus

The **syndrome of inappropriate antidiuretic hormone secretion**, abbreviated SIADH, causes **water retention** and **dilutional hyponatraemia** despite normal or increased total body water, distinguishing it from dehydration, and typically arises in the context of central nervous system disorders, pneumonia, or certain medications, with management centred on **fluid restriction** rather than fluid replacement.

Diabetes insipidus, by contrast, results from either inadequate **antidiuretic hormone secretion**, termed central diabetes insipidus, or inadequate renal **response** to antidiuretic hormone, termed nephrogenic diabetes insipidus, both causing excessive **dilute urine output** and a tendency towards hypernatraemic dehydration if free water intake cannot keep pace with ongoing urinary losses, a pattern that can be distinguished from typical dehydration by the disproportionately large urine volume relative to intake.

Fill in the blank: SIADH causes water retention and dilutional hyponatraemia, managed primarily with fluid _____.

2.4.3 a structured approach to the child with a fluid disorder

A structured approach to any child with a suspected fluid disorder should combine a careful **history**, including fluid intake and output, any relevant underlying illness, and medication use, with **examination findings**, particularly weight trend, presence of oedema, and standard hydration assessment findings, and, where available, **relevant laboratory investigations**, including serum sodium, urea, and osmolality, to reach an accurate diagnosis rather than assuming every unwell child with abnormal fluid status is simply dehydrated.

This integrative approach, bringing together the physiology, examination, and investigation principles covered across this Series, prepares you to manage the wide range of fluid and electrolyte disturbances you will encounter in general paediatric practice, and forms an essential foundation for the remaining Series of this course, which turn to the evaluation and disorders of the gastrointestinal and genitourinary systems specifically.

Fill in the blank: A structured approach to a fluid disorder should combine history, examination, and relevant _____ investigations.

Comparing Fluid Disorders

Key Parameters	Dehydration (Hypovolemia)	Fluid Overload (Hypervolemia)	SIADH (Syndrome of Inappropriate Antidiuretic Hormone)	Diabetes Insipidus (DI)
Definition	Deficit of water and electrolytes	Excess of total body water and sodium	Excess water retention due to high ADH	Large volume of dilute urine due to low ADH (Central) or kidney resistance (Nephrogenic)
Primary Problem	Fluid loss (vomiting, diarrhea, hemorrhage)	Retention of fluid Heart failure Kidney failure Liver failure	Impaired water excretion	Water loss
Serum Sodium (Na+)	Increased Na+ (>145 mEq/L)	Normal or Decreased Na+ (<135 mEq/L)	Decreased Na+ (<135 mEq/L)	Increased Na+ (>145 mEq/L)
Serum Osmolality	Increased Osmolality (>295 mOsm/kg)	Normal or Decreased Osmolality (<285 mOsm/kg)	Decreased Osmolality (<285 mOsm/kg)	Increased Osmolality (>295 mOsm/kg)
Urine Osmolality	Increased Osmolality	Variable	Increased Osmolality (>100 mOsm/kg)	Decreased Osmolality (<300 mOsm/kg)
Urine Output	Decreased	Increased	Concentrated urine	Massively increased (Dilute urine)
Key Clinical Signs	Tachycardia Dry mucous membranes Hypotension Poor skin turgor	Edema (peripheral, pulmonary) Weight gain Hypertension	Headache, Confusion, Seizures (due to hyponatremia) Weight gain	Polyuria, Polydipsia Dehydration signs if fluid intake is insufficient
Common Causes	Gastroenteritis Excessive sweating Diuretics	Congestive Heart Failure Renal Failure Iatrogenic fluid excess	Small cell lung cancer CNS disorders (trauma, stroke) Medications (e.g., SSRIs)	Central (Head trauma, Pituitary tumor) Nephrogenic (Lithium, Kidney disease

Figure 2.4: A comparison of the clinical features of dehydration, fluid overload, SIADH, and diabetes insipidus.

Pilot questions 2.4

1. List two clinical features that distinguish fluid overload from dehydration. (Linked to SLO 2.8)
2. Describe the pathophysiology of SIADH and its primary management. (Linked to SLO 2.8)
3. Distinguish central from nephrogenic diabetes insipidus. (Linked to SLO 2.8)
4. Explain why diabetes insipidus can lead to hypernatraemic dehydration. (Linked to SLO 2.8)
5. Describe the components of a structured approach to a child with a suspected fluid disorder. (Linked to SLO 2.8)

Series Summary

This Series examined the **types of fluids** used in paediatric practice and the **principles of fluid therapy**, including maintenance, deficit, and ongoing loss replacement, before turning to the common **sodium and potassium disorders** encountered in unwell children.

We then examined the physiology of **acid-base balance** and its disorders, before concluding by **differentiating dehydration** from other important fluid disorders, including fluid overload, SIADH, and diabetes insipidus. Having worked through this Series, you should now be able to prescribe appropriate fluid therapy and recognise and manage common electrolyte and acid-base disturbances. The next Series turns to **hepatobiliary and gastrointestinal disorders**.

Glossary of Terms

1. **Crystalloid:** an intravenous fluid solution of small molecules that distributes freely across the vascular membrane.
2. **Holliday-Segar formula:** a weight-based formula used to calculate maintenance fluid requirements in children.
3. **Hyponatraemia:** a serum sodium concentration below 135 millimoles per litre.
4. **Osmotic demyelination syndrome:** a severe neurological injury caused by overly rapid correction of chronic hyponatraemia.
5. **Hyperkalaemia:** a serum potassium concentration above 5.5 millimoles per litre, potentially causing dangerous cardiac arrhythmia.
6. **Anion gap:** a calculated value used to help distinguish causes of metabolic acidosis.

7. **Metabolic acidosis:** acidosis arising from a primary fall in bicarbonate concentration.
8. **Respiratory acidosis:** acidosis arising from a primary rise in carbon dioxide due to inadequate ventilation.
9. **Fluid overload:** an excess accumulation of fluid in the body, presenting with oedema and, in severe cases, pulmonary oedema.
10. **SIADH:** syndrome of inappropriate antidiuretic hormone secretion, causing water retention and dilutional hyponatraemia.
11. **Diabetes insipidus:** a condition of inadequate antidiuretic hormone secretion or response, causing excessive dilute urine output.
12. **Compensation:** the physiological response of the non-primarily-affected system to minimise the change in blood pH.

Concept Check Questions [Series 2]

Objective questions

- Isotonic crystalloids such as normal saline are generally preferred for fluid _____.
- Overly rapid correction of chronic hyponatraemia risks osmotic _____ syndrome.
- Calcium gluconate is given in severe hyperkalaemia to stabilise the _____ membrane.
- A raised anion gap in metabolic acidosis suggests excess _____ production.
- SIADH causes water retention and dilutional hyponatraemia, managed primarily with fluid _____.

Short-answer questions

1. State the Holliday-Segar formula for calculating maintenance fluid requirements.
2. List the three components that together make up total paediatric fluid therapy.
3. Distinguish metabolic from respiratory acidosis.
4. List two ECG changes seen in hyperkalaemia.
5. Distinguish central from nephrogenic diabetes insipidus.

Long-answer questions

6. Discuss the principles of paediatric fluid therapy, including maintenance, deficit, and ongoing losses. (Linked to SLO 2.1, SLO 2.2)
7. Discuss the causes, clinical features, and management of hyponatraemia and hypernatraemia. (Linked to SLO 2.3)

8. Discuss the causes, clinical features, and management of hypokalaemia and hyperkalaemia. (Linked to SLO 2.4)
9. Discuss the physiology of acid-base balance and the classification of its disorders. (Linked to SLO 2.5, SLO 2.6)
10. Discuss how dehydration is differentiated from other disorders of fluid balance in children. (Linked to SLO 2.8)

Answers to Concept Check Questions [Series 2]

Objective questions

11. Resuscitation.
12. Demyelination.
13. Cardiac.
14. Acid.
15. Restriction.

Short-answer questions

16. 100 millilitres per kilogram for the first 10 kilograms, 50 millilitres per kilogram for the next 10 kilograms, and 20 millilitres per kilogram for each kilogram above 20 kilograms.
17. Maintenance, deficit replacement, and ongoing losses.
18. Metabolic acidosis arises from a primary fall in bicarbonate, while respiratory acidosis arises from a primary rise in carbon dioxide due to inadequate ventilation.
19. Peaked T waves and a widened QRS complex.
20. Central diabetes insipidus results from inadequate antidiuretic hormone secretion, while nephrogenic diabetes insipidus results from inadequate renal response to antidiuretic hormone.

Long-answer questions

21. **Basic response:** Fluid therapy combines maintenance, calculated using the Holliday-Segar formula, deficit replacement based on assessed dehydration, and replacement of ongoing losses such as continuing diarrhoea.

Value-added response: Failing to account for ongoing losses can leave a child persistently dehydrated despite apparently adequate fluid administration, so all three components must be calculated and combined.

22. **Basic response:** Hyponatraemia results from excess water relative to sodium or excess sodium loss, while hypernatraemia results from water loss exceeding sodium loss or excess sodium intake.

Value-added response: Both must be corrected slowly, since rapid correction of hyponatraemia risks osmotic demyelination syndrome, while rapid correction of hypernatraemia risks cerebral oedema and seizures.

23. **Basic response:** Hypokalaemia commonly results from gastrointestinal losses or diuretics, causing weakness and arrhythmia, while hyperkalaemia is more immediately dangerous, causing life-threatening cardiac arrhythmia.

Value-added response: Severe hyperkalaemia is managed with calcium gluconate to stabilise the cardiac membrane, together with insulin and glucose or salbutamol to shift potassium intracellularly.

24. **Basic response:** Acid-base balance is maintained by buffer systems, respiratory compensation, and renal compensation, and disorders are classified as metabolic or respiratory, acidosis or alkalosis.

Value-added response: The anion gap helps distinguish causes of metabolic acidosis, and correction generally focuses on treating the underlying cause rather than direct bicarbonate administration.

25. **Basic response:** Dehydration is distinguished from fluid overload, SIADH, and diabetes insipidus by considering weight trend, presence of oedema, serum sodium, and urine output together.

Value-added response: A structured approach combining history, examination, and relevant investigations is essential, since assuming every unwell child with abnormal fluid status is simply dehydrated can lead to inappropriate management.

Answers to Pilot Questions [Series 2]

Part 2.1

1. Crystalloids are small molecules distributing freely across the vascular membrane; examples include normal saline and Ringer's lactate.

2. Isotonic crystalloids are preferred for resuscitation since they remain within the extracellular space and restore circulating volume effectively.
3. 100 mL/kg for the first 10 kg, 50 mL/kg for the next 10 kg, and 20 mL/kg for each kilogram above 20 kg.
4. Fever increases insensible losses and therefore increases fluid requirements.
5. Maintenance, deficit replacement, and ongoing losses.

Part 2.2

6. Hyponatraemia is a serum sodium below 135 mmol/L; causes include excess water intake or retention and excess sodium loss such as from diarrhoea.
7. Rapid correction risks osmotic demyelination syndrome, a severe and potentially permanent neurological injury.
8. Hypernatraemia is a serum sodium above 145 mmol/L, typically causing irritability and a high-pitched cry.
9. Flattened T waves and prominent U waves are seen in hypokalaemia.
10. Calcium gluconate to stabilise the cardiac membrane, with insulin and glucose or salbutamol to shift potassium intracellularly, alongside treating the cause.

Part 2.3

11. Buffer systems, respiratory compensation, and renal compensation together maintain acid-base balance.
12. Metabolic acidosis arises from a primary fall in bicarbonate, while respiratory acidosis arises from a primary rise in carbon dioxide.
13. A raised anion gap suggests excess acid production, while a normal anion gap suggests bicarbonate loss or a renal cause.
14. Loss of gastric acid, as in pyloric stenosis, and excessive diuretic use are causes of metabolic alkalosis.
15. Correction focuses primarily on treating the underlying cause rather than direct bicarbonate administration.

Part 2.4

1. Oedema, weight gain, and respiratory findings such as pulmonary oedema distinguish fluid overload from dehydration.
2. SIADH causes water retention and dilutional hyponatraemia, managed primarily with fluid restriction.
3. Central diabetes insipidus results from inadequate hormone secretion, while nephrogenic results from inadequate renal response to the hormone.

4. Excessive dilute urine output can exceed free water intake, leading to hypernatraemic dehydration.
5. A structured approach combines history of intake and output, examination for weight trend and oedema, and relevant laboratory investigations.

References and Attributions [Series 2]

1. Kliegman, R. M., St Geme, J. W., Blum, N. J., Shah, S. S., Tasker, R. C. and Wilson, K. M. (2020). Nelson Textbook of Pediatrics (21st ed.). Elsevier.
2. Holliday, M. A. and Segar, W. E. (1957). The maintenance need for water in parenteral fluid therapy. Pediatrics, 19(5), 823 to 832.
3. Somers, M. J. G. (2021). Maintenance fluid therapy in children. UpToDate Clinical Reference.
4. Greenbaum, L. A. (2020). Fluid and electrolyte disorders. In Nelson Textbook of Pediatrics (21st ed.). Elsevier.

Figures, Plates, Tables, and Boxes

1. Table 2.1: Common intravenous fluids used in paediatric practice. AI-generated using the prompt: "Create a clean reference table titled Common Intravenous Fluids in Paediatric Practice, listing fluid, composition summary, and typical use. Simple clinical reference table style with outside border."
2. Box 2.2: Emergency management steps for severe, symptomatic hyperkalaemia. AI-generated using the prompt: "Create a simple single-column reference box titled Emergency Management of Severe Hyperkalaemia, listing sequential steps. Clean clinical reference box style."
3. Figure 2.3: A simple approach to classifying acid-base disorders using pH, bicarbonate, and carbon dioxide. AI-generated using the prompt: "Create a professional medical flowchart titled Classifying Acid-Base Disorders, branching by pH, bicarbonate, and carbon dioxide changes. Clean clinical educational diagram style."
4. Figure 2.4: A comparison of the clinical features of dehydration, fluid overload, SIADH, and diabetes insipidus. AI-generated using the prompt: "Create a professional medical reference chart titled Comparing Fluid Disorders, contrasting dehydration, fluid overload, SIADH, and diabetes insipidus. Clean clinical educational diagram style."

