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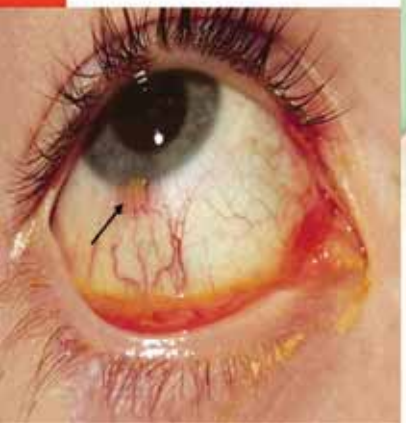
Indumathy Santhanam

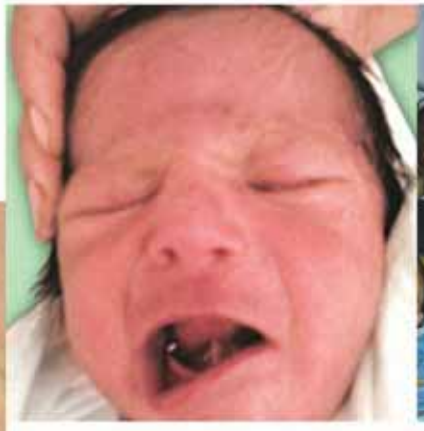
Foreword
T Dorairajan

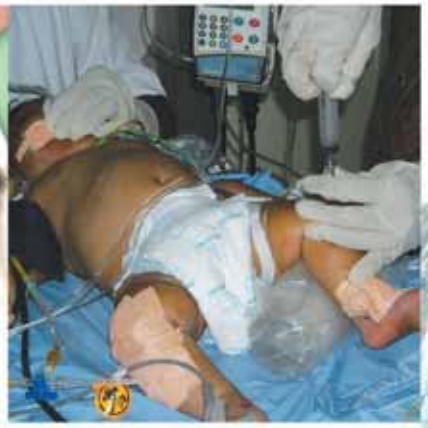
Illustrated Textbook of
PEDIATRICS

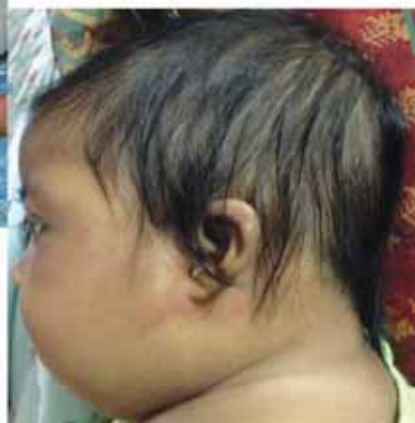
Highlights

- Exclusive chapter on Pediatric Resuscitation and Emergency Medicine
- Presented with more than 650 clinical photographs
- Includes color atlas on how to perform neurological examination of an infant
- Attractive history panel with every chapter.









Illustrated Textbook of PEDIATRICS

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Foreword
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Dedicated to

My father Sri PCM Santhanam, a karma veer,
who tread the path of dharma despite extreme adversity
whilst pursuing his goal with unwavering zeal.

Tamaso mā jyotirgamaya
(Lead Me from Darkness to Light)

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FOREWORD

It is a pleasure to write the foreword for this *Illustrated Textbook of Pediatrics* for the undergraduate students. This concise book is written by the experienced teaching faculty from the Institute of Child Health attached to the Madras Medical College, Chennai, Tamil Nadu, India. The lucid prose, clear diagrams and timelines make this an attractive and enchanting book for the medical students entering pediatric practise.

Understanding disease processes in children requires a robust grasp of physiology, anatomy and biochemistry. More often than not, basic sciences is forgotten by the time the student reaches clinical postings. I am happy to note that this book has made an attempt to bridge this gap. The correlation, which this books attempts, is so crucial in the understanding of disease processes.

The medical community of today, stand on the shoulders of giants. It is a pleasure to note the importance given to pioneers and scientists who have made valuable contributions to our understanding of medicine.

The chapter on how to resuscitate critically ill children with comprehensive clinical photographs, is one more unique feature. The chapter on "how to examine the central nervous system of an infant" is another special feature. As one reads this book, one gets the impression that each one of the contributors have shared a tremendous passion for sharing their expertise with students.

This textbook contains excellent information that is relevant to the Indian subcontinent. I wish this edition all success.

T Dorairajan MS FRACS
An alumni of Madras Medical College, trained in
Pediatric Surgery in 1961 in Melbourne Royal Children's Hospital
In a meritorious career spanning five decades,
he spearheaded excellence in surgical care of children in India

PREFACE

Way back in the 1980s, undergraduates (UGs) rotated for 3 months every year in medicine, surgery, and obstetrics and gynecology. Medical students would rush to reach the outpatient department (OPD) before 7 am. It was not uncommon for a patient to be auscultated concurrently by several eager students. In the dark and dingy OPD, teeming with patients, we jostled to hear what our teachers taught. Those of us, not close enough, were eagerly sought by residents, who would point us to other interesting cases. Assistant professors, residents, final years' students and house officers would vie with each other to teach us. A large number of students would prefer festival days to come to the hospital, since the OPD was less crowded and teachers would have more time to teach. Hutchinson was a constant pocket companion. Evening classes by duty surgeons and physicians were the norm. Harrison, Robbins, Ganong, Katzung, Harper, Goodman Gilman were treasured. Students who could recap from these books were revered! It was not easy to pass medical examinations, perhaps resulting in a robust foundation in clinical medicine!

Spending time with patients in the OPD and in the wards, helped develop values important in the practise of medicine, handle clinical responsibilities, do tedious tasks, empathise with patients and interact with colleagues to improve patient care.

Fast forward into the now: As per the Medical Council of India (MCI) norms, the continuous 3-month postings in core subjects have been eliminated. Case discussions have been shifted from the real world into the OPD classroom. Separate teachers are unavailable for every batch, resulting in poor exposure for the first and second year students. Students, tend to skip OPD and ward sessions, preferring to enrol for postgraduate (PG) preparation. Interest in clinical medicine appears to have waned. Medical information and technology has exponentially increased. Standard textbooks have been replaced by on-line resources. Inundated with voluminous information, and lacking in sustained clinical exposure, the young medico is unable to understand foundation facts. On the contrary, passing UG examinations has never been so easy! Perhaps, as a result of this malady, the majority who entered residency, seemed woefully under prepared for postgraduate training.

Hence, when I received a call from the M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India in 2015 requesting to write an undergraduate textbook of pediatrics, I agreed.

The Madras Medical College, established in 1835, has an annual intake of 250 undergraduates and around 50 postgraduates. Sharing a common concern, a group of motivated and talented pediatricians primarily from the Institute of Child Health, Madras Medical College, Chennai, Tamil Nadu, India agreed to work towards creating a resource for undergraduates.