Series 3: Hepatobiliary and Gastrointestinal Disorders

- **Part 3.1: Jaundice, Hepatitis, and Hepatic Encephalopathy**
    - Sub Part 3.1.1: Definition, causes, and evaluation of jaundice
    - Sub Part 3.1.2: Hepatitis: acute and chronic
    - Sub Part 3.1.3: Hepatic encephalopathy
- **Part 3.2: Intestinal Parasites**
    - Sub Part 3.2.1: Types and life cycle of common intestinal parasites
    - Sub Part 3.2.2: Clinical features and impact on child health
    - Sub Part 3.2.3: Treatment and prevention of intestinal parasitic infection
- **Part 3.3: Abdominal Pain and Malabsorption**
    - Sub Part 3.3.1: Evaluation of acute abdominal pain and acute abdomen
    - Sub Part 3.3.2: Chronic abdominal pain
    - Sub Part 3.3.3: Classification and evaluation of malabsorption
- **Part 3.4: Gastrointestinal Bleeding, Oedema, and Ascites**
    - Sub Part 3.4.1: Classification of gastrointestinal bleeding
    - Sub Part 3.4.2: Evaluation and management of gastrointestinal bleeding
    - Sub Part 3.4.3: Oedema and ascites: pathophysiology and evaluation

Introduction

In the last Series, we explored fluid therapy and the electrolyte and acid-base disorders that so often accompany gastrointestinal illness. We now turn to a broader range of gastrointestinal and hepatobiliary conditions. Picture a four year old brought to clinic with yellowing of the eyes and skin, and consider how differently you would approach this from a ten year old with three months of intermittent abdominal pain. This Series introduces the assessment and management of jaundice, hepatitis, intestinal parasites, abdominal pain, malabsorption, and gastrointestinal bleeding, conditions that together make up a substantial part of everyday paediatric practice.

The aim of this Series is to equip you with the knowledge required to evaluate and manage a child with jaundice, hepatitis, intestinal parasitic infection, abdominal pain, malabsorption, gastrointestinal bleeding, or oedema and ascites.

This Series begins with **jaundice, hepatitis, and hepatic encephalopathy**, before turning to **intestinal parasites**. Next, we examine **abdominal pain and malabsorption**, before concluding with **gastrointestinal bleeding, oedema, and ascites**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 1.1:** define jaundice and describe the causes and evaluation of a child presenting with jaundice,
- **SLO 1.2:** describe the aetiology and clinical features of acute and chronic hepatitis,
- **SLO 1.3:** describe the clinical manifestations, complications, and management of hepatic encephalopathy,
- **SLO 1.4:** describe the types, life cycle, transmission, and clinical features of common intestinal parasites,
- **SLO 1.5:** describe the evaluation and management of a child with acute or chronic abdominal pain,
- **SLO 1.6:** describe the classification, evaluation, and management of malabsorption,
- **SLO 1.7:** describe the classification and management of gastrointestinal bleeding, and
- **SLO 1.8:** describe the pathophysiology, evaluation, and management of oedema and ascites.

3.1 Jaundice, Hepatitis, and Hepatic Encephalopathy

3.1.1 definition, causes, and evaluation of jaundice

Age matters as much as the bilirubin level

Jaundice refers to the yellow discolouration of the skin and sclerae caused by an elevated **serum bilirubin**, and is classified according to the predominant type of bilirubin involved into **unconjugated hyperbilirubinaemia**, commonly seen in **physiological neonatal jaundice** or **haemolysis**, and **conjugated hyperbilirubinaemia**, which is always pathological and points towards **hepatocellular** or **obstructive biliary** disease requiring urgent further evaluation.

Evaluation of a jaundiced child should establish the **age of onset**, since causes differ considerably between the **neonatal period** and **older childhood**, together with associated symptoms such as **pale stools** and **dark urine**, which suggest a conjugated, obstructive pattern,

and relevant history including **family history** of liver disease, **medication use**, and any history of **blood transfusion** or contact with **hepatitis**.

Fill in the blank: Conjugated hyperbilirubinaemia is always _____ and points towards hepatocellular or obstructive disease.

3.1.2 hepatitis: acute and chronic

Hepatitis refers to inflammation of the liver, most commonly caused by **viral infection**, with **hepatitis A** being the most common cause of **acute hepatitis** in children in Nigeria, typically transmitted by the **faecal-oral route** and usually self-limiting, while **hepatitis B** and **hepatitis C** are more commonly associated with **chronic infection**, which can progress over years to **cirrhosis** and, rarely, **hepatocellular carcinoma** if not identified and managed appropriately.

Clinical features of acute hepatitis include **jaundice**, **malaise**, **anorexia**, **nausea**, and **right upper quadrant discomfort**, with markedly elevated **liver transaminases** on laboratory testing, while chronic hepatitis is frequently **asymptomatic** for long periods, meaning screening of at-risk children, such as those born to mothers with chronic hepatitis B infection, is an important strategy for early detection.

Fill in the blank: Hepatitis A is typically transmitted by the _____ route.

3.1.3 hepatic encephalopathy

Hepatic encephalopathy is a complication of significant liver dysfunction, resulting from the **accumulation of neurotoxic substances**, particularly **ammonia**, that are normally cleared by a functioning liver, and presents with a spectrum of **neurological and cognitive changes**, ranging from subtle **behavioural change** and **confusion** in early stages to **drowsiness**, **asterixis**, or flapping tremor, and, in advanced cases, **coma**.

Laboratory features supporting the diagnosis include an elevated **serum ammonia**, though the level does not always correlate closely with clinical severity, and management focuses on **identifying and correcting precipitating factors**, such as gastrointestinal bleeding, infection, or constipation, together with measures to reduce ammonia production and absorption, such as **lactulose**, alongside general supportive care and treatment of the underlying liver disease.

Fill in the blank: Hepatic encephalopathy results from accumulation of neurotoxic substances, particularly _____.

Table 3.1: Causes of jaundice by bilirubin type.

Bilirubin Type	Example Causes	Clinical Significance
Unconjugated (Indirect) Hyperbilirubinemia	Hemolysis (e.g., sickle cell disease, G6PD deficiency), ineffective erythropoiesis, physiologic jaundice of newborn, Gilbert syndrome	Results from increased bilirubin production or impaired conjugation; bilirubin is lipid-soluble and can cross the blood-brain barrier, risking kernicterus in neonates.
Conjugated (Direct) Hyperbilirubinemia	Biliary obstruction (e.g., choledocholithiasis, biliary atresia), hepatocellular dysfunction (e.g., hepatitis, cirrhosis), Dubin–Johnson syndrome, Rotor syndrome	Indicates impaired excretion of water-soluble bilirubin; associated with dark urine and pale stools due to reduced bile pigment in intestine.
Mixed Hyperbilirubinemia	Severe hepatitis, advanced cirrhosis, sepsis, drug-induced liver injury	Reflects combined hepatocellular injury and cholestasis; both conjugated and unconjugated bilirubin elevated, requiring comprehensive evaluation.

Pilot questions 3.1

- Define jaundice and distinguish unconjugated from conjugated hyperbilirubinaemia. (Linked to SLO 3.1)
- What do pale stools and dark urine suggest in a jaundiced child? (Linked to SLO 3.1)
- Which hepatitis virus is the most common cause of acute hepatitis in Nigerian children, and how is it transmitted? (Linked to SLO 3.2)
- Describe the clinical features of hepatic encephalopathy. (Linked to SLO 3.3)
- Describe the general principles of managing hepatic encephalopathy. (Linked to SLO 3.3)

3.2 Intestinal Parasites

3.2.1 types and life cycle of common intestinal parasites

Soil-transmitted worms share a similar life cycle

Common intestinal parasites affecting children include the **soil-transmitted helminths**, **Ascaris lumbricoides**, **Trichuris trichiura**, and **hookworm**, together with **Enterobius vermicularis**, or

pinworm, and protozoal infections such as **Giardia lamblia**. The soil-transmitted helminths share a broadly similar **life cycle**, in which eggs or larvae present in **contaminated soil** are ingested or, in the case of hookworm, penetrate the skin directly, before maturing within the gastrointestinal tract.

Modes of transmission generally relate to poor sanitation and hygiene, with **faecal-oral transmission** important for Ascaris, Trichuris, and Enterobius, and **direct skin penetration** by larvae in soil important for hookworm, meaning prevention strategies for this group of infections overlap substantially with those for diarrhoeal disease discussed earlier in this course.

Fill in the blank: Hookworm larvae typically infect a child by direct _____ penetration.

3.2.2 clinical features and impact on child health

Clinical features of intestinal parasitic infection vary by organism and **worm burden**, ranging from **asymptomatic** infection in light burdens to **abdominal pain**, **diarrhoea**, and, with heavy Ascaris infection, **intestinal obstruction** from a bolus of worms. **Hookworm** is a particularly important cause of **iron deficiency anaemia** in endemic areas due to chronic intestinal blood loss, while **Trichuris** infection can cause **rectal prolapse** in severe cases.

Beyond these direct effects, chronic intestinal parasitic infection has a well recognised impact on **child health and development**, contributing to **malnutrition**, **growth faltering**, and impaired **cognitive development** through chronic blood loss, competition for nutrients, and low-grade chronic inflammation, making control of intestinal parasites an important public health priority in endemic regions.

Fill in the blank: Hookworm is a particularly important cause of iron deficiency _____ in endemic areas.

3.2.3 treatment and prevention of intestinal parasitic infection

Treatment of most soil-transmitted helminth and pinworm infections involves a short course of an **anthelminthic** medication, such as **albendazole** or **mebendazole**, while **Giardia lamblia**, a protozoal rather than helminthic infection, requires treatment with **metronidazole** or **tinidazole**, since anthelminthics are not effective against protozoa.

Prevention strategies mirror those for diarrhoeal disease and include improved access to **safe water** and **sanitation**, promotion of **hand hygiene** and **footwear use** to reduce hookworm skin penetration, and, in many endemic settings, periodic **mass deworming programmes** targeting school-age children, which have been shown to meaningfully reduce the burden of intestinal

parasitic infection and its downstream effects on growth and cognitive development at a population level.

Fill in the blank: Giardia lamblia requires treatment with metronidazole or _____, since anthelminthics are ineffective against protozoa.

Box 3.2: Common intestinal parasites and their key distinguishing features.

Common intestinal parasites in children
Ascaris lumbricoides: large roundworm, faecal-oral transmission, risk of intestinal obstruction in heavy infection.
Trichuris trichiura: whipworm, faecal-oral transmission, risk of rectal prolapse in severe infection.
Hookworm: transmitted by skin penetration, important cause of iron deficiency anaemia.
Enterobius vermicularis: pinworm, faecal-oral transmission, causes perianal itching, especially at night.
Giardia lamblia: protozoal infection, causes diarrhoea and malabsorption, treated with metronidazole.

Pilot questions 3.2

1. List four common intestinal parasites affecting children. (Linked to SLO 3.4)
2. Describe the mode of transmission of hookworm. (Linked to SLO 3.4)
3. Explain why hookworm infection is an important cause of anaemia. (Linked to SLO 3.4)
4. Describe the impact of chronic intestinal parasitic infection on child growth and development. (Linked to SLO 3.4)
5. Describe the treatment of Ascaris infection compared with Giardia infection. (Linked to SLO 3.4)

3.3 Abdominal Pain and Malabsorption

3.3.1 evaluation of acute abdominal pain and acute abdomen

Recognising when abdominal pain is a surgical emergency

Acute abdominal pain in children has a broad differential diagnosis that varies by age, ranging from common, benign causes such as **constipation** and **gastroenteritis**, to potentially serious **surgical causes** requiring urgent intervention, collectively referred to as an **acute abdomen**, including **appendicitis**, **intussusception**, and **intestinal malrotation with volvulus**.

Evaluation should establish the **site**, **onset**, **character**, and **radiation** of pain, together with associated symptoms such as **vomiting**, **fever**, and **change in bowel habit**, and careful examination for **peritonism**, including **guarding**, **rebound tenderness**, and **rigidity**, which, if present, suggests a surgical cause requiring urgent surgical assessment rather than conservative management alone.

Fill in the blank: Signs of peritonism, including guarding and rebound tenderness, suggest a _____ cause of abdominal pain.

3.3.2 chronic abdominal pain

Chronic abdominal pain, generally defined as pain persisting or recurring over at least two months, is common in school-age children and is most often **functional**, meaning no specific structural or biochemical cause is identified despite appropriate evaluation, though this diagnosis should only be reached after actively considering and excluding **organic causes** such as **coeliac disease**, **inflammatory bowel disease**, and **peptic ulcer disease**.

Red flag features that should prompt more extensive investigation for an organic cause include **weight loss**, **growth faltering**, **nocturnal pain** that wakes the child from sleep, **blood in the stool**, and a **family history** of inflammatory bowel disease, and their presence should lower the threshold for referral and further investigation, including laboratory and, where indicated, endoscopic evaluation.

Fill in the blank: Nocturnal pain that wakes a child from sleep is a _____ feature suggesting an organic cause of chronic abdominal pain.

3.3.3 classification and evaluation of malabsorption

Malabsorption refers to impaired absorption of one or more nutrients from the gastrointestinal tract, and can be classified by the specific nutrient affected, including **carbohydrate malabsorption**, as in lactose intolerance, **fat malabsorption**, as in coeliac disease or exocrine pancreatic insufficiency in cystic fibrosis, and **protein malabsorption**, and clinical consequences include **chronic diarrhoea**, **failure to thrive**, **abdominal distension**, and specific **nutrient deficiencies** depending on the pattern involved.

Evaluation of suspected malabsorption includes **stool studies**, such as faecal fat estimation, **specific blood tests**, including **coeliac serology**, and, where indicated, **endoscopy with small bowel biopsy**, and treatment depends entirely on the specific underlying cause identified, ranging from **dietary exclusion**, as in coeliac disease or lactose intolerance, to **pancreatic enzyme replacement** in cystic fibrosis.

Fill in the blank: Coeliac disease is an example of _____ malabsorption.

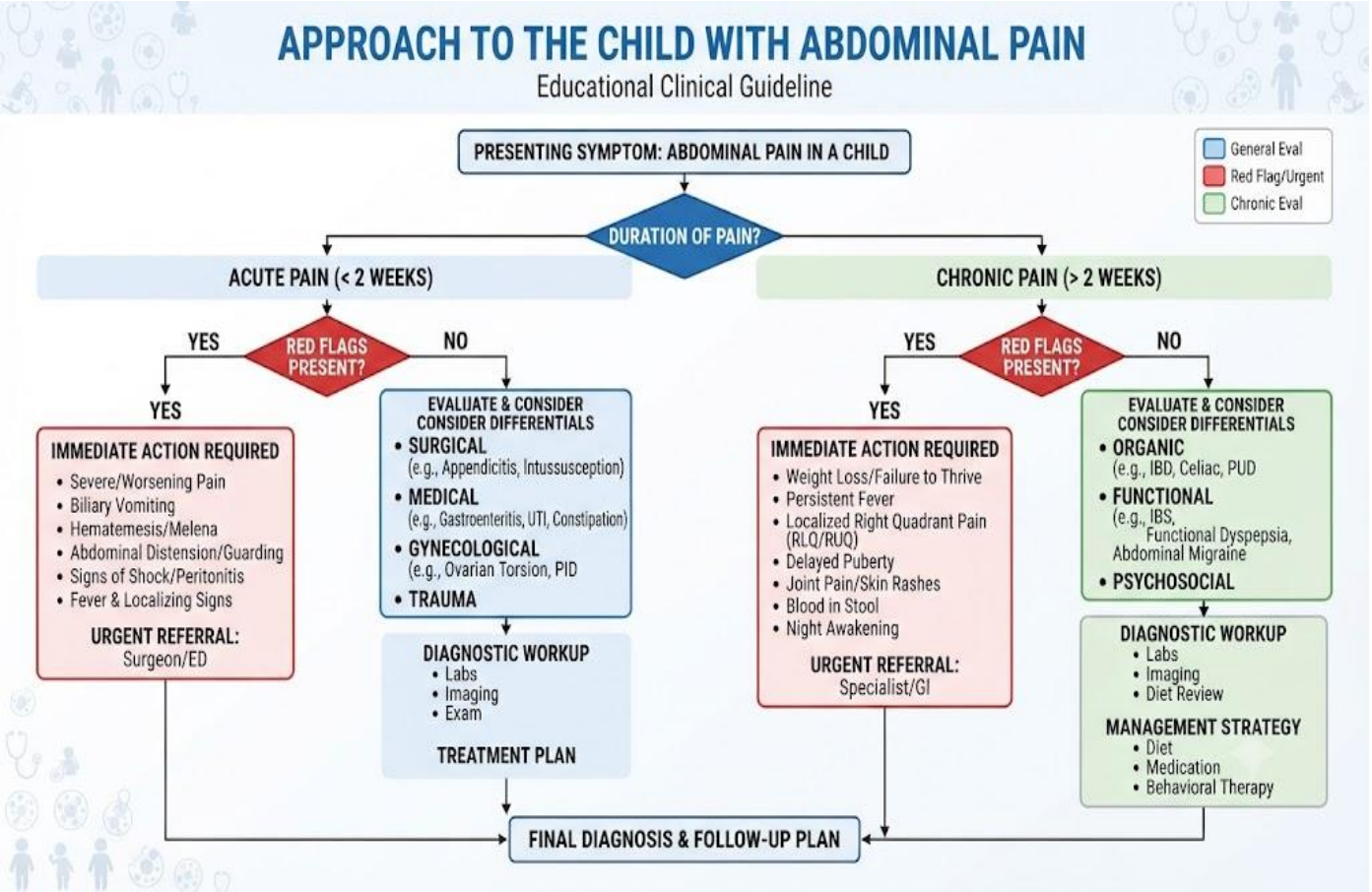

Figure 3.3: A structured approach to evaluating a child with abdominal pain.

Pilot questions 3.3

1. List two benign and two surgical causes of acute abdominal pain in children. (Linked to SLO 3.5)
2. Describe the significance of peritonism on examination of a child with abdominal pain. (Linked to SLO 3.5)
3. List three red flag features of chronic abdominal pain suggesting an organic cause. (Linked to SLO 3.5)
4. Classify malabsorption by the type of nutrient affected, giving an example of each. (Linked to SLO 3.6)
5. Describe the evaluation of a child with suspected malabsorption. (Linked to SLO 3.6)

3.4 Gastrointestinal Bleeding, Oedema, and Ascites

3.4.1 classification of gastrointestinal bleeding

The site of bleeding shapes both presentation and priority

Gastrointestinal bleeding is classified by the site of origin into **upper gastrointestinal bleeding**, arising proximal to the ligament of Treitz, presenting with **haematemesis**, vomiting of blood, or **melaena**, black, tarry stool from digested blood, and **lower gastrointestinal bleeding**, arising distal to this point, presenting with **haematochezia**, the passage of fresh red blood per rectum.

Common causes vary by age and site, with **oesophagitis**, **peptic ulcer disease**, and, in children with underlying liver disease, **oesophageal varices** important causes of upper gastrointestinal bleeding, and **anal fissure**, **infectious colitis**, **intussusception**, and, in infants, **cow's milk protein allergy** among the important causes of lower gastrointestinal bleeding, though a careful history and examination is essential in every case, since the apparent site is not always accurate.

Fill in the blank: Black, tarry stool from digested blood is called _____.

3.4.2 evaluation and management of gastrointestinal bleeding

Evaluation of a child with gastrointestinal bleeding begins with an assessment of **haemodynamic stability**, since significant bleeding can cause **hypovolaemic shock** requiring immediate **fluid resuscitation**, followed by a focused history establishing the **nature and quantity** of blood loss, associated symptoms, and relevant background such as known **liver disease** or **bleeding disorders**, together with examination for signs of **chronic liver disease**, such as **splenomegaly**, which would suggest varices as a potential source.

Management combines general resuscitative measures with **specific treatment** directed at the identified cause, which may include **acid suppression** for peptic disease, **endoscopic intervention** for active bleeding or varices, and correction of any underlying **coagulopathy**, with the overall priority always being to first stabilise the child before proceeding to definitive diagnostic and therapeutic measures.

Fill in the blank: Significant gastrointestinal bleeding can cause hypovolaemic _____ requiring immediate fluid resuscitation.

3.4.3 oedema and ascites: pathophysiology and evaluation

Oedema results from an imbalance of the **Starling forces** governing fluid movement between the intravascular and interstitial spaces, commonly due to **reduced plasma oncotic pressure**, as

in significant **hypoalbuminaemia** from nephrotic syndrome or liver disease, **increased capillary hydrostatic pressure**, as in heart failure, or **increased capillary permeability**, as in severe inflammation or sepsis. **Ascites**, the accumulation of fluid within the peritoneal cavity, shares overlapping mechanisms and commonly results from significant **liver disease** with portal hypertension, or, less commonly, from severe **hypoalbuminaemia** or certain renal or cardiac conditions.

Evaluation of a child with oedema or ascites should include careful assessment of **liver, renal, and cardiac function**, together with **serum albumin**, and, where ascites is present and its cause is unclear, **diagnostic paracentesis** may be performed to analyse the ascitic fluid, helping distinguish causes such as portal hypertension from infection or malignancy through analysis of protein content, cell count, and, where indicated, culture.

Fill in the blank: Oedema results from an imbalance of the _____ forces governing fluid movement between compartments.

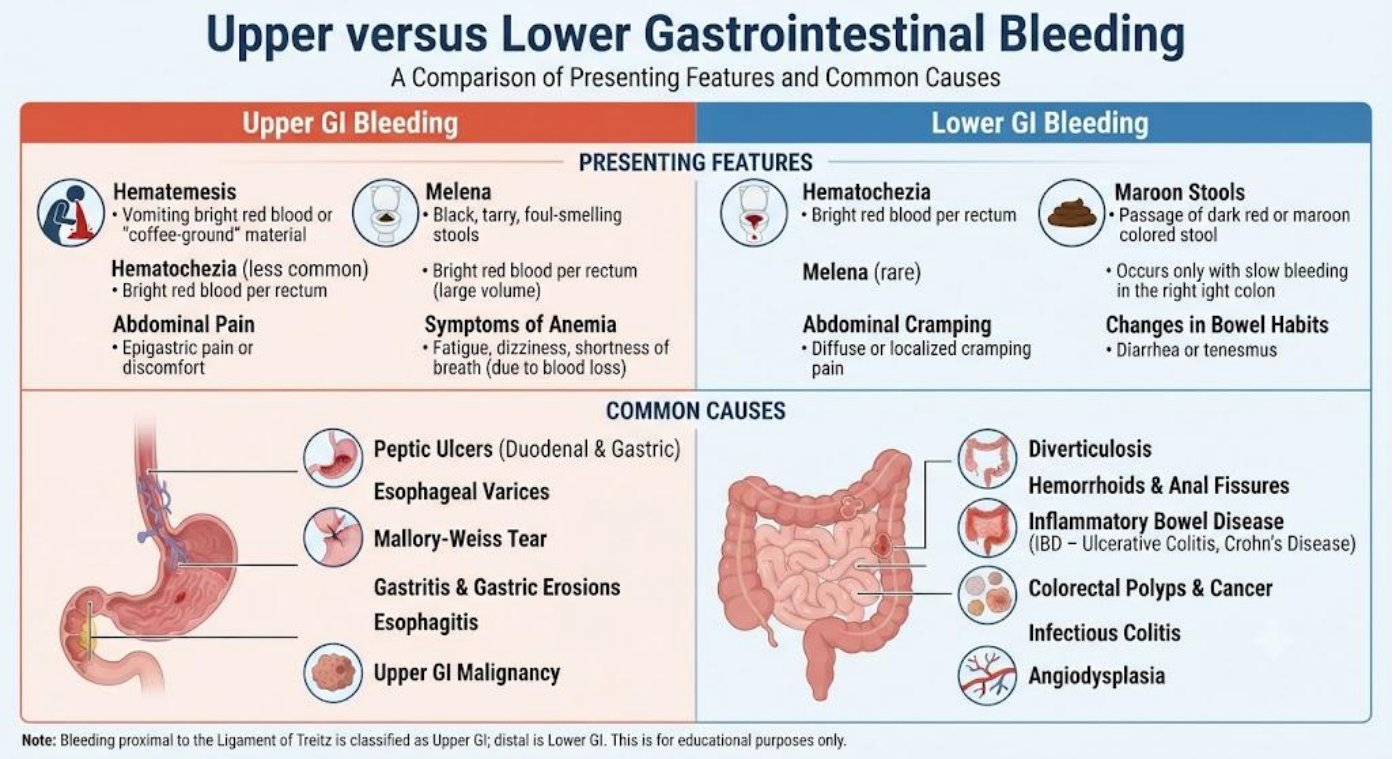

Figure 3.4: Distinguishing upper from lower gastrointestinal bleeding by presenting feature.

Pilot questions 3.4

1. Distinguish upper from lower gastrointestinal bleeding and their typical presenting features. (Linked to SLO 3.7)
2. List two causes each of upper and lower gastrointestinal bleeding. (Linked to SLO 3.7)
3. Describe the initial priority in evaluating a child with significant gastrointestinal bleeding. (Linked to SLO 3.7)
4. List three mechanisms that can cause oedema. (Linked to SLO 3.8)
5. Describe the evaluation of a child presenting with ascites. (Linked to SLO 3.8)

Series Summary

This Series examined a broad range of **hepatobiliary and gastrointestinal disorders**, beginning with **jaundice, hepatitis, and hepatic encephalopathy**, before turning to **intestinal parasites** and their impact on child health.

We then examined the evaluation of **abdominal pain and malabsorption**, before concluding with **gastrointestinal bleeding, oedema, and ascites**. Having worked through this Series, you should now be able to evaluate and manage the common hepatobiliary and gastrointestinal conditions encountered in paediatric practice. The next Series turns to the **evaluation of the genitourinary system and glomerular disorders**.

Glossary of Terms

1. **Jaundice:** yellow discolouration of the skin and sclerae caused by an elevated serum bilirubin.
2. **Conjugated hyperbilirubinaemia:** an elevation of conjugated bilirubin, always pathological, suggesting hepatocellular or obstructive disease.
3. **Hepatitis:** inflammation of the liver, most commonly caused by viral infection.
4. **Hepatic encephalopathy:** a neurological complication of significant liver dysfunction caused by accumulation of neurotoxic substances, particularly ammonia.
5. **Soil-transmitted helminths:** intestinal worms, including Ascaris, Trichuris, and hookworm, transmitted via contaminated soil.
6. **Acute abdomen:** a surgical emergency presenting with severe abdominal pain and peritonism, requiring urgent surgical assessment.
7. **Malabsorption:** impaired absorption of one or more nutrients from the gastrointestinal tract.
8. **Coeliac disease:** an autoimmune condition causing malabsorption triggered by dietary gluten.
9. **Haematemesis:** vomiting of blood, a feature of upper gastrointestinal bleeding.
10. **Melaena:** black, tarry stool resulting from digested blood, typically from upper gastrointestinal bleeding.
11. **Haematochezia:** the passage of fresh red blood per rectum, typically from lower gastrointestinal bleeding.
12. **Ascites:** the accumulation of fluid within the peritoneal cavity.

Concept Check Questions [Series 3]

Objective questions

- Conjugated hyperbilirubinaemia is always _____ and points towards hepatocellular or obstructive disease.
- Hepatic encephalopathy results from accumulation of neurotoxic substances, particularly _____.
- Hookworm is a particularly important cause of iron deficiency _____ in endemic areas.
- Signs of peritonism, including guarding and rebound tenderness, suggest a _____ cause of abdominal pain.
- Black, tarry stool from digested blood is called _____.

Short-answer questions

1. Distinguish unconjugated from conjugated hyperbilirubinaemia.
2. Describe the clinical features of hepatic encephalopathy.
3. List two modes of transmission of common intestinal parasites.
4. List three red flag features of chronic abdominal pain suggesting an organic cause.
5. Distinguish haematemesis, melaena, and haematochezia.

Long-answer questions

6. Discuss the causes and evaluation of a child presenting with jaundice. (Linked to SLO 3.1)
7. Discuss the clinical features and management of hepatic encephalopathy. (Linked to SLO 3.3)
8. Discuss the clinical impact of chronic intestinal parasitic infection on child health. (Linked to SLO 3.4)
9. Discuss the evaluation of a child with acute abdominal pain, including recognition of an acute abdomen. (Linked to SLO 3.5)
10. Discuss the classification and evaluation of gastrointestinal bleeding. (Linked to SLO 3.7)

Answers to Concept Check Questions [Series 3]

Objective questions

11. Pathological.
12. Ammonia.
13. Anaemia.
14. Surgical.
15. Melaena.

Short-answer questions

16. Unconjugated hyperbilirubinaemia is often benign, as in physiological jaundice, while conjugated hyperbilirubinaemia is always pathological.
17. Behavioural change and confusion in early stages, progressing to drowsiness, asterixis, and, in advanced cases, coma.
18. Faecal-oral transmission and direct skin penetration by larvae in soil.
19. Weight loss, growth faltering, and nocturnal pain that wakes the child from sleep.
20. Haematemesis is vomiting of blood, melaena is black tarry stool from digested blood, and haematochezia is fresh red blood per rectum.

Long-answer questions

21. **Basic response:** Evaluation of jaundice establishes age of onset, associated symptoms such as pale stools and dark urine, and relevant history, distinguishing unconjugated from conjugated hyperbilirubinaemia.

Value-added response: Conjugated hyperbilirubinaemia is always pathological and requires urgent evaluation for hepatocellular or obstructive biliary disease, unlike many causes of unconjugated hyperbilirubinaemia.

22. **Basic response:** Hepatic encephalopathy presents with a spectrum from behavioural change to coma, caused by accumulation of neurotoxic substances, particularly ammonia, from significant liver dysfunction.