The focus would be to make it simple, attractive with an emphasis on understanding the relationship between basic science with clinical medicine. Pretty cartoons were included to make complex facts, easy to understand. History panels were designed to provide the young medico a reminder of our past.

A chapter on Emergency Medicine was added. Infectious diseases were pictorially represented with time lines. An atlas of neurological examination of an infant was added. The national programs were presented with an emphasis on the understanding on life cycles of vectors. Potentially dull topics, such as fluid and electrolytes, acid-base balance and diabetes ketoacidosis, were made immensely attractive. All topics were edited with the intention of making every aspect of pediatrics a passion!

It is our hope that this book will help in a big way to achieve our collective dream to inspire and reach out to the next generation medicos!

The royalties from this book will go to SEH Trust which will be donated for the care and academic activities in relationship to the critically ill children in the Pediatric Emergency Department of the Institute of Child Health, Madras Medical College.

Indumathy Santhanam

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I thank my colleagues at the Institute of Child Health, Madras Medical College (MMC), Chennai, Tamil Nadu, India, for their huge support and effort taken to create this resource. I thank Dr Vijayalakshmi MD DMRD, Professor, Department of Pediatric Radiology, Institute of Child Health and Hospital for Children, Madras Medical College, Chennai, Tamil Nadu, India, for sharing her expertise and collection of radiological images for this book. I am extremely grateful to Dr B Ramesh Babu MD DCH (Ped), Associate Professor, Department of Pediatrics, Government Dharmapuri Medical College and Hospital, Dharmapuri, Tamil Nadu, India, whose generosity in sharing pictures from the Department of Neonatology, Government Dharmapuri Medical College and Hospital, has vastly enriched the content.

I also thank the parents who gave consent to take pictures and use them for educational purposes.

I thank the MMC students' editorial committee, Dr Vignesh, Dr Veena, Dr Varshini and Dr Sai Siva, for checking out the chapters for clarity and cuteness index!

My thanks to Dr Sharada Satish and Dr Akila, who helped in proofreading some of the chapters.

My deepest gratitude to my best friend, Dr Ramesh Dorairajan, my daughter Varshini Ramesh, my parents, Subashini and Kicchamma, who never let me forget that this book should inspire and enlighten the next generation of young medicos.

My special thanks to Shri Jitendar P Vij (Group Chairman), Mr Ankit Vij (Group President), Ms Ritu Sharma (Director–Content Strategy), Ms Chetna Malhotra Vohra (Associate Director–Content Strategy), Dr Madhu Choudhary (Senior Content Strategist), Ms Sunita Katla (PA to Group Chairman and Publishing Manager), Mr Manish Pahuja (General Manager–Production), Mr Sumit Kumar (Team Leader–Designers, Production), Mr Laxmidhar Padhiary (Proofreader) and Mr Kapil Dev Sharma (DTP Operator), for making this book possible. Ms Ritu Sharma and Dr Madhu Choudhary have virtually resuscitated this textbook. Their prompt response, dedication and excellence in medical editing helped us achieve our collective goals for quality.

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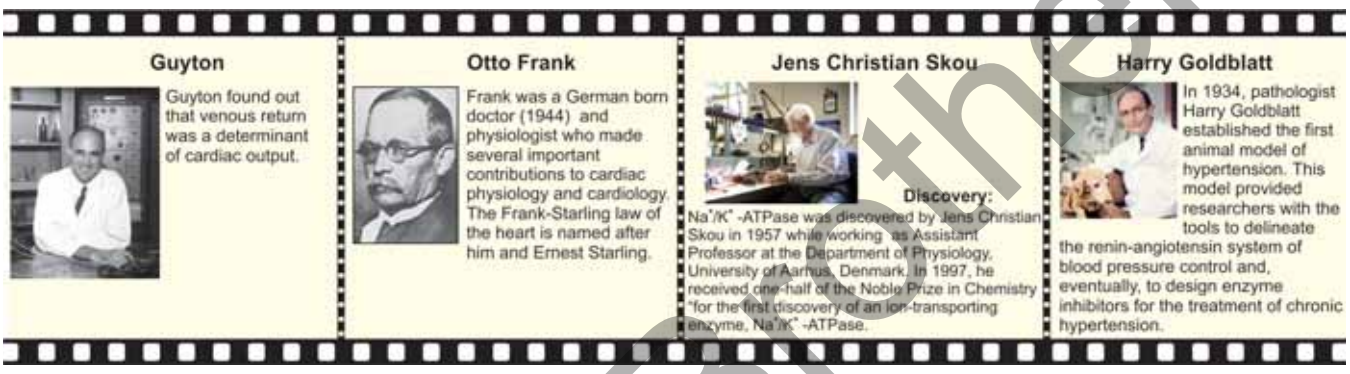
CHAPTER

13

Fluid and Electrolyte Disturbances

J Balaji, S Thangavelu, Indumathy Santhanam



OBJECTIVES

1. Applied physiology of fluids
2. Serum osmolarity and tonicity
3. How do we manage loss of body fluids
4. Role played by sodium, potassium and calcium in our body
5. What happens when these electrolytes increase or decrease
6. Management of electrolyte disturbances

PHYSIOLOGY: FLUIDS

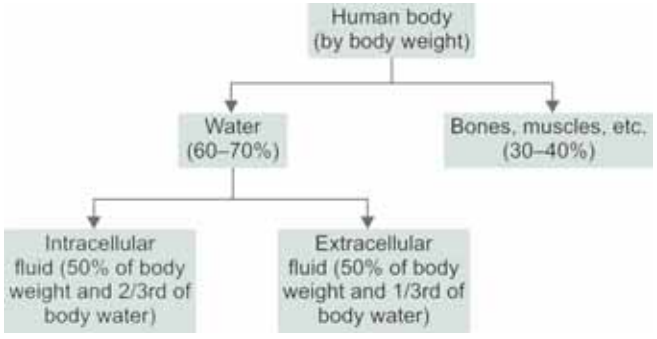
Two-thirds of body weight is contributed by water, i.e. total body water (TBW) (Table 13.1). One-third of TBW is derived from extracellular fluid (ECF) and two-thirds from intracellular fluid (ICF) (Flowchart 13.1 and Fig. 13.1).

Table 13.1: Body water compartments in various ages (% of body weight).

				Adult	
	Preterm	*Term newborn*	*Infant and child*	*Male*	*Female*
ICF	–	40	40	40	40
ECF	–	30	25	20	15
TBW	80	70	65	60	55

(ICF: intracellular fluid; ECF: extracellular fluid; TBW: total body water)

Flowchart 13.1: Body water by body weight.



One-third of ECF is derived from plasma and two-thirds from interstitial fluid (ISF) which surrounds the

cells. Transcellular fluid contributes to 1.5% of body weight. The latter is fluid within the pleura, peritoneum, pericardium and cerebrospinal compartment.

Total body water constitutes around 80% of birth weight of preterms. As the preterm matures, TBW shrinks to 70% in the term neonate. During intrauterine life and soon thereafter ECF exceeds ICF. At 1 year of age, TBW drops to 60% and remains constant until puberty. Increased fat in girls during puberty leads to further drop of TBW (55%). However, the overall ICF compartment remains constant (40%) in age groups (Fig. 13.2). The gradual decrease in TBW with increasing age is reflected in the shrinking of ECF compartment.

Fat contains less water than other tissues. Hence obesity is associated with proportionately less TBW.

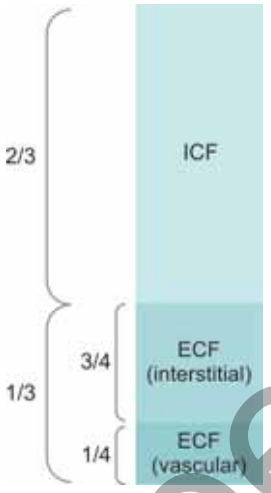


Fig. 13.1: Distribution of total body water. (ICF: intracellular fluid; ECF: extracellular fluid)

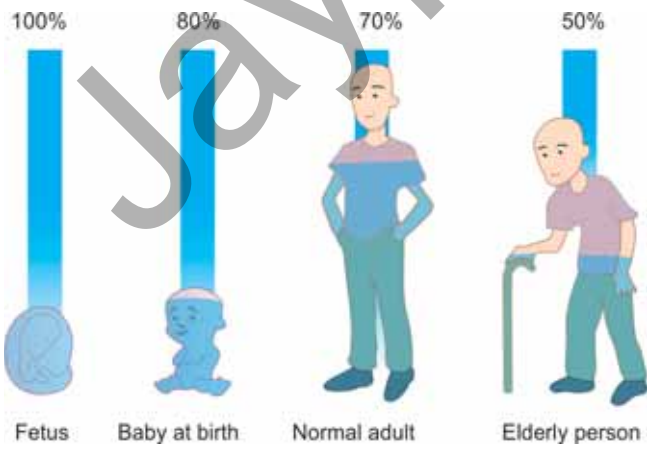


Fig. 13.2: Percentage of water in the human body at different stages of life.

FLUID COMPARTMENTS

A delicate balance exists between fluid in the intravascular fluid compartment and fluid in the interstitial fluid compartment (Fig. 13.3). This balance is maintained by Starling's forces.

Starling's force is defined as the filtration of fluid across the wall of a capillary that is dependent on the balance between hydrostatic pressure and oncotic pressure across the capillary.

The hydrostatic pressure within the vessel pushes fluid into the interstitium at the arteriolar end of the capillaries. At the venous end, the colloid oncotic pressure leads to entry of fluid from the interstitium into the vascular compartment (Fig. 13.4). The net fluid into the interstitial space is drained into the lymphatics.

Disturbances of the equilibrium of Starling's forces result in edema formation.

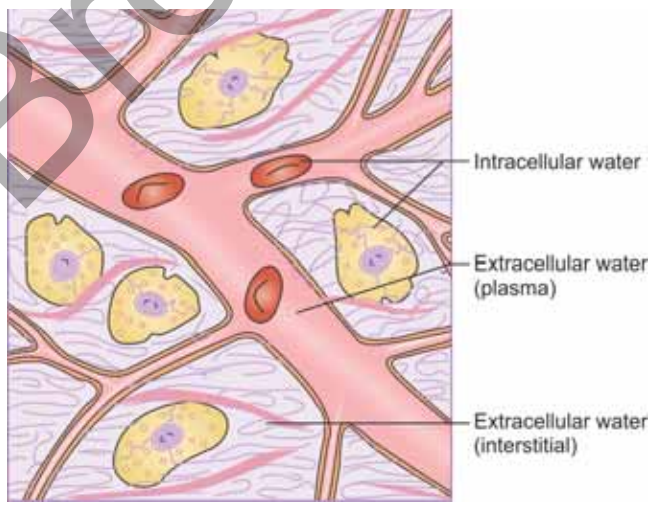


Fig. 13.3: Body fluid compartments (graphical representation).

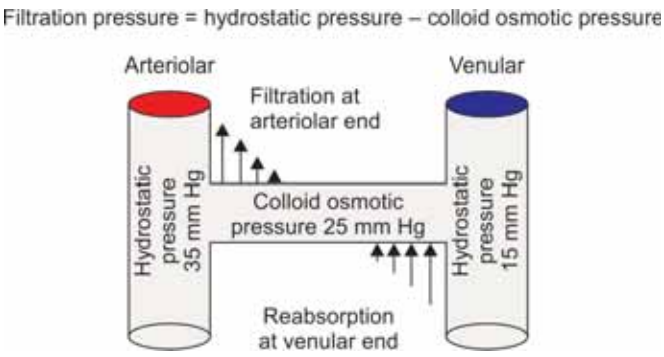


Fig. 13.4: Equilibrium between plasma and interstitial fluid.

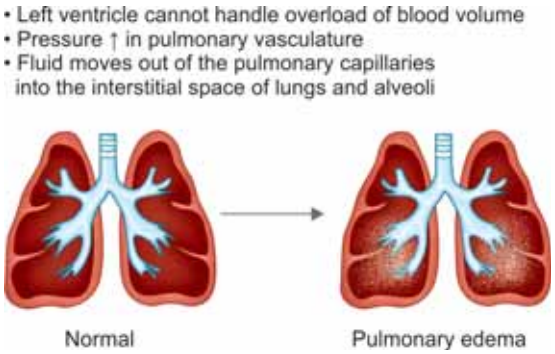


Fig. 13.5: Pulmonary edema.

Myocardial dysfunction leads to increased hydrostatic pressure within the pulmonary capillaries. The latter causes leakage of fluid into the alveoli (pulmonary edema, Fig. 13.5).

Failure to excrete due to acute glomerulonephritis results in increased intravascular volume and increased hydrostatic pressure in the tissues. The latter causes accumulation of fluid in the interstitium and edema.

Albumin contributes to the colloid oncotic pressure of the plasma. Loss of albumin due to nephrotic syndrome, malnutrition or liver failure, leads to retention of fluid within the interstitium thereby causing anasarca.

Increased capillary permeability due to sepsis can also cause interstitial edema.

LOSS OF WATER

The human body tends to lose water via sensible and insensible routes. Insensible water loss occurs by evaporation from the skin and respiratory tract (water evaporates when air passes through the respiratory tract). Water is lost without loss of solutes. The extent of loss via insensible route cannot be evaluated. Loss via urine, stool and sweat is categorized as sensible water loss (Fig. 13.6).