Value-added response: Management focuses on identifying and correcting precipitating factors such as bleeding or infection, together with lactulose to reduce ammonia production and absorption.

23. **Basic response:** Chronic intestinal parasitic infection contributes to malnutrition, growth faltering, and impaired cognitive development through chronic blood loss, nutrient competition, and inflammation.

Value-added response: Hookworm in particular is an important cause of iron deficiency anaemia, making mass deworming programmes an important public health intervention in endemic settings.

24. **Basic response:** Evaluation of acute abdominal pain establishes site, onset, character, and associated symptoms, with examination for peritonism to identify an acute abdomen.

Value-added response: Signs of peritonism such as guarding and rebound tenderness suggest a surgical cause such as appendicitis or intussusception, requiring urgent surgical assessment rather than conservative management.

25. **Basic response:** Gastrointestinal bleeding is classified as upper, presenting with haematemesis or melaena, or lower, presenting with haematochezia, with evaluation prioritising haemodynamic stability.

Value-added response: Management combines resuscitation with specific treatment such as acid suppression or endoscopic intervention, always stabilising the child before definitive diagnostic and therapeutic measures.

Answers to Pilot Questions [Series 3]

Part 3.1

1. Jaundice is yellow discolouration from elevated bilirubin; unconjugated is often benign while conjugated is always pathological.
2. Pale stools and dark urine suggest a conjugated, obstructive pattern of jaundice.
3. Hepatitis A is the most common cause, transmitted by the faecal-oral route.
4. Behavioural change and confusion progressing to drowsiness, asterixis, and coma in advanced cases.
5. Management focuses on identifying and correcting precipitating factors and using lactulose to reduce ammonia.

Part 3.2

6. Ascaris, Trichuris, hookworm, and Enterobius vermicularis are common intestinal parasites.

7. Hookworm larvae penetrate the skin directly, typically from contaminated soil.
8. Hookworm causes chronic intestinal blood loss, leading to iron deficiency anaemia.
9. It contributes to malnutrition, growth faltering, and impaired cognitive development.
10. Ascaris is treated with an anthelminthic such as albendazole, while Giardia requires metronidazole since it is protozoal.

Part 3.3

11. Benign causes include constipation and gastroenteritis; surgical causes include appendicitis and intussusception.
12. Peritonism suggests a surgical cause requiring urgent surgical assessment.
13. Weight loss, growth faltering, nocturnal pain, blood in stool, and family history of inflammatory bowel disease.
14. Carbohydrate malabsorption as in lactose intolerance, fat malabsorption as in coeliac disease, and protein malabsorption.
15. Stool studies, specific blood tests including coeliac serology, and, where indicated, endoscopy with biopsy.

Part 3.4

1. Upper GI bleeding presents with haematemesis or melaena; lower GI bleeding presents with haematochezia.
2. Upper causes include peptic ulcer disease and varices; lower causes include anal fissure and intussusception.
3. The initial priority is assessing haemodynamic stability and providing fluid resuscitation if needed.
4. Reduced plasma oncotic pressure, increased capillary hydrostatic pressure, and increased capillary permeability can cause oedema.
5. Evaluation includes assessment of liver, renal, and cardiac function, serum albumin, and, where indicated, diagnostic paracentesis.

References and Attributions [Series 3]

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2. World Health Organization (2017). Guideline: Preventive Chemotherapy to Control Soil-Transmitted Helminth Infections in At-Risk Population Groups. WHO Press.
3. Squires, R. H. et al. (2022). Evaluation of the child with hepatomegaly and liver disease. In Nelson Textbook of Pediatrics (21st ed.). Elsevier.
4. Rasquin, A. et al. (2016). Childhood functional gastrointestinal disorders. Gastroenterology, 130(5), 1527 to 1537.

Figures, Plates, Tables, and Boxes

1. Table 3.1: Causes of jaundice by bilirubin type. AI-generated using the prompt: "Create a clean reference table titled Causes of Jaundice by Bilirubin Type, listing bilirubin type, example causes, and clinical significance. Simple clinical reference table style with outside border."
2. Box 3.2: Common intestinal parasites and their key distinguishing features. AI-generated using the prompt: "Create a simple single-column reference box titled Common Intestinal Parasites in Children. Clean clinical reference box style."
3. Figure 3.3: A structured approach to evaluating a child with abdominal pain. AI-generated using the prompt: "Create a professional medical flowchart titled Approach to the Child with Abdominal Pain, branching by acute versus chronic pain and red flag features. Clean clinical educational diagram style."
4. Figure 3.4: Distinguishing upper from lower gastrointestinal bleeding by presenting feature. AI-generated using the prompt: "Create a professional medical reference chart titled Upper versus Lower Gastrointestinal Bleeding, comparing presenting features and common causes. Clean clinical educational diagram style."

Series 4: Evaluation of the Genitourinary System and Glomerular Disorders

- **Part 4.1: Anatomy, Physiology, and Evaluation of the Genitourinary System**
    - Sub Part 4.1.1: Brief anatomy and physiology of the urogenital system
    - Sub Part 4.1.2: History taking in genitourinary disorders
    - Sub Part 4.1.3: Physical examination of the genitourinary system
- **Part 4.2: Investigation of the Urinary System and Glomerular Filtration Rate**
    - Sub Part 4.2.1: Examination of urine
    - Sub Part 4.2.2: Investigations for urinary system disorders
    - Sub Part 4.2.3: Evaluation of the glomerular filtration rate
- **Part 4.3: Nephrotic Syndrome**
    - Sub Part 4.3.1: Pathogenesis and aetiology of nephrotic syndrome
    - Sub Part 4.3.2: Clinical features of nephrotic syndrome
    - Sub Part 4.3.3: Work up, management, and complications of nephrotic syndrome
- **Part 4.4: Acute Glomerulonephritis**
    - Sub Part 4.4.1: Aetiology and pathophysiology of acute glomerulonephritis
    - Sub Part 4.4.2: Clinical features of acute glomerulonephritis
    - Sub Part 4.4.3: Work up, management, and outcome of acute glomerulonephritis

Introduction

In the last Series, we explored hepatobiliary and gastrointestinal disorders. We now turn to the genitourinary system, beginning with its evaluation. Picture a five year old brought to clinic with puffy eyes that his mother first noticed on waking, gradually worsening over several days to involve his legs and abdomen. Recognising this presentation, and understanding the underlying glomerular disorders that commonly cause it, is the focus of this Series.

The aim of this Series is to equip you with the knowledge required to evaluate a child with a suspected genitourinary disorder, interpret relevant investigations including the glomerular filtration rate, and recognise and understand the management of nephrotic syndrome and acute glomerulonephritis.

This Series begins with the **anatomy, physiology, and evaluation** of the genitourinary system, before turning to **investigation of the urinary system and glomerular filtration rate**. Next, we examine **nephrotic syndrome**, before concluding with **acute glomerulonephritis**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 1.1:** describe the brief anatomy and physiology of the urogenital system,
- **SLO 1.2:** describe the history and examination relevant to disorders of the urogenital system,
- **SLO 1.3:** describe the examination of urine and the investigations required in evaluating urinary system disorders,
- **SLO 1.4:** describe the evaluation of the glomerular filtration rate,
- **SLO 1.5:** describe the pathogenesis, aetiology, and clinical features of nephrotic syndrome,
- **SLO 1.6:** describe the work up, management, complications, and outcome of nephrotic syndrome,
- **SLO 1.7:** describe the aetiology, pathophysiology, and clinical features of acute glomerulonephritis, and
- **SLO 1.8:** describe the work up, management, and outcome of acute glomerulonephritis.

4.1 Anatomy, Physiology, and Evaluation of the Genitourinary System

4.1.1 brief anatomy and physiology of the urogenital system

The nephron as the functional unit of the kidney

The urogenital system comprises the **kidneys**, **ureters**, **bladder**, and **urethra**, together with the reproductive structures, and the functional unit of the kidney is the **nephron**, comprising the **glomerulus**, where blood is filtered, and a series of **tubules** that reabsorb and secrete substances to produce the final urine. Each kidney contains approximately one million nephrons, and the kidneys together perform essential functions including **fluid and electrolyte regulation**, **acid-base balance**, **waste product excretion**, and **hormone production**, including erythropoietin and the active form of vitamin D.

In children, **renal function matures progressively** after birth, with glomerular filtration rate relatively low at birth and increasing over the first two years of life to reach adult-comparable

values when corrected for body surface area, an important consideration when interpreting renal function tests or dosing renally excreted medications in young infants.

Fill in the blank: The functional unit of the kidney is the _____.

4.1.2 history taking in genitourinary disorders

A focused genitourinary history should establish **urinary symptoms**, including **frequency**, **dysuria**, or pain on passing urine, **urgency**, **enuresis**, or involuntary urination, and any change in the **colour** or **appearance** of urine, such as visible blood or unusual odour, together with **systemic symptoms** such as fever, oedema, or reduced urine output that may accompany a broader renal disorder.

Relevant background history includes any **antenatal history** of abnormalities detected on antenatal ultrasound, **family history** of kidney disease or hearing loss, which may suggest an inherited condition, and, in older children, a **sexual history** where clinically relevant and age-appropriate, obtained with the same care and confidentiality principles discussed earlier in this course.

Fill in the blank: Involuntary urination is termed _____.

4.1.3 physical examination of the genitourinary system

Examination of the genitourinary system should include assessment of **blood pressure**, since hypertension is an important finding in many renal conditions, general inspection for **oedema**, particularly **periorbital** and **dependent** oedema, and abdominal examination for **renal masses**, **bladder distension**, or **tenderness**, together with careful, age-appropriate examination of the **external genitalia** where clinically indicated.

Growth parameters should also be assessed, since chronic renal disease can significantly impair growth, and examination for **dysmorphic features** or other **congenital anomalies** may provide clues to an underlying syndromic cause in a child with a structural urinary tract abnormality, since certain genetic syndromes are associated with specific patterns of renal malformation.

Fill in the blank: Hypertension is an important finding to actively assess for in many _____ conditions.

Table 4.1: Key components of the genitourinary history and examination.

Component	Key features to assess
Urinary symptoms	Frequency, dysuria, urgency, enuresis, change in colour or odour
Systemic symptoms	Fever, oedema, reduced urine output
Background history	Antenatal findings, family history, growth trajectory
Examination	Blood pressure, oedema, abdominal masses, external genitalia

Pilot questions 4.1

- Describe the functional unit of the kidney and its two main components. (Linked to SLO 4.1)
- List two essential functions performed by the kidneys. (Linked to SLO 4.1)
- List the key urinary symptoms to establish in a genitourinary history. (Linked to SLO 4.2)
- Why is family history relevant when evaluating a child with a genitourinary disorder? (Linked to SLO 4.2)
- Describe the key components of physical examination of the genitourinary system. (Linked to SLO 4.2)

4.2 Investigation of the Urinary System and Glomerular Filtration Rate

4.2.1 examination of urine

A urine sample can answer many questions at once

Urinalysis provides rapid, valuable information and typically includes **dipstick testing** for **protein**, **blood**, **leucocytes**, **nitrites**, and **glucose**, together with **microscopy**, which examines for red and white blood cells, casts, crystals, and organisms, and, where infection is suspected, **urine culture** to identify a causative organism and guide antibiotic selection.

The **method of urine collection** matters considerably for accurate interpretation, with a **clean-catch midstream sample** generally preferred in toilet-trained children to reduce contamination, while collection in infants may require a **urine bag**, **catheter sample**, or, particularly for a reliable culture, **suprapubic aspiration**, since urine bag samples are prone to contamination and can give misleading results.

Fill in the blank: A _____-catch midstream sample is generally preferred in toilet-trained children to reduce contamination.

4.2.2 investigations for urinary system disorders

Beyond urinalysis, investigation of suspected urinary system disorders may include **serum urea and creatinine** to assess renal function, **serum electrolytes**, and **imaging studies**, particularly **renal ultrasound**, which assesses kidney size, structure, and the presence of any obstruction or structural anomaly, and, in selected cases, more specialised imaging such as **micturating cystourethrogram** to assess for vesicoureteric reflux, or **nuclear medicine scans** to assess differential renal function or scarring.

The choice and sequence of investigations should be guided by the specific clinical question, avoiding unnecessary invasive or radiation-exposing investigations where a less invasive alternative would answer the same question, and always interpreting results in the context of the child's **age-appropriate reference ranges**, since normal values for serum creatinine, in particular, change substantially with age and muscle mass.

Fill in the blank: Serum urea and creatinine are used to assess _____ function.

4.2.3 evaluation of the glomerular filtration rate

The **glomerular filtration rate**, abbreviated GFR, is the best overall measure of kidney function, representing the volume of plasma filtered by the glomeruli per unit time, and while directly measured GFR using substances such as **inulin** or **iohexol clearance** is the gold standard, it is impractical for routine clinical use, so **estimated GFR** is calculated instead using serum creatinine, height, and, in some formulae, additional variables.

The **Schwartz formula** is the most widely used method for estimating GFR in children, calculated as a constant multiplied by the child's **height in centimetres** divided by the **serum creatinine**, and provides a practical, widely accessible estimate of renal function that is used to detect and monitor kidney disease, guide medication dosing, and determine the stage of chronic kidney disease when present.

Fill in the blank: The Schwartz formula estimates GFR using a constant multiplied by height divided by serum _____.

Box 4.2: The Schwartz formula and its practical application in estimating GFR.

The Schwartz formula for estimating GFR
Formula: estimated GFR = (k x height in cm) / serum creatinine.

The constant k varies with age and sex, reflecting differences in muscle mass.
Height must be in centimetres and creatinine in appropriate matched units.
Used to detect, monitor, and stage kidney disease, and to guide medication dosing.
Estimated GFR should always be interpreted alongside the full clinical picture.

Pilot questions 4.2

1. List the components typically assessed on urine dipstick testing. (Linked to SLO 4.3)
2. Why is a clean-catch midstream sample generally preferred over a urine bag sample? (Linked to SLO 4.3)
3. List two imaging investigations used in evaluating urinary system disorders. (Linked to SLO 4.3)
4. Define glomerular filtration rate and explain why it is considered the best overall measure of kidney function. (Linked to SLO 4.4)
5. State the Schwartz formula for estimating GFR in children. (Linked to SLO 4.4)

4.3 Nephrotic Syndrome

4.3.1 pathogenesis and aetiology of nephrotic syndrome

A leaky glomerular filtration barrier drives the whole syndrome

Nephrotic syndrome results from significantly increased **permeability** of the glomerular filtration barrier to protein, leading to the characteristic combination of **heavy proteinuria**, **hypoalbuminaemia**, and **oedema**, often accompanied by **hyperlipidaemia**. In children, the most common underlying cause is **minimal change disease**, so named because the glomeruli appear essentially normal on light microscopy despite the significant clinical abnormality, and which typically responds well to corticosteroid treatment.