Physiological Water Loss (100 mL/kg)

Insensible water loss:

- Lungs: 15 mL/kg
- Skin: 30 mL/kg.

Sensible water loss:

- Urine: 50 mL/kg (40–70 mL/kg)
- Stool: 5 mL/kg
- Sweat: 0–20 mL/kg.

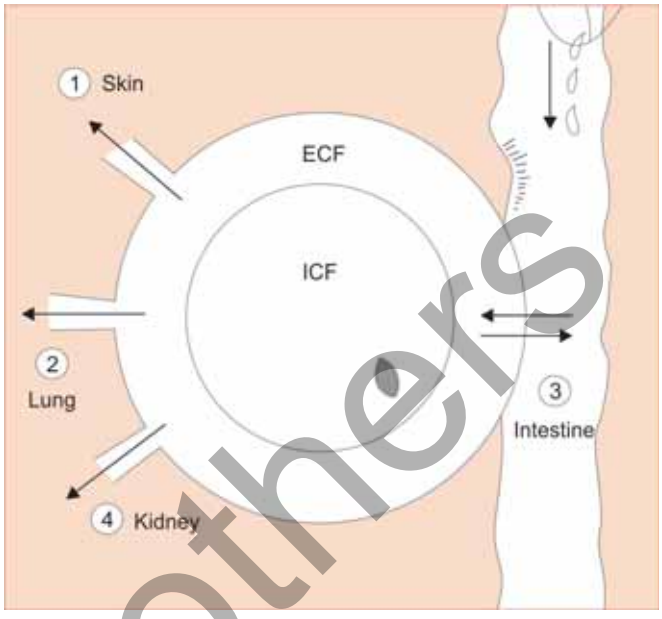


Fig. 13.6: Different routes of physiological water losses. (ICF: intracellular fluid; ECF: extracellular fluid)

Pathological Fluid Loss

Loss of fluids can occur in various conditions. Gastrointestinal losses result from diarrhea and vomiting. Burns causes aggravate loss from skin surface. Polyuria causes dehydration and shock in diabetes. Blood loss occurs in trauma. Capillary leak and vasodilation lead to relative and absolute hypovolemia in severe sepsis (Fig. 13.7 and Flowchart 13.2).

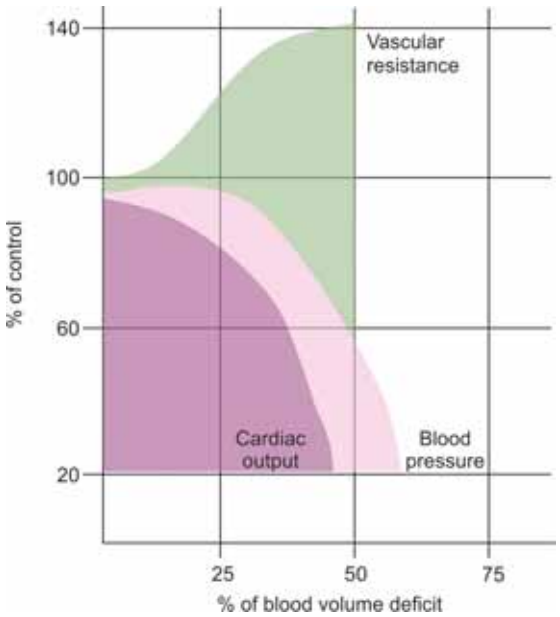
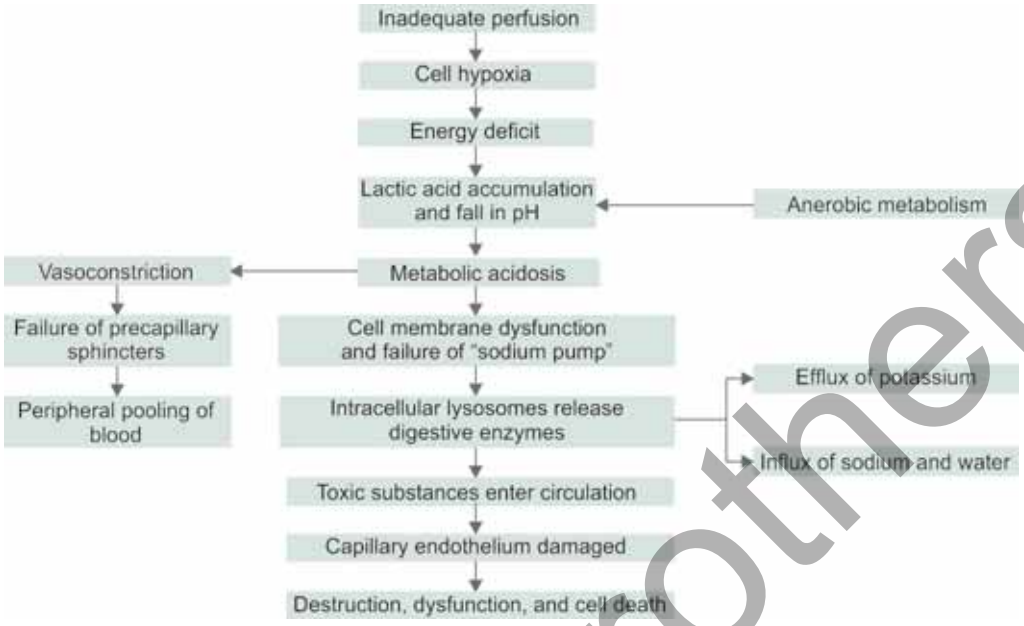


Fig. 13.7: Hypovolemic shock.

Flowchart 13.2: Hemodynamic response to shock.



Shock

Maintenance of the intravascular volume is important for adequate tissue perfusion. Loss of 25% of effective circulating volume results in shock. The initial sympathetic response results in tachycardia and peripheral vasoconstriction, which increases systolic blood pressure (BP). Later as compensation fails, cardiac output drops and BP falls leading to irreversible shock and cardiac arrest.

Dehydration

Dehydration is classified as no dehydration, some dehydration and severe based on clinical signs and percentage of weight loss (Table 13.2).

Table 13.2: Body water compartment affected in various types of dehydration.

Severity/types of dehydration	*Body fluid compartment affected*
• Mild/moderate dehydration	• Loss of fluid from the interstitial compartment
• Severe dehydration	• Plasma volume contracts
• Hyperosmolar dehydration (DKA, hypernatremia)	• Loss of fluid from the intracellular compartment

(DKA: diabetic ketoacidosis)

Some dehydration (fluid deficit equaling 5–10% of body weight): Affected children have any two of the following signs:

- Restless/irritable
- Sunken eyes
- Drinks eagerly/thirsty
- Skin pinch goes back slowly.

Children classified as "some dehydration" have features of "mild" and "moderate" dehydration.

Severe dehydration: Sunken eyes, skin pinch goes back very slowly, and not able to drink.

Severe dehydration occurs when the child with diarrhea losses 10% of effective circulating volume.

ELECTROLYTES

Electrolyte distribution varies. Potassium, an anion, is found predominantly in the ICF along with proteins and phosphates (cations). Sodium and chloride are the predominant anion and cation, respectively in the ECF (Table 13.3). The active extrusion of sodium from cells and influx of potassium into the cells occurs as a result of the Na^+ K^- adenosine triphosphatase (ATPase) pump. The difference between intracellular and extracellular potassium causes greater negativity of the intracellular space relative to the extracellular space.

Serum concentration of an electrolyte may not reflect the total body content. This is especially true for potassium

and phosphorus, which are both found predominantly within the intracellular space.

Table 13.3: Distribution of anions and cations in extracellular fluid (ECF) and intracellular fluid (ICF).

	Electrolytes	*ECF (in mEq/L)*	*ICF (in mEq/L)*
Cations	Sodium (Na^+)	140	10
	Potassium (K^+)	4	158
Anions	Chloride (Cl^-)	103	4
	Bicarbonate (HCO_3^-)	25	10

Intravenous (IV) fluids are administered to maintain the intravascular compartment. Normal saline (NS) and Ringer's lactate (RL) remain within the intravascular compartment since they are isotonic fluids. For this reason, these glucose-free isotonic fluids are preferred in the management of shock.

Definitions

Osmosis is the net movement of water, from an area of lower solute concentration to an area of higher solute concentration across a semipermeable membrane.

Sodium, an impermeable solute, exerts an osmotic force across a semipermeable membrane. Urea, which is permeable, does not exert any osmotic force and hence is defined as "ineffective osmoles".

Osmolality or osmolarity is the measure of osmotically active particles available in a solution per kilogram of solvent.

Tonicity is the ability of ECF, to cause the movement of water into or out of a cell by osmosis. Solutes, that are permeable across the semipermeable membrane, do not contribute to tonicity (Table 13.4).

Osmolality can be calculated by the following formula:
Osmolality =2 (Sodium) + (Glucose/18) + (Urea/6)
e.g.

1. Sodium = 140
   Glucose = 90
   Urea = 30
   Osmolality = 2 × 140 + (90/18) + (30/6)
   = 280 + 5 + 5 = 290 mOsmol/L
2. Sodium = 140
   Glucose = 540
   Urea = 30
   Osmolality = 280 + 30 + 5 = 315 mOsmol/L

Normal plasma osmolality ranges between 285 mOsm/kg and 295 mOsm/kg.

Measured osmolality is around 10 mOsm/kg more than the calculated osmolality. A difference greater than 10 mOsm/kg (osmolal gap) is indicative of unmeasured anions.

Increased osmolal gap due to excess anion, occurs in poisoning due to ethanol, methanol or ethylene glycol.

Regulation

Plasma osmolality is regulated by water whereas intravascular volume is regulated by sodium.

An increase in plasma osmolality sensitizes the osmoreceptors in the hypothalamus which releases

Table 13.4: Osmolality and tonicity of commercially available IV fluids.

Solution	*Osmolality (mOsmol/L)*	*Sodium content (mEq/L)*	*Osmolality (compared to plasma)*	*Tonicity (with reference to cell membrane)*
Sodium chloride 0.9%	308	154	Iso-osmolar	Isotonic
Sodium chloride 0.45%	154	77	Hypo-osmolar	Hypotonic
Sodium chloride 0.45% and glucose 5%	432	75	Hyperosmolar	Hypotonic
Glucose 5%	278	–	Iso-osmolar	Hypotonic
Glucose 10%	555	–	Hyperosmolar	Hypotonic
Sodium chloride 0.9% and glucose 5%	586	150	Hyperosmolar	Isotonic
Sodium chloride 0.18% and glucose 5% (marketed as Isolyte P)	284	31	Iso-osmolar	Hypotonic
Ringer's lactate	278	131	Iso-osmolar	Isotonic

Source: National Patient Safety Agency—NHS United Kingdom. Patient safety alert 22—reducing the risk of hyponatremia when administering intravenous infusions to children 2007.

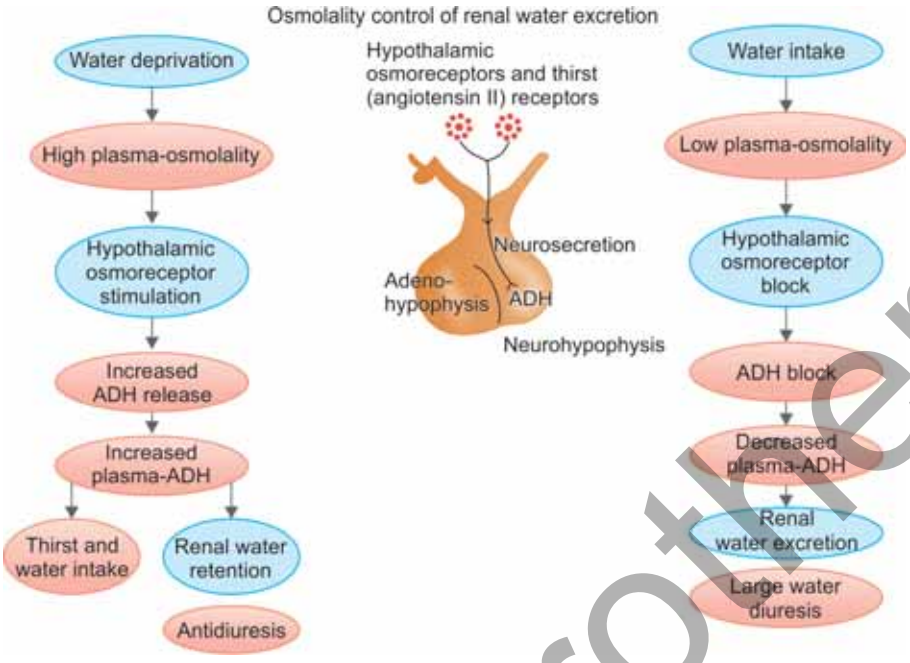


Fig. 13.8: Antidiuretic hormone (ADH) and thirst maintain osmolality within narrow limits.

antidiuretic hormone (ADH). ADH acts on the distal collecting tubule thereby increasing water reabsorption and decreasing water excretion (Fig. 13.8).

Increased plasma osmolality also stimulates the thirst center increasing intake of water. The resultant decrease in plasma osmolality leads to decreased ADH secretion. Less ADH results in diuresis of dilute urine.