Less common causes in children include **focal segmental glomerulosclerosis**, which is generally more resistant to steroid treatment and carries a higher risk of progression to chronic kidney disease, and **secondary nephrotic syndrome**, resulting from an underlying systemic condition such as lupus nephritis, though this pattern is considerably less common in children than in adults.

Fill in the blank: The most common underlying cause of nephrotic syndrome in children is _____ disease.

4.3.2 clinical features of nephrotic syndrome

The clinical hallmark of nephrotic syndrome is **oedema**, classically beginning as **periorbital puffiness**, most noticeable on waking, and progressing over days to become **generalised**, involving the lower limbs, abdomen, in the form of **ascites**, and, in severe cases, the genitalia. Parents often first notice the periorbital swelling and may initially attribute it to an allergic reaction, so recognising this classic pattern is important for prompt diagnosis.

Additional features include **frothy urine**, reflecting heavy proteinuria, and, in some children, signs of **hypovolaemia** despite the overall fluid overload, since much of the excess fluid has shifted into the interstitial space rather than remaining in circulation, a distinction that has important implications for safe fluid management in an affected child.

Fill in the blank: Oedema in nephrotic syndrome classically begins as _____ puffiness, most noticeable on waking.

4.3.3 work up, management, and complications of nephrotic syndrome

Work up of suspected nephrotic syndrome includes **urinalysis** confirming heavy proteinuria, **serum albumin**, **lipid profile**, and assessment of **renal function**, and a **renal biopsy** is generally reserved for children with atypical features, such as significant haematuria, hypertension, reduced renal function, or steroid resistance, rather than performed routinely in typical, steroid-responsive childhood nephrotic syndrome.

First-line management is **corticosteroid therapy**, typically prednisolone, to which most children with minimal change disease respond well, though **relapses** are common and may require repeated courses of treatment. Important **complications** to actively monitor for include increased susceptibility to **infection**, particularly **spontaneous bacterial peritonitis** and cellulitis, related to loss of immunoglobulins in the urine, and an increased risk of **thromboembolism**, related to loss of anticoagulant proteins and a relatively concentrated, hyperviscous blood state.

Fill in the blank: Nephrotic syndrome increases susceptibility to infection due to loss of _____ in the urine.

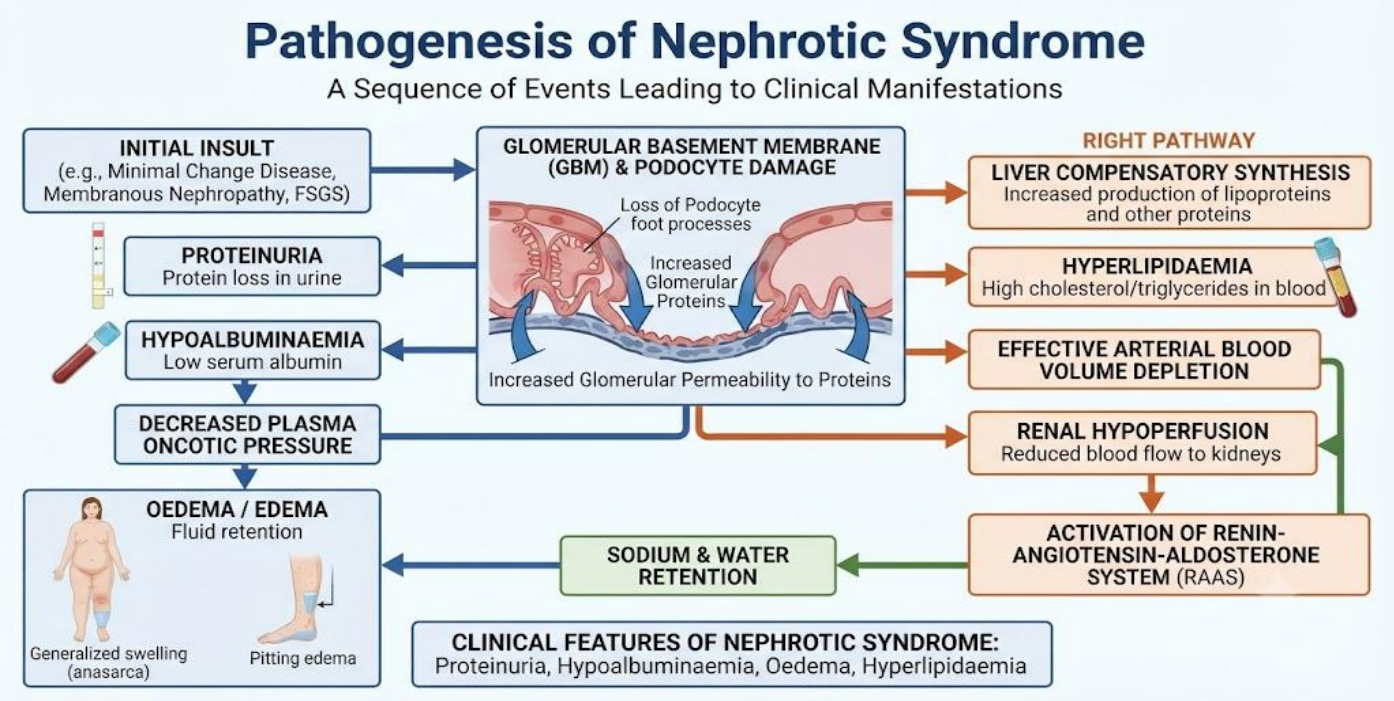


Figure 4.3: The pathogenesis of nephrotic syndrome from glomerular protein leak to clinical features.

Pilot questions 4.3

1. Define nephrotic syndrome by its characteristic clinical triad. (Linked to SLO 4.5)
2. Name the most common cause of nephrotic syndrome in children. (Linked to SLO 4.5)
3. Describe the classic pattern of oedema seen in nephrotic syndrome. (Linked to SLO 4.5)
4. Describe the first-line management of childhood nephrotic syndrome. (Linked to SLO 4.6)
5. List two important complications of nephrotic syndrome and their underlying mechanism. (Linked to SLO 4.6)

4.4 Acute Glomerulonephritis

4.4.1 aetiology and pathophysiology of acute glomerulonephritis

An immune-mediated attack on the glomerulus

Acute glomerulonephritis, abbreviated AGN, is an **inflammatory process** affecting the glomerulus, most commonly triggered by an **immune-mediated response**, and the prototype and

most common cause in children is **post-streptococcal glomerulonephritis**, occurring one to three weeks after infection with a **nephritogenic strain** of Group A beta-haemolytic streptococcus, typically following either a throat or skin infection.

The underlying mechanism involves deposition of **immune complexes** within the glomerulus, triggering an inflammatory response that damages the glomerular filtration barrier, leading to the characteristic clinical picture of **haematuria**, from disruption of the filtration barrier allowing red blood cells to pass into the urine, and **reduced glomerular filtration**, leading to fluid retention and, in some children, hypertension.

Fill in the blank: Post-streptococcal glomerulonephritis typically occurs one to _____ weeks after streptococcal infection.

4.4.2 clinical features of acute glomerulonephritis

The classic clinical presentation of AGN, often called the **nephritic syndrome**, includes **haematuria**, frequently described as **tea-coloured** or **cola-coloured urine**, **oedema**, typically less pronounced than in nephrotic syndrome and often most noticeable **periorbitally**, **hypertension**, which can occasionally be severe enough to cause **hypertensive encephalopathy**, and a **reduction in urine output**, or **oliguria**, reflecting the reduced glomerular filtration rate.

This combination of findings, haematuria, oedema, hypertension, and reduced urine output, distinguishes the typical **nephritic** presentation of AGN from the predominantly **oedema and heavy proteinuria** picture of nephrotic syndrome discussed in the previous Part, though some overlap in clinical features can occur and should not be used to exclude either diagnosis in isolation.

Fill in the blank: Haematuria in acute glomerulonephritis is frequently described as tea-coloured or _____-coloured.

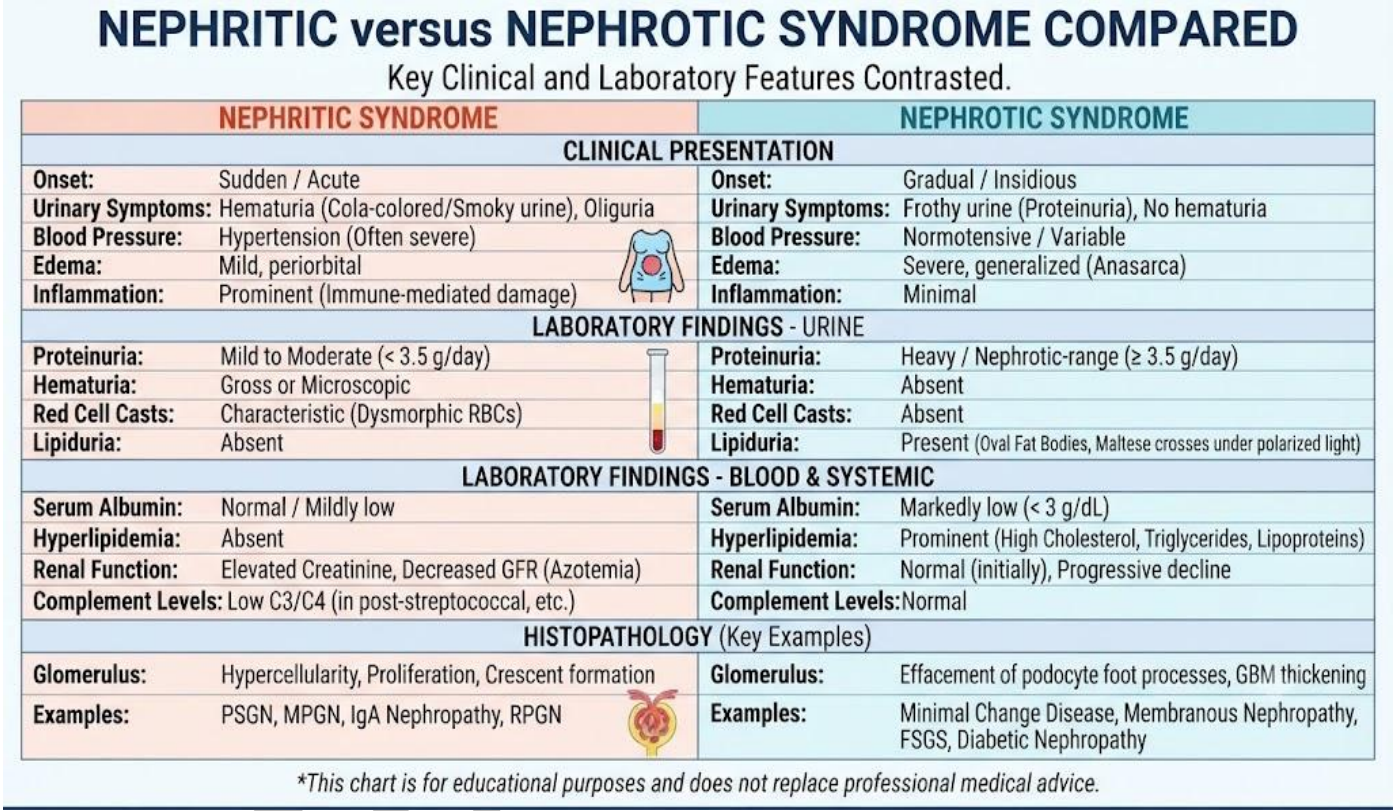
4.4.3 work up, management, and outcome of acute glomerulonephritis

Work up of suspected AGN includes **urinalysis**, confirming haematuria and typically mild to moderate proteinuria, **serum urea and creatinine** to assess renal function, **complement levels**, particularly **C3**, which is characteristically **low** in post-streptococcal glomerulonephritis and typically normalises within six to eight weeks, and evidence of **preceding streptococcal infection**, such as a raised **anti-streptolysin O titre**.

Management of typical post-streptococcal AGN is largely **supportive**, including careful monitoring and management of **fluid balance** and **blood pressure**, since the condition is generally **self-limiting** with an excellent prognosis in most children, with the large majority

making a full recovery, though rare cases with severe or rapidly progressive disease may require more intensive treatment and closer long-term follow up to monitor for the small risk of persistent renal impairment.

Fill in the blank: Serum complement C3 is characteristically _____ in post-streptococcal glomerulonephritis.



NEPHRITIC versus NEPHROTIC SYNDROME COMPARED

Key Clinical and Laboratory Features Contrasted.

	NEPHRITIC SYNDROME		NEPHROTIC SYNDROME
CLINICAL PRESENTATION			
Onset:	Sudden / Acute	**Onset:**	Gradual / Insidious
Urinary Symptoms:	Hematuria (Cola-colored/Smoky urine), Oliguria	**Urinary Symptoms:**	Frothy urine (Proteinuria), No hematuria
Blood Pressure:	Hypertension (Often severe)	**Blood Pressure:**	Normotensive / Variable
Edema:	Mild, periorbital	**Edema:**	Severe, generalized (Anasarca)
Inflammation:	Prominent (Immune-mediated damage)	**Inflammation:**	Minimal
LABORATORY FINDINGS - URINE			
Proteinuria:	Mild to Moderate (< 3.5 g/day)	**Proteinuria:**	Heavy / Nephrotic-range (≥ 3.5 g/day)
Hematuria:	Gross or Microscopic	**Hematuria:**	Absent
Red Cell Casts:	Characteristic (Dysmorphic RBCs)	**Red Cell Casts:**	Absent
Lipiduria:	Absent	**Lipiduria:**	Present (Oval Fat Bodies, Maltese crosses under polarized light)
LABORATORY FINDINGS - BLOOD & SYSTEMIC			
Serum Albumin:	Normal / Mildly low	**Serum Albumin:**	Markedly low (< 3 g/dL)
Hyperlipidemia:	Absent	**Hyperlipidemia:**	Prominent (High Cholesterol, Triglycerides, Lipoproteins)
Renal Function:	Elevated Creatinine, Decreased GFR (Azotemia)	**Renal Function:**	Normal (initially), Progressive decline
Complement Levels:	Low C3/C4 (in post-streptococcal, etc.)	**Complement Levels:**	Normal
HISTOPATHOLOGY (Key Examples)			
Glomerulus:	Hypercellularity, Proliferation, Crescent formation	**Glomerulus:**	Effacement of podocyte foot processes, GBM thickening
Examples:	PSGN, MPGN, IgA Nephropathy, RPGN	**Examples:**	Minimal Change Disease, Membranous Nephropathy, FSGS, Diabetic Nephropathy

**This chart is for educational purposes and does not replace professional medical advice.*

Figure 4.4: Comparing the nephritic and nephrotic clinical pictures.