However, volume depletion takes precedence over regulation of osmolality.

Hypovolemia stimulates both ADH and thirst, leading to water retention irrespective of the osmolality.

Fluid and Electrolyte Disturbance (Fig. 13.9)

History

Enquire for history of vomiting, diarrhea, polyuria and oliguria.

Examination

Look for signs of dehydration: Anterior fontanelle is depressed in young infants. Sunken eyes, dry tongue, dry mucosa, loss of skin turgor, absence of tears, thirst and oliguria are suggestive of dehydration. Doughy feel

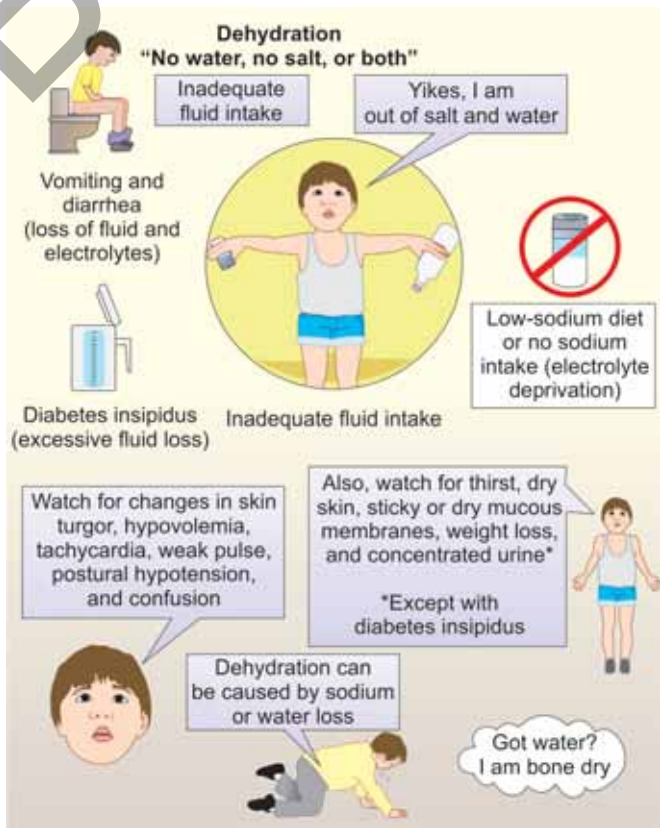


Fig. 13.9: Signs and symptoms of electrolyte imbalance.

of subcutaneous tissue is suggestive of hypernatremic dehydration. Hypotonia, head lag and bradycardia are indicative of coexisting hypokalemia.

Investigations

- *Complete blood count:* Sepsis
- *Serum electrolytes, chloride, bicarbonate:* Electrolyte imbalance
- *Urea, creatinine:* Renal parameters are deranged in prerenal uremia
- *Glucose:* Hypoglycemia complicates dehydration in severe acute malnutrition (SAM) children
- *Calcium:* Hypocalcemia is common in malnourished children presenting with dehydration
- *Electrocardiogram (ECG):* Potassium is lost in diarrhea. Hypokalemia can cause life-threatening bradycardia. Since K^+ is an intracellular ion, serum potassium values do not reflect, the extent of hypokalemia. Hence, ECG monitoring is mandatory during resuscitation of SAM kids with dehydration or hypovolemic shock.

Osmolality and urine analysis are additionally necessary in critically-ill children.

Frequent clinical assessments are essential to evaluate response to fluid therapy. Weight, rapid cardiopulmonary cerebral assessment, input-output chart and laboratory investigations will supplement the clinical assessment.

Intravenous Fluid Therapy

- Isotonic fluid bolus to correct shock, e.g. septic shock, dengue shock
- Deficit replacement, e.g. diarrheal dehydration
- Maintenance fluid: Perioperative IV fluids when nil per oral (NPO) guidelines are being enforced
- Replacement of the ongoing losses: Nasogastric aspirate or drainage from ostomies.

Shock Correction

(Refer to the chapter on "Emergency")

Loss of fluid or blood that exceeds 25% of circulation volume leads to shock.

Effective circulating volume in children: 80 mL/kg body weight

↓

Loss of 25% of 80 mL/kg = 20 mL/kg (the volume of one bolus of normal saline or Ringer's lactate).

Normal plasma osmolarity ranges between 285 mOsmol/L and 295 mOsmol/L. Fluid having osmolarity similar to plasma is termed isotonic fluid (Table 13.5).

Table 13.5: Fluid calculation based on type of shock.

Types of shock	*Clinical condition*	*Volume*	*Time*
Hypovolemic	Diarrhea, vomiting, acute blood loss	20 mL/kg	20 min
Distributive shock (septic shock, anaphylactic shock, spinal shock)	Infection (pneumonia, abscess), drug/vaccine induced Spinal cord injury	20 mL/kg	20 min
Cardiogenic shock	Myocarditis, congenital heart disease	5–10 mL/kg	20 min
Dengue shock	Severe dengue	10 mL/kg	1 hour

Dehydration

Fluids calculation is based on severity of dehydration, maintenance, requirement and deficiency (Table 13.6).

Table 13.6: Quantity of fluid loss based on severity of dehydration.

Mild	*Moderate*	*Severe*
30–40 mL/kg	50–70 mL/kg	100 mL/kg

Mild dehydration for 10 kg child:
Oral rehydration salt (ORS) is preferred, unless the child has persistent vomiting or refuses ORS.

Correction of dehydration:
- *0–6 hours:* NS/RL 40 mL/kg (400 mL) for deficit
- *7–24 hours:* Maintenance 100 mL/kg/24 hours (1,000 mL).

Moderate dehydration:
Fluid deficit is corrected in three phases:
1. 0–1 hour: Rapid restoration of intravascular volume
   - 20 mL/kg NS/RL is given over 1 hour (200 mL)
2. 1–6 hour: Deficit correction
   - 50 mL/kg NS/RL (500 mL)
3. 7–24 hours: Maintenance fluid + replacement of ongoing losses
   - 100 mL/kg of ½ GNS + KCL at the rate of 20 mEq/1,000 mL.

Integrated Management of Neonatal and Childhood Illnesses—Management of Dehydration

Some dehydration: Loss of fluid up to 5–10% of body weight ("mild and moderate" dehydration).

Treatment:

- ORS solution at the rate of 75 mL/kg over 4 hours (Plan B)
- Further management is based on reassessment after 4 hours
- Feeding is continued.

Severe dehydration is corrected with intravenous fluids (Table 13.7).

Table 13.7: Severe dehydration correction: for a child weighing 10 kg.