Pilot questions 4.4

1. Describe the pathophysiology of post-streptococcal acute glomerulonephritis. (Linked to SLO 4.7)
2. List the features of the classic nephritic syndrome presentation. (Linked to SLO 4.7)
3. Describe the typical urine appearance in acute glomerulonephritis. (Linked to SLO 4.7)
4. What laboratory finding regarding complement levels is characteristic of post-streptococcal glomerulonephritis? (Linked to SLO 4.8)
5. Describe the general management approach and prognosis of typical post-streptococcal glomerulonephritis. (Linked to SLO 4.8)

Series Summary

This Series introduced the **evaluation of the genitourinary system**, beginning with relevant anatomy, physiology, history, and examination, before turning to the **investigation of the urinary system**, including urinalysis and the evaluation of glomerular filtration rate.

We then examined **nephrotic syndrome** and **acute glomerulonephritis**, the two most important glomerular disorders encountered in paediatric practice. Having worked through this Series, you should now be able to evaluate a child with a suspected genitourinary disorder and recognise these two important glomerular conditions. The next Series turns to **renal failure and urinary tract disorders**.

Glossary of Terms

1. **Nephron:** the functional unit of the kidney, comprising the glomerulus and its associated tubules.
2. **Glomerular filtration rate:** the volume of plasma filtered by the glomeruli per unit time, the best overall measure of kidney function.
3. **Schwartz formula:** a formula used to estimate glomerular filtration rate in children from height and serum creatinine.
4. **Nephrotic syndrome:** a condition characterised by heavy proteinuria, hypoalbuminaemia, and oedema, most commonly due to minimal change disease in children.
5. **Minimal change disease:** the most common cause of nephrotic syndrome in children, in which the glomeruli appear normal on light microscopy.
6. **Periorbital oedema:** swelling around the eyes, classically the first noticed sign of nephrotic syndrome.
7. **Acute glomerulonephritis:** an inflammatory process affecting the glomerulus, most commonly post-streptococcal in children.
8. **Nephritic syndrome:** the clinical picture of haematuria, oedema, hypertension, and reduced urine output seen in acute glomerulonephritis.
9. **Post-streptococcal glomerulonephritis:** acute glomerulonephritis occurring after infection with a nephritogenic strain of Group A streptococcus.
10. **Complement C3:** a laboratory marker characteristically low in post-streptococcal glomerulonephritis, normalising over six to eight weeks.

11. **Anti-streptolysin O titre:** a laboratory test providing evidence of preceding streptococcal infection.
12. **Suprapubic aspiration:** a method of urine collection, particularly useful for reliable culture in infants.

Concept Check Questions [Series 4]

Objective questions

- The functional unit of the kidney is the _____.
- The Schwartz formula estimates GFR using a constant multiplied by height divided by serum _____.
- The most common underlying cause of nephrotic syndrome in children is _____ disease.
- Oedema in nephrotic syndrome classically begins as _____ puffiness, most noticeable on waking.
- Serum complement C3 is characteristically _____ in post-streptococcal glomerulonephritis.

Short-answer questions

1. List two essential functions performed by the kidneys.
2. Describe the components typically assessed on urine dipstick testing.
3. Define nephrotic syndrome by its characteristic clinical triad.
4. List the features of the classic nephritic syndrome presentation.
5. Describe the first-line management of childhood nephrotic syndrome.

Long-answer questions

6. Discuss the anatomy and physiology of the urogenital system and its relevance to clinical evaluation. (Linked to SLO 4.1, SLO 4.2)
7. Discuss the investigation of a child with a suspected urinary system disorder, including evaluation of GFR. (Linked to SLO 4.3, SLO 4.4)
8. Discuss the pathogenesis, clinical features, and management of nephrotic syndrome. (Linked to SLO 4.5, SLO 4.6)
9. Discuss the pathophysiology and clinical features of acute glomerulonephritis. (Linked to SLO 4.7)
10. Compare the nephritic and nephrotic clinical presentations. (Linked to SLO 4.7, SLO 4.8)

Answers to Concept Check Questions [Series 4]

Objective questions

11. Nephron.

12. Creatinine.
13. Minimal change.
14. Periorbital.
15. Low.

Short-answer questions

16. Fluid and electrolyte regulation, acid-base balance, waste excretion, and hormone production.
17. Protein, blood, leucocytes, nitrites, and glucose.
18. Heavy proteinuria, hypoalbuminaemia, and oedema.
19. Haematuria, oedema, hypertension, and reduced urine output.
20. Corticosteroid therapy, typically prednisolone.

Long-answer questions

21. **Basic response:** The nephron, comprising the glomerulus and tubules, is the functional unit of the kidney, and evaluation includes focused history of urinary and systemic symptoms with examination for blood pressure and oedema.

Value-added response: Family history and antenatal findings are particularly relevant, since certain genitourinary conditions have an inherited or congenital basis with implications for further evaluation.

22. **Basic response:** Investigation includes urinalysis, serum urea and creatinine, and imaging such as renal ultrasound, with GFR estimated using the Schwartz formula from height and serum creatinine.

Value-added response: The choice and sequence of investigations should be guided by the clinical question, avoiding unnecessary invasive or radiation-exposing tests where a less invasive alternative suffices.

23. **Basic response:** Nephrotic syndrome results from increased glomerular permeability causing heavy proteinuria, hypoalbuminaemia, and oedema, most commonly due to minimal change disease, managed with corticosteroids.

Value-added response: Important complications include increased infection risk from urinary immunoglobulin loss and increased thromboembolism risk from loss of anticoagulant proteins, both requiring active monitoring.

24. **Basic response:** Acute glomerulonephritis results from immune complex deposition in the glomerulus, most commonly following streptococcal infection, causing haematuria and reduced glomerular filtration.

Value-added response: The nephritic syndrome of haematuria, oedema, hypertension, and oliguria contrasts with the predominantly proteinuria and oedema picture of nephrotic syndrome, though some overlap can occur.

25. **Basic response:** Post-streptococcal glomerulonephritis is confirmed by low complement C3 and evidence of preceding streptococcal infection such as a raised anti-streptolysin O titre.

Value-added response: Management is largely supportive, monitoring fluid balance and blood pressure, since the condition is generally self-limiting with an excellent prognosis in the majority of children.

Answers to Pilot Questions [Series 4]

Part 4.1

1. The nephron, comprising the glomerulus and tubules, is the functional unit of the kidney.
2. Fluid and electrolyte regulation and waste product excretion are essential kidney functions.
3. Frequency, dysuria, urgency, enuresis, and change in urine colour or odour.
4. Family history may suggest an inherited condition affecting the kidneys.
5. Blood pressure, oedema, abdominal masses, and examination of the external genitalia.

Part 4.2

6. Protein, blood, leucocytes, nitrites, and glucose are assessed on dipstick testing.
7. A urine bag sample is prone to contamination, giving potentially misleading culture results.
8. Renal ultrasound and micturating cystourethrogram are commonly used imaging investigations.
9. GFR is the volume of plasma filtered by the glomeruli per unit time, and is the best overall measure of kidney function.
10. Estimated GFR equals a constant multiplied by height in centimetres divided by serum creatinine.

Part 4.3

11. Nephrotic syndrome is defined by heavy proteinuria, hypoalbuminaemia, and oedema.
12. Minimal change disease is the most common cause of nephrotic syndrome in children.
13. Oedema begins as periorbital puffiness, most noticeable on waking, progressing to become generalised.
14. First-line management is corticosteroid therapy, typically prednisolone.
15. Increased infection risk from immunoglobulin loss, and increased thromboembolism risk from loss of anticoagulant proteins.

Part 4.4

1. Immune complex deposition in the glomerulus following streptococcal infection triggers inflammation and reduced filtration.
2. Haematuria, oedema, hypertension, and reduced urine output make up the nephritic syndrome.
3. Urine is typically described as tea-coloured or cola-coloured.
4. Complement C3 is characteristically low, normalising over six to eight weeks.
5. Management is largely supportive, monitoring fluid balance and blood pressure, with an excellent prognosis in most children.

References and Attributions [Series 4]

1. Kliegman, R. M., St Geme, J. W., Blum, N. J., Shah, S. S., Tasker, R. C. and Wilson, K. M. (2020). Nelson Textbook of Pediatrics (21st ed.). Elsevier.
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Figures, Plates, Tables, and Boxes

1. Table 4.1: Key components of the genitourinary history and examination. AI-generated using the prompt: "Create a clean reference table titled Genitourinary History and Examination Checklist, listing component and key features. Simple clinical reference table style with outside border."
2. Box 4.2: The Schwartz formula and its practical application in estimating GFR. AI-generated using the prompt: "Create a simple single-column reference box titled The Schwartz Formula for Estimating GFR. Clean clinical reference box style."
3. Figure 4.3: The pathogenesis of nephrotic syndrome from glomerular protein leak to clinical features. AI-generated using the prompt: "Create a professional medical flowchart titled Pathogenesis of Nephrotic Syndrome, showing the sequence from glomerular permeability to proteinuria, hypoalbuminaemia, and oedema. Clean clinical educational diagram style."
4. Figure 4.4: Comparing the nephritic and nephrotic clinical pictures. AI-generated using the prompt: "Create a professional medical reference chart titled Nephritic versus Nephrotic Syndrome Compared, contrasting key clinical and laboratory features. Clean clinical educational diagram style."

Series 5: Renal Failure and Urinary Tract Disorders

- **Part 5.1: Acute Kidney Injury**
    - Sub Part 5.1.1: Definition, classification, and aetiology of acute kidney injury
    - Sub Part 5.1.2: Clinical presentation and investigation of acute kidney injury
    - Sub Part 5.1.3: Management of acute kidney injury
- **Part 5.2: Chronic Kidney Disease**
    - Sub Part 5.2.1: Definition, classification, and aetiology of chronic kidney disease
    - Sub Part 5.2.2: Clinical presentation of chronic kidney disease
    - Sub Part 5.2.3: Work up and management of chronic kidney disease
- **Part 5.3: Congenital Anomalies of the Kidney and Urinary Tract**
    - Sub Part 5.3.1: Overview of congenital anomalies of the kidney and urinary tract
    - Sub Part 5.3.2: Posterior urethral valves
    - Sub Part 5.3.3: Vesicoureteric reflux
- **Part 5.4: Urinary Tract Infections, Haematuria, and Proteinuria**
    - Sub Part 5.4.1: Definition, aetiology, and forms of presentation of urinary tract infection
    - Sub Part 5.4.2: Pathogenesis, diagnosis, and management of urinary tract infection
    - Sub Part 5.4.3: Evaluation and management of haematuria and proteinuria

Introduction

In the last Series, we explored the evaluation of the genitourinary system and the two most important glomerular disorders, nephrotic syndrome and acute glomerulonephritis. We now turn to renal failure and structural and infective urinary tract disorders. Picture a three year old admitted with severe dehydration whose urine output has fallen sharply and whose blood tests now show a rising creatinine, alongside a six month old with recurrent fevers ultimately found to have a structural urinary tract abnormality. This Series brings together acute and chronic kidney failure, congenital urinary tract anomalies, urinary tract infection, and the evaluation of haematuria and proteinuria, completing your foundation in paediatric genitourinary disorders.

The aim of this Series is to equip you with the knowledge required to recognise and understand the management of acute kidney injury, chronic kidney disease, congenital anomalies of the kidney and urinary tract, urinary tract infection, and haematuria and proteinuria.

This Series begins with **acute kidney injury**, before turning to **chronic kidney disease**. Next, we examine **congenital anomalies of the kidney and urinary tract**, before concluding with **urinary tract infections, haematuria, and proteinuria**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 1.1:** define acute kidney injury and describe its aetiology and classification,
- **SLO 1.2:** describe the pathophysiology, clinical presentation, investigation, and management of acute kidney injury,
- **SLO 1.3:** define chronic kidney disease and describe its aetiology and classification,
- **SLO 1.4:** describe the pathophysiology, clinical presentation, work up, and management of chronic kidney disease,
- **SLO 1.5:** describe the types, pathogenesis, and clinical presentation of congenital anomalies of the kidney and urinary tract, including posterior urethral valves and vesicoureteric reflux,
- **SLO 1.6:** define urinary tract infection and describe its aetiology, risk factors, forms of presentation, and pathogenesis,
- **SLO 1.7:** describe the diagnosis and management of urinary tract infections, and
- **SLO 1.8:** describe the pathophysiology, causes, evaluation, and management of haematuria and proteinuria.

5.1 Acute Kidney Injury

5.1.1 definition, classification, and aetiology of acute kidney injury

Three categories describe where the problem lies

Acute kidney injury, abbreviated AKI, is defined as a sudden decline in kidney function, reflected by a **rise in serum creatinine**, a **fall in urine output**, or both, occurring over hours to days. Causes are classified into three broad categories according to the site of the underlying problem: **prerenal**, resulting from reduced renal perfusion, as in significant dehydration or shock, **intrinsic renal**, resulting from direct damage to the kidney itself, as in acute

glomerulonephritis or acute tubular necrosis, and **postrenal**, resulting from obstruction to urine flow anywhere from the renal pelvis to the urethra.

Prerenal AKI is the most common category in children and is often **reversible** with prompt restoration of adequate renal perfusion, though if renal hypoperfusion is severe or prolonged, it can progress to **intrinsic renal injury** through **acute tubular necrosis**, meaning early recognition and correction of the underlying cause is essential to prevent this progression.

Fill in the blank: Prerenal AKI is often reversible with prompt restoration of adequate renal _____.

5.1.2 clinical presentation and investigation of acute kidney injury

Clinical presentation of AKI varies according to the underlying cause but commonly includes **reduced urine output**, or **oliguria**, **oedema** from fluid retention, and, in more severe cases, symptoms related to **uraemia**, such as **nausea**, **vomiting**, and **lethargy**, together with features of the underlying precipitating illness, such as signs of dehydration in prerenal AKI or the specific features of acute glomerulonephritis in intrinsic renal AKI.

Investigation includes **serum urea and creatinine** to confirm and stage the degree of renal impairment, **serum electrolytes**, since AKI is frequently accompanied by **hyperkalaemia** and **metabolic acidosis**, **urinalysis**, and **renal ultrasound**, which is particularly important for identifying or excluding a **postrenal, obstructive cause**, since this category of AKI often requires specific urological intervention rather than medical management alone.

Fill in the blank: AKI is frequently accompanied by hyperkalaemia and metabolic _____.

5.1.3 management of acute kidney injury

Management of AKI focuses on **treating the underlying cause**, such as fluid resuscitation in prerenal AKI or relief of obstruction in postrenal AKI, together with careful **fluid balance management**, avoiding both under-replacement, which can worsen prerenal injury, and over-replacement, which can precipitate fluid overload in a child with reduced urine output. **Electrolyte abnormalities**, particularly hyperkalaemia, must be actively monitored and corrected, using the emergency measures discussed earlier in this course where severe.

In children with **severe AKI**, particularly with severe electrolyte disturbance, significant fluid overload, or uraemic symptoms not responding to medical management, **renal replacement therapy**, such as **peritoneal dialysis** or **haemodialysis**, may be required, and the overall prognosis of AKI in children is generally favourable when the underlying cause is identified and

treated promptly, though some children, particularly those with severe or prolonged intrinsic renal injury, can experience lasting renal impairment.

Fill in the blank: Renal replacement therapy such as peritoneal dialysis may be required in _____ acute kidney injury.

Table 5.1: Classification of acute kidney injury by underlying category.

Category	Mechanism	Example cause
Prerenal	Reduced renal perfusion	Severe dehydration or shock
Intrinsic renal	Direct damage to the kidney	Acute glomerulonephritis, acute tubular necrosis
Postrenal	Obstruction to urine flow	Posterior urethral valves, severe bilateral hydronephrosis

Pilot questions 5.1

- Define acute kidney injury. (Linked to SLO 5.1)
- Distinguish prerenal, intrinsic renal, and postrenal acute kidney injury. (Linked to SLO 5.1)
- Explain why prerenal AKI can progress to intrinsic renal injury if untreated. (Linked to SLO 5.1)
- List the key investigations used in evaluating a child with suspected acute kidney injury. (Linked to SLO 5.2)
- Describe when renal replacement therapy may be required in acute kidney injury. (Linked to SLO 5.2)

5.2 Chronic Kidney Disease

5.2.1 definition, classification, and aetiology of chronic kidney disease

A problem defined by duration, not just severity

Chronic kidney disease, abbreviated CKD, is defined as abnormalities of kidney structure or function, present for **three months or longer**, with implications for health, distinguishing it from the acute, potentially reversible process of AKI. CKD is classified into **stages**, from stage 1, representing normal or increased GFR with other evidence of kidney damage, through to stage 5, representing **kidney failure** requiring renal replacement therapy, based primarily on the estimated glomerular filtration rate.

In children, the leading causes of CKD differ from those in adults, with **congenital anomalies of the kidney and urinary tract**, discussed further in the next Part of this Series, accounting for a substantial proportion of cases, alongside **glomerular diseases**, such as focal segmental glomerulosclerosis, and, less commonly in children than adults, conditions such as diabetic nephropathy.

Fill in the blank: Chronic kidney disease is defined as abnormalities of kidney structure or function present for _____ months or longer.

5.2.2 clinical presentation of chronic kidney disease

Clinical presentation of CKD in children is often **insidious**, with early stages frequently **asymptomatic** and detected only through screening or incidental investigation, while more advanced disease presents with **growth faltering**, reflecting the combined effects of chronic uraemia, metabolic bone disease, and often poor appetite, **anaemia**, resulting from reduced renal production of **erythropoietin**, and **renal osteodystrophy**, a disturbance of bone metabolism resulting from disordered calcium, phosphate, and vitamin D handling.

As CKD progresses towards **kidney failure**, symptoms of **uraemia** become more prominent, including fatigue, nausea, and reduced appetite, alongside **fluid overload** and **electrolyte disturbance**, and the cumulative impact of chronic illness, dietary restriction, and multiple medications can also have a significant psychosocial impact on the child and family that should not be overlooked in overall management.

Fill in the blank: Anaemia in chronic kidney disease results from reduced renal production of _____.

5.2.3 work up and management of chronic kidney disease

Work up of suspected CKD includes **serum urea and creatinine** with **estimated GFR**, **electrolytes**, **calcium**, **phosphate**, and **parathyroid hormone** to assess for renal osteodystrophy, **full blood count** to assess for anaemia, and **renal ultrasound** to assess for an underlying structural cause, with the specific underlying diagnosis guiding any further, more targeted investigation required.

Management is **multidisciplinary** and includes addressing the **underlying cause** where possible, **nutritional support** to optimise growth, **management of anaemia**, often with **erythropoiesis-stimulating agents**, **management of renal osteodystrophy**, with **phosphate binders** and active vitamin D analogues, and, in **advanced kidney failure**, planning for **renal replacement therapy**, whether dialysis or, ultimately, **kidney transplantation**, which offers the best long-term outcome for eligible children.

Fill in the blank: Management of renal osteodystrophy typically includes phosphate binders and active _____ D analogues.

Box 5.2: Leading causes of chronic kidney disease in children.

Leading causes of paediatric CKD
Congenital anomalies of the kidney and urinary tract: the most common overall category.
Glomerular diseases: including focal segmental glomerulosclerosis and chronic glomerulonephritis.
Hereditary nephropathies: including cystic kidney diseases and other inherited conditions.
Vascular and other acquired causes: relatively less common than in adults.
Sequelae of severe or repeated acute kidney injury: in some children.

Pilot questions 5.2

1. Define chronic kidney disease and distinguish it from acute kidney injury. (Linked to SLO 5.3)
2. Name the leading category of cause of CKD in children. (Linked to SLO 5.3)
3. Describe the clinical presentation of advanced chronic kidney disease. (Linked to SLO 5.4)
4. Explain the cause of anaemia in chronic kidney disease. (Linked to SLO 5.4)
5. Describe the general principles of managing a child with chronic kidney disease. (Linked to SLO 5.4)

5.3 Congenital Anomalies of the Kidney and Urinary Tract

5.3.1 overview of congenital anomalies of the kidney and urinary tract

CAKUT spans a wide spectrum of severity

Congenital anomalies of the kidney and urinary tract, abbreviated **CAKUT**, encompass a broad spectrum of structural abnormalities arising from disordered embryological development of the urinary system, ranging from relatively minor findings, such as mild **hydronephrosis** that resolves spontaneously, to severe abnormalities such as **renal agenesis** or **severe bilateral obstruction**, which can be life-threatening in the neonatal period.

CAKUT is increasingly identified through **antenatal ultrasound screening**, which has substantially improved early detection and allowed planned postnatal evaluation and management, and collectively, CAKUT represents the leading cause of chronic kidney disease

and kidney failure in children, underscoring the clinical importance of recognising and appropriately following up even apparently minor antenatally detected abnormalities.

Fill in the blank: CAKUT collectively represents the leading cause of chronic kidney disease and kidney failure in _____.

5.3.2 posterior urethral valves

Posterior urethral valves, abbreviated PUV, are abnormal membranous folds within the posterior urethra found exclusively in **male infants**, causing varying degrees of **bladder outlet obstruction**. This obstruction can have significant effects on the developing urinary tract even before birth, including **bilateral hydronephrosis**, **bladder wall thickening**, and, in severe cases, impaired lung development from associated **oligohydramnios**, since adequate fetal urine output is essential for normal amniotic fluid volume.

PUV should be suspected antenatally when **bilateral hydronephrosis** with a **thickened bladder wall** is identified on ultrasound in a male fetus, and postnatally may present with a **poor urinary stream**, a **palpable bladder**, or, in less severe cases, may be identified later during evaluation for a urinary tract infection. Diagnosis is confirmed with a **micturating cystourethrogram**, and management involves **prompt bladder drainage**, typically with a urinary catheter, followed by **surgical valve ablation** once the child is stable.

Fill in the blank: Posterior urethral valves occur exclusively in _____ infants.

5.3.3 vesicoureteric reflux

Vesicoureteric reflux, abbreviated VUR, refers to the **retrograde flow of urine** from the bladder back up the ureter, and sometimes into the renal pelvis, resulting from an abnormality of the normally one-way valve mechanism at the **vesicoureteric junction**. VUR is graded from **I to V** according to severity, based on the extent of reflux and any associated dilation seen on imaging, with higher grades associated with a greater risk of complications.

The principal clinical significance of VUR is an increased risk of **urinary tract infection**, and, particularly with **febrile UTIs** in the context of higher grade reflux, a risk of **renal scarring**, which can, over time, contribute to hypertension and chronic kidney disease if recurrent and unrecognised. Management ranges from **conservative observation** with prompt treatment of any UTI for lower grade reflux, since many cases resolve spontaneously with growth, to **surgical or endoscopic correction** for higher grade or persistent reflux associated with recurrent infection.

Fill in the blank: Vesicoureteric reflux is graded from I to _____ according to severity.

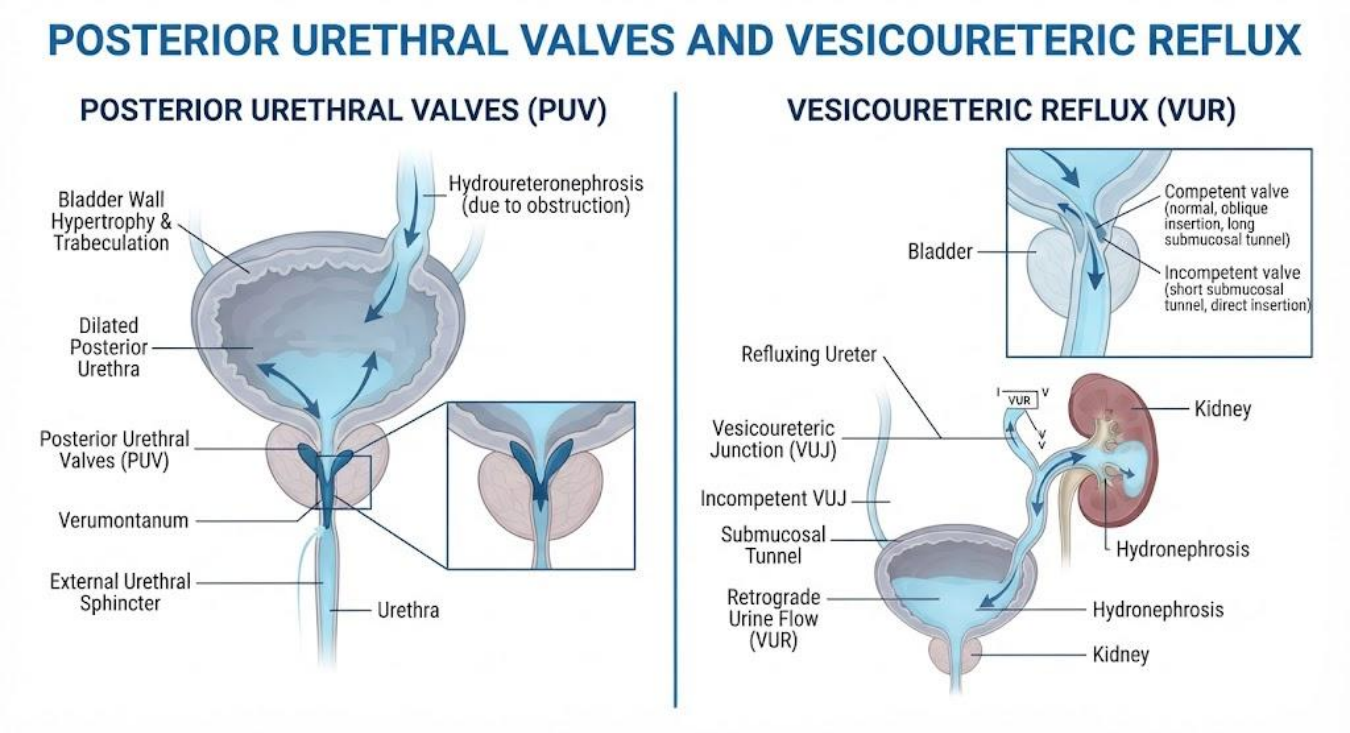

Figure 5.3: Comparing the anatomical basis of posterior urethral valves and vesicoureteric reflux.

Pilot questions 5.3

1. Define CAKUT and explain its overall clinical significance. (Linked to SLO 5.5)
2. Describe the antenatal ultrasound findings that should raise suspicion for posterior urethral valves. (Linked to SLO 5.5)
3. Describe the management of confirmed posterior urethral valves. (Linked to SLO 5.5)
4. Define vesicoureteric reflux and describe how it is graded. (Linked to SLO 5.5)
5. Explain the principal clinical significance of vesicoureteric reflux. (Linked to SLO 5.5)

5.4 Urinary Tract Infections, Haematuria, and Proteinuria

5.4.1 definition, aetiology, and forms of presentation of urinary tract infection

Presentation varies dramatically with age

A **urinary tract infection**, abbreviated UTI, is defined as the presence of significant **bacteriuria** together with symptoms or signs of infection, and is classified anatomically into **lower UTI**, or **cystitis**, confined to the bladder, and **upper UTI**, or **pyelonephritis**, involving the kidney itself, a distinction that carries different implications for both treatment and the risk of long-term renal scarring. The most common causative organism is **Escherichia coli**, though other Gram-negative organisms are also implicated.

Risk factors for UTI include female sex beyond early infancy, uncircumcised status in male infants, **constipation**, and underlying **structural urinary tract abnormalities** such as vesicoureteric reflux, and **presentation varies considerably by age**, with infants often presenting non-specifically with **fever**, **poor feeding**, or **vomiting** without localising urinary symptoms, while older children more typically present with classic symptoms such as **dysuria**, **frequency**, or **loin pain**.

Fill in the blank: UTI in infants often presents non-specifically with fever, poor feeding, or _____, without localising urinary symptoms.

5.4.2 pathogenesis, diagnosis, and management of urinary tract infection

The pathogenesis of UTI most commonly involves **ascending infection**, in which bacteria from the perineal or periurethral area migrate up the urethra into the bladder, and, in the presence of **vesicoureteric reflux** or other predisposing factors, may ascend further to involve the kidney, causing pyelonephritis, which carries a greater risk of subsequent **renal scarring** than a straightforward lower UTI confined to the bladder.

Diagnosis relies on **urine microscopy and culture**, obtained using an appropriate collection method as discussed earlier in this Series, with a **significant colony count** of a single organism supporting the diagnosis. Management includes appropriate **antibiotic therapy**, guided by local resistance patterns and later adjusted according to culture sensitivity results, with the **route and duration** depending on the child's age, severity of illness, and whether pyelonephritis is suspected, together with **imaging follow up**, such as renal ultrasound, particularly after a first febrile UTI in a young child, to identify any underlying structural abnormality requiring further evaluation.

Fill in the blank: Ascending infection with subsequent involvement of the kidney is termed _____.

5.4.3 evaluation and management of haematuria and proteinuria

Haematuria, the presence of blood in the urine, may be **macroscopic**, visible to the naked eye, or **microscopic**, detected only on dipstick or microscopy, and causes range from **benign**, such as a recent viral illness or vigorous exercise, to **significant glomerular or urological pathology**, such as glomerulonephritis, UTI, or, less commonly in children, urinary tract calculi or tumour, meaning persistent or significant haematuria always warrants further evaluation rather than being dismissed.