	WHO criteria	
	30 mL/kg (300 mL)	70 mL/kg (700 mL)
Infant (<1 year)	0–1 hour	1–6 hours
>1 year	0–1/2 hours	½–3 hours

Maintenance Fluids

Dehydration, starvation ketosis and protein degradation require IV fluids to avoid electrolyte disorders (Table 13.8).

Composition of Maintenance Fluid

Commercially available maintenance fluid comprises of water, glucose, sodium and potassium. Glucose provides 20% of the total caloric requirement, thereby reducing risk of starvation ketosis and protein degradation. It also increases osmolarity. Sodium and potassium have been included to replace daily losses. Calcium bicarbonate and phosphate are not indispensable and hence not included. Ideally, IV maintenance fluids should be cheap, easily available, have a long shelf life and avoid causing complications. A child who is exclusively on maintenance fluids will lose around 0.5–1% weight everyday. Table 13.8 shows the rate of calculation of maintenance fluid.

Table 13.8: Maintenance fluid (water) requirement.

Body weight in kg	*Volume per 24 hours*	*Volume per hour*
First 10 kg	100 mL/kg	4 mL/kg/hour
11–20 kg	1,000 mL plus 50 mL/kg for each 1 kg >10	40 mL/hour plus 2 mL/kg for each 1 kg>10
21 kg	1,500 mL plus 20 mL/kg for each 1 kg >20	60 mL/hour plus 1 mL/kg/hour for each 1 kg >20
For example: Weight 10 kg: 40 mL/hr; 15 kg: 40 + 10 = 50 mL/hr; 25 kg: 40 + 20 + 5 = 65 mL/hr		

Hyponatremia occurs with the use of hypotonic maintenance fluids. Critically-ill children are prone for syndrome of inappropriate ADH secretion that is aggravated by use of hypotonic solution. The resultant hyponatremia causes encephalopathy caused by influx of water into the intracellular space leading to cerebral edema, seizures and brainstem herniation.

Hyponatremic encephalopathy thus is often an iatrogenic complication due to administration of hypotonic maintenance fluids!

Half NS 0.45% with 5% dextrose is a safe option for maintenance fluid. If risk of nonosmotic ADH secretion exists, dextrose normal saline (DNS) is used as maintenance fluid. Normal saline or RL are ideal during surgical and postoperative period.

If, excess ADH secretion is anticipated, maintenance fluid is restricted to two-thirds of normal recommended volume.

Caution

If potassium is needed 5 mL of KCl = 10 mEq/L is added to 500 mL of G5 ½ NS. The fluid bag is labeled and mixed thoroughly prior to usage.

Potassium-rich fluid is administered, after ensuring that the child has voided urine.

During fluid administration the following care is provided:

- Prescription, should include, type of fluid, volume, duration and rate of flow. The Holliday-Segar formula and rate of flow per hour are given in Table 13.8.
- The rapid cardiopulmonary cerebral assessment is documented serially.
- Weight and urine output are noted.
- Laboratory data includes daily report of serum electrolytes, urea, creatinine and hematocrit (HCT).

HYPONATREMIA

Definition

Normal range of serum sodium is between 135 mEq/L and 145 mEq/L (Fig. 13.10 and Flowchart 13.3).

Serum sodium level less than 135 mEq/L is defined as hyponatremia.

Causes

Hyponatremia can occur due to pseudohyponatremia or true hyponatremia.

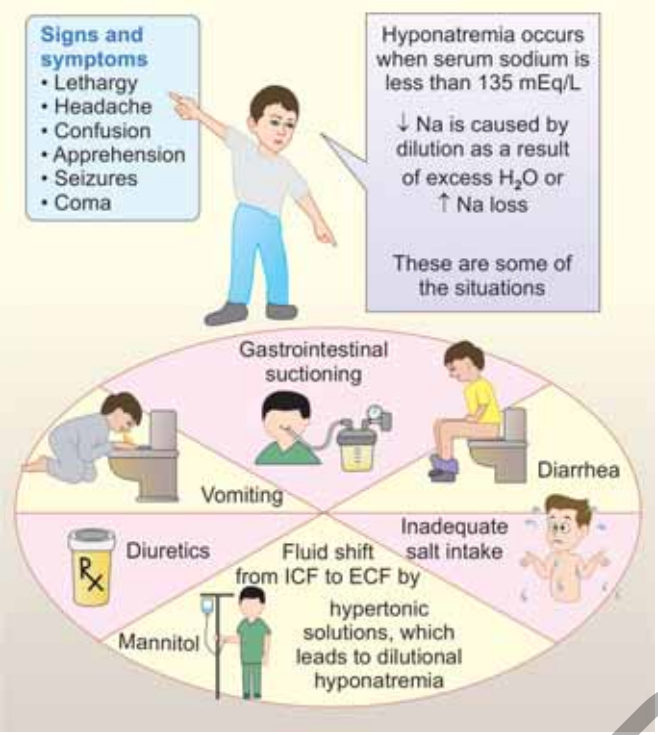


Fig. 13.10: Signs and symptoms of hyponatremia.

True hyponatremia is associated with low measured osmolality.

Pseudohyponatremia

If serum osmolality is normal or high, pseudohyponatremia is a possibility.

- Blood sample, taken from the vein proximal to point of entry of an IV infusate through which hypotonic fluid is being administered.
- Laboratory artifact that is noted in hyperlipidemia or hyperproteinemia. Normal osmolality, rules out true hyponatremia.
- Hyponatremia has also been noted in hyperglycemia and mannitol therapy. High osmolality causes movement of water into the vascular compartment resulting in hyponatremia.