Proteinuria, an excess of protein in the urine, may be **transient**, related to fever or exercise, or **persistent**, which is more concerning and requires further evaluation, including **quantification** of the degree of proteinuria and consideration of an underlying glomerular cause, since persistent, significant proteinuria, particularly when accompanied by haematuria, hypertension, or reduced renal function, is an important marker of chronic kidney disease and warrants referral for specialist nephrology evaluation.

Fill in the blank: Persistent, significant proteinuria is an important marker of chronic _____ disease.

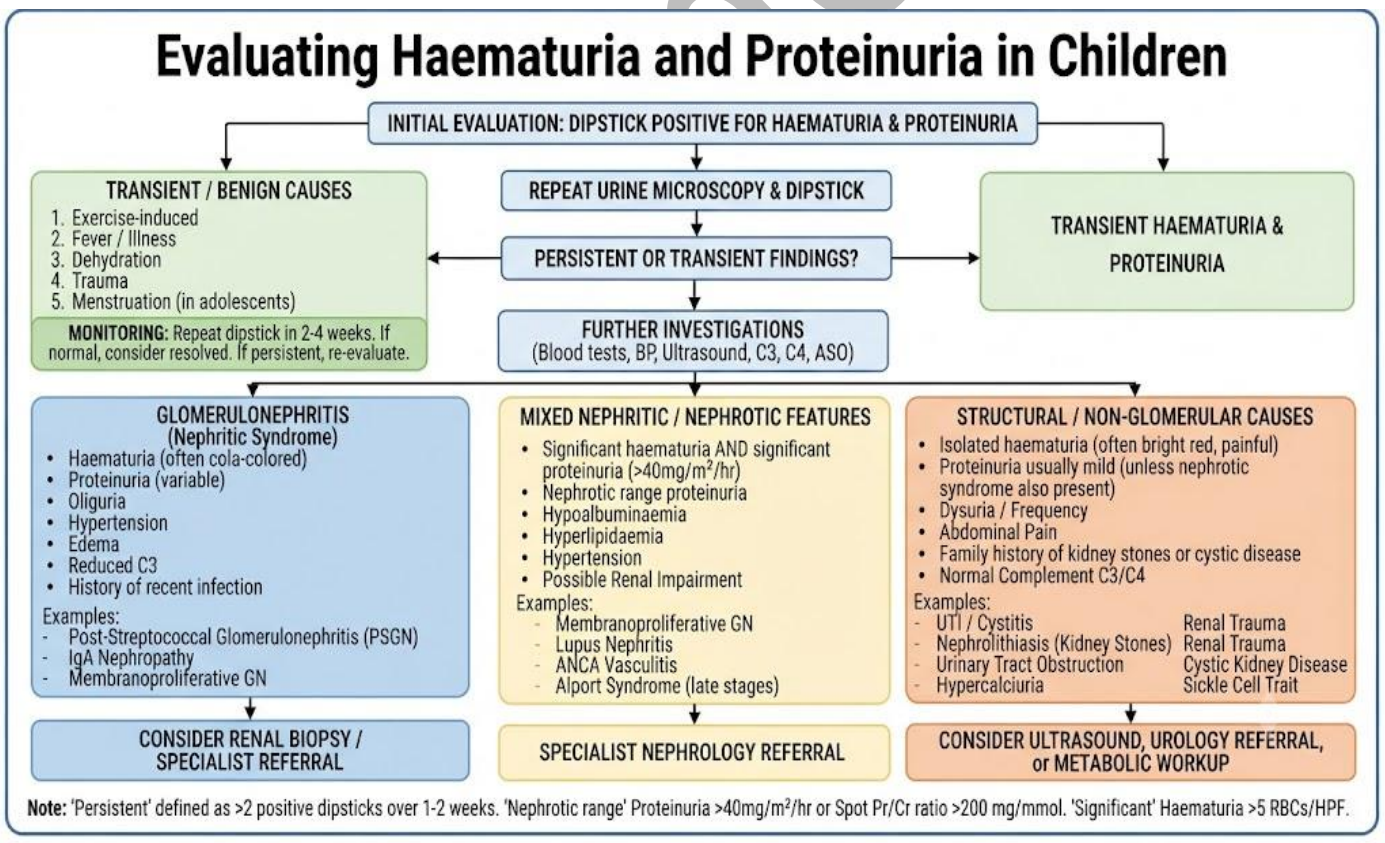

Figure 5.4: A structured approach to evaluating a child with haematuria or proteinuria.

Pilot questions 5.4

1. Define urinary tract infection and distinguish cystitis from pyelonephritis. (Linked to SLO 5.6)
2. List two risk factors for urinary tract infection in children. (Linked to SLO 5.6)
3. Describe the typical presentation of UTI in an infant compared with an older child. (Linked to SLO 5.6)
4. Describe the diagnosis and general management of a urinary tract infection. (Linked to SLO 5.7)
5. Distinguish transient from persistent proteinuria and explain the significance of the distinction. (Linked to SLO 5.8)

Series Summary

This Series examined **renal failure and urinary tract disorders**, beginning with **acute kidney injury** and its classification, before turning to **chronic kidney disease** and its distinct paediatric causes and presentation.

We then examined **congenital anomalies of the kidney and urinary tract**, including posterior urethral valves and vesicoureteric reflux, before concluding with **urinary tract infections** and the evaluation of **haematuria and proteinuria**. Having worked through this Series, and indeed this course, you should now be equipped with a systematic approach to the evaluation and management of the common gastrointestinal and genitourinary conditions encountered in paediatric practice.

Glossary of Terms

1. **Acute kidney injury:** a sudden decline in kidney function reflected by a rise in creatinine, a fall in urine output, or both.
2. **Prerenal AKI:** acute kidney injury resulting from reduced renal perfusion, often reversible with prompt correction.
3. **Acute tubular necrosis:** intrinsic renal injury that can result from severe or prolonged renal hypoperfusion.
4. **Chronic kidney disease:** abnormalities of kidney structure or function present for three months or longer.
5. **Renal osteodystrophy:** a disturbance of bone metabolism resulting from disordered calcium, phosphate, and vitamin D handling in chronic kidney disease.

6. **CAKUT:** congenital anomalies of the kidney and urinary tract, the leading cause of chronic kidney disease in children.
7. **Posterior urethral valves:** abnormal membranous folds in the male posterior urethra causing bladder outlet obstruction.
8. **Vesicoureteric reflux:** retrograde flow of urine from the bladder up the ureter due to an incompetent vesicoureteric junction.
9. **Pyelonephritis:** upper urinary tract infection involving the kidney, carrying a greater risk of renal scarring than cystitis.
10. **Cystitis:** lower urinary tract infection confined to the bladder.
11. **Haematuria:** the presence of blood in the urine, either macroscopic or microscopic.
12. **Proteinuria:** an excess of protein in the urine, which may be transient or persistent.

Concept Check Questions [Series 5]

Objective questions

- Prerenal AKI is often reversible with prompt restoration of adequate renal _____.
- Chronic kidney disease is defined as abnormalities of kidney structure or function present for _____ months or longer.
- Posterior urethral valves occur exclusively in _____ infants.
- Vesicoureteric reflux is graded from I to _____ according to severity.
- Ascending infection with subsequent involvement of the kidney is termed _____.

Short-answer questions

1. Distinguish prerenal, intrinsic renal, and postrenal acute kidney injury.
2. Name the leading category of cause of chronic kidney disease in children.
3. Describe the antenatal ultrasound findings suggestive of posterior urethral valves.
4. Describe the typical presentation of a urinary tract infection in an infant.
5. Distinguish transient from persistent proteinuria.

Long-answer questions

6. Discuss the classification, presentation, and management of acute kidney injury. (Linked to SLO 5.1, SLO 5.2)
7. Discuss the aetiology, presentation, and management of chronic kidney disease in children. (Linked to SLO 5.3, SLO 5.4)

8. Discuss the pathogenesis and clinical presentation of posterior urethral valves and vesicoureteric reflux. (Linked to SLO 5.5)
9. Discuss the diagnosis and management of urinary tract infection in children. (Linked to SLO 5.6, SLO 5.7)
10. Discuss the evaluation of a child presenting with haematuria or proteinuria. (Linked to SLO 5.8)

Answers to Concept Check Questions [Series 5]

Objective questions

11. Perfusion.
12. Three.
13. Male.
14. Five.
15. Pyelonephritis.

Short-answer questions

16. Prerenal results from reduced perfusion, intrinsic renal from direct kidney damage, and postrenal from obstruction to urine flow.
17. Congenital anomalies of the kidney and urinary tract are the leading cause of CKD in children.
18. Bilateral hydronephrosis with a thickened bladder wall in a male fetus.
19. Fever, poor feeding, or vomiting without localising urinary symptoms.
20. Transient proteinuria relates to fever or exercise and resolves, while persistent proteinuria requires further evaluation for an underlying glomerular cause.

Long-answer questions

21. **Basic response:** Acute kidney injury is classified as prerenal, intrinsic renal, or postrenal, presenting with oliguria and uraemic symptoms, and managed by treating the underlying cause and correcting fluid and electrolyte disturbance.

Value-added response: Severe cases with refractory electrolyte disturbance, fluid overload, or uraemic symptoms may require renal replacement therapy, though the overall prognosis is generally favourable when the cause is treated promptly.

22. **Basic response:** Chronic kidney disease in children is most commonly caused by congenital anomalies of the kidney and urinary tract, presenting insidiously with growth faltering, anaemia, and renal osteodystrophy.

Value-added response: Management is multidisciplinary, addressing the underlying cause, nutrition, anaemia, and bone disease, with renal replacement therapy or transplantation considered in advanced kidney failure.

23. **Basic response:** Posterior urethral valves cause bladder outlet obstruction in male infants, suspected antenatally by bilateral hydronephrosis, while vesicoureteric reflux causes retrograde urine flow up the ureter.

Value-added response: Both conditions increase the risk of urinary tract infection and renal scarring, with management ranging from prompt drainage and valve ablation in PUV to conservative or surgical management in VUR depending on severity.

24. **Basic response:** UTI is diagnosed by urine microscopy and culture, classified as cystitis or pyelonephritis, and managed with antibiotics guided by local resistance and culture sensitivity.

Value-added response: Imaging follow up such as renal ultrasound is important after a first febrile UTI in a young child to identify an underlying structural abnormality such as vesicoureteric reflux.

25. **Basic response:** Haematuria and proteinuria range from benign, transient findings to markers of significant glomerular or urological pathology requiring further evaluation.

Value-added response: Persistent findings, particularly when accompanied by hypertension or reduced renal function, warrant specialist nephrology referral, since they are important markers of underlying chronic kidney disease.

Answers to Pilot Questions [Series 5]

Part 5.1

1. AKI is a sudden decline in kidney function reflected by a rise in creatinine, a fall in urine output, or both.
2. Prerenal results from reduced perfusion, intrinsic renal from direct kidney damage, and postrenal from obstruction to urine flow.
3. Severe or prolonged hypoperfusion can cause acute tubular necrosis, converting reversible prerenal injury into intrinsic renal damage.
4. Serum urea and creatinine, electrolytes, urinalysis, and renal ultrasound are key investigations.
5. Renal replacement therapy may be required in severe AKI with refractory electrolyte disturbance, fluid overload, or uraemic symptoms.

Part 5.2

6. CKD is defined by abnormalities of kidney structure or function present for three months or longer, distinguishing it from the acute, often reversible process of AKI.

7. Congenital anomalies of the kidney and urinary tract are the leading cause of CKD in children.
8. Advanced CKD presents with growth faltering, anaemia, renal osteodystrophy, and eventually uraemic symptoms.
9. Anaemia results from reduced renal production of erythropoietin.
10. Management is multidisciplinary, addressing the underlying cause, nutrition, anaemia, bone disease, and planning for renal replacement therapy if needed.

Part 5.3

11. CAKUT encompasses structural urinary tract abnormalities and is the leading cause of chronic kidney disease in children.
12. Bilateral hydronephrosis with a thickened bladder wall in a male fetus suggests posterior urethral valves.
13. Management involves prompt bladder drainage followed by surgical valve ablation once stable.
14. VUR is retrograde flow of urine from the bladder up the ureter, graded from I to V by severity.
15. The principal significance of VUR is an increased risk of urinary tract infection and renal scarring.

Part 5.4

1. UTI is significant bacteriuria with symptoms or signs of infection; cystitis is confined to the bladder while pyelonephritis involves the kidney.
2. Female sex, uncircumcised status, constipation, and structural urinary tract abnormalities are risk factors.
3. Infants present non-specifically with fever, poor feeding, or vomiting, while older children present with dysuria, frequency, or loin pain.
4. Diagnosis relies on urine microscopy and culture, and management involves appropriate antibiotics and imaging follow up.
5. Transient proteinuria relates to fever or exercise and resolves, while persistent proteinuria requires evaluation for an underlying glomerular cause.

References and Attributions [Series 5]

1. Kliegman, R. M., St Geme, J. W., Blum, N. J., Shah, S. S., Tasker, R. C. and Wilson, K. M. (2020). Nelson Textbook of Pediatrics (21st ed.). Elsevier.
2. Kidney Disease: Improving Global Outcomes (KDIGO) (2012). Clinical Practice Guideline for Acute Kidney Injury.
3. National Institute for Health and Care Excellence (2018). Urinary Tract Infection in Under 16s: Diagnosis and Management. NICE Guideline.
4. Woolf, A. S. and Thiruchelvam, N. (1998). Congenital obstructive uropathy: its origin and contribution to end-stage renal disease in children. Advances in Renal Replacement Therapy, 5(4), 279 to 289.

Figures, Plates, Tables, and Boxes

1. Table 5.1: Classification of acute kidney injury by underlying category. AI-generated using the prompt: "Create a clean reference table titled Classification of Acute Kidney Injury, listing category, mechanism, and example cause. Simple clinical reference table style with outside border."
2. Box 5.2: Leading causes of chronic kidney disease in children. AI-generated using the prompt: "Create a simple single-column reference box titled Leading Causes of Chronic Kidney Disease in Children. Clean clinical reference box style."
3. Figure 5.3: Comparing the anatomical basis of posterior urethral valves and vesicoureteric reflux. AI-generated using the prompt: "Create a professional medical diagram titled Posterior Urethral Valves and Vesicoureteric Reflux, showing the anatomical basis of each condition side by side. Clean clinical educational diagram style."
4. Figure 5.4: A structured approach to evaluating a child with haematuria or proteinuria. AI-generated using the prompt: "Create a professional medical flowchart titled Evaluating Haematuria and Proteinuria in Children, branching by transient versus persistent findings and associated features. Clean clinical educational diagram style."