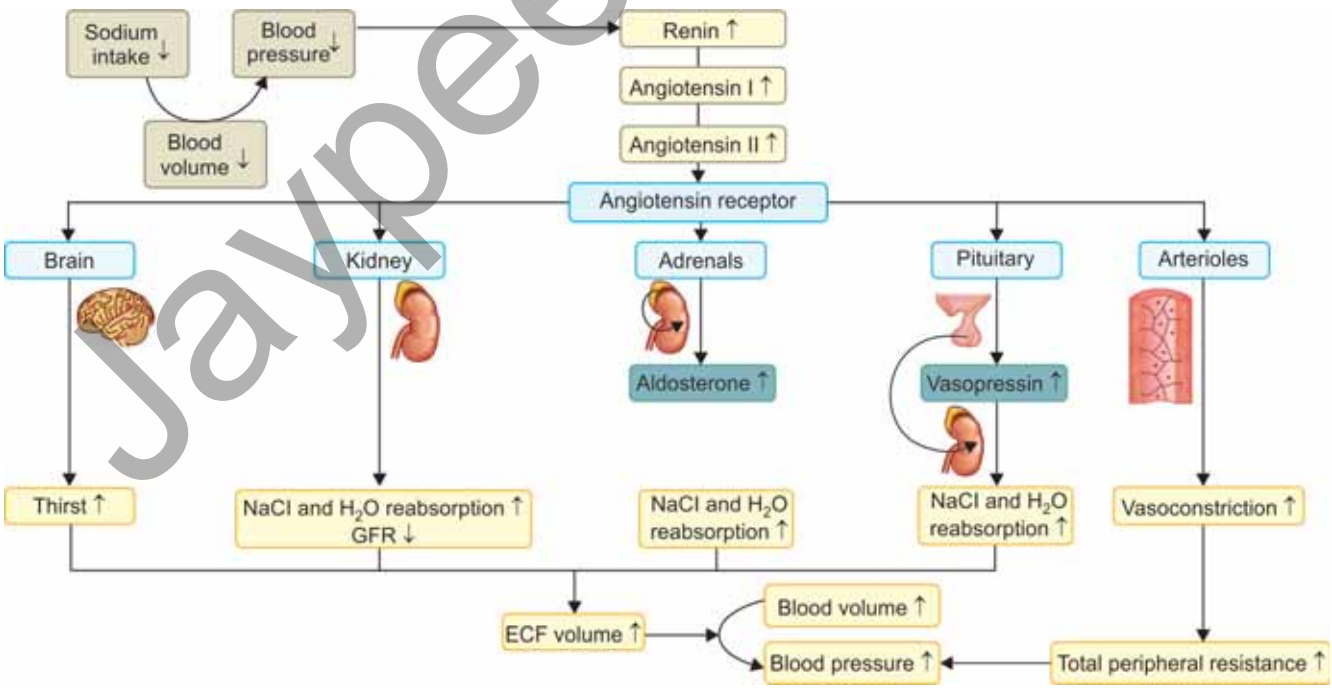
The causes of true hyponatremia are shown in Table 13.9.

Since sodium concentration depends on water balance, hyponatremia is categorized as hypovolemic hyponatremia, hypervolemic hyponatremia and euvolemic hyponatremia.

Pathophysiology

The decrease in extracellular osmolality causes water to down the osmotic gradient into the intracellular space where osmolality is higher.

Flowchart 13.3: Hormonal control of sodium intake.



(ECF: extracellular fluid; GFR: glomerular filtration rate)

Table 13.9: Causes of hyponatremia.

Hypovolemic hyponatremia	*Euvolemic hyponatremia*	*Hypervolemic hyponatremia*
Water ↓ and Na ↓↓	*Water excess*	*Water ↑↑ and Na ↑*
Causes: • Extrarenal loss (urine Na <20 mEq/L)—vomiting, diarrhea, third spacing • Renal loss (urine Na >20 mEq/L)—RTA, cerebral salt wasting, osmotic diuresis, DKA, diuretic therapy. Adrenal insufficiency, pseudohypoaldosteronism	*Causes:* • Water intoxication: use of 5% dextrose in postoperative period, psychogenic water drinking, tap water edema • SIADH	*Causes* • Renal failure (urine Na >40 mEq/L) • Nephrotic syndrome • Congestive heart failure • Protein-energy malnutrition • Cirrhosis of liver

(RTA: renal tubular acidosis; DKA: diabetic ketoacidosis; SIADH: syndrome of inappropriate antidiuretic hormone secretion)

Entry of water into the cells of the brain from the ECF, results in cerebral edema. Since, hyponatremia develops gradually, brain cells adapt by extruding intracellular potassium, chloride and a variety of small organic molecules thereby reducing intracellular osmolality.

Clinical Features

Severity of symptoms is dependent on the magnitude and rapidity of hyponatremia. Apathy, anorexia, nausea and vomiting occur when sodium drops to less than 130 mEq/L.

Sodium less than 120 mEq/L leads to muscular twitching, headache, coma and seizures.

Physical Examination

Evaluate for the following:

- Signs of dehydration, coma, seizures
- Pigmentation, stigmata of liver or renal disease, rickets.

Investigations (Flowchart 13.4)

- Serum electrolytes, glucose, urea, creatinine, chloride, X-ray chest
- Serum osmolality, urine osmolality and urine sodium
  - Urine Na <20 mEq/L indicates extrarenal loss
  - Urine Na >20 mEq/L indicates renal loss.

Management (Flowchart 13.5)

- It depends upon the hydration status
- Presence of neurological symptoms
- Duration of the problem (acute < 48 hr or chronic > 48 hr).

Symptomatic Hyponatremia: Low Sodium associated with Altered Mental Status or Convulsions

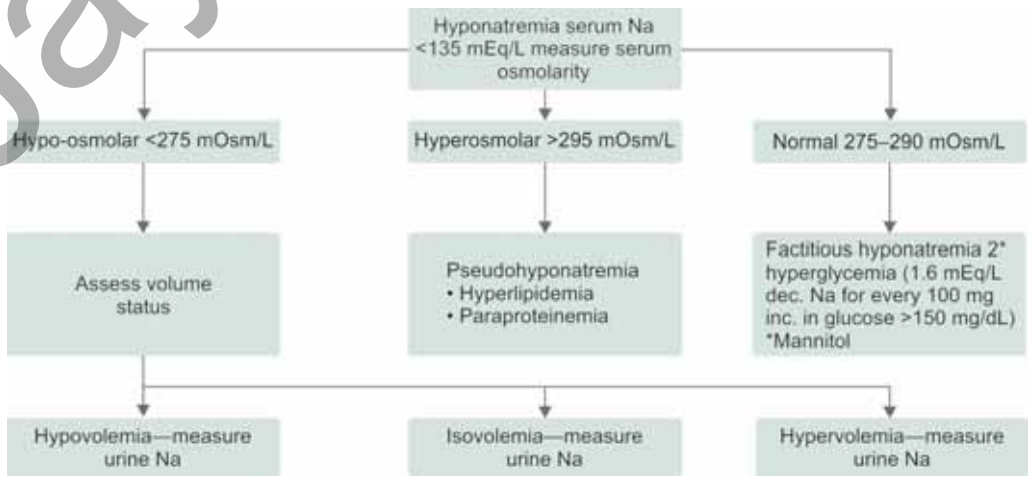
Administration of 3% saline at the rate of 5 mL/kg over 30–60 minutes. This strategy increases serum sodium by 5 mEq/L and resolves symptoms.

Asymptomatic Hyponatremia

- Cerebral edema is less severe.

Rapid treatment can cause central pontine myelinolysis or even death.

Flowchart 13.4: Diagnostic algorithm for hyponatremia.



- Hence, correction is gradual with rise in sodium at the rate of 0.5 mEq/hr or 12 mEq/day.

The sodium correction is determined as follows:

Sodium deficit (mEq) = (Desired Na^+ – Measured Sodium) × Weight (kg) × 0.6.

For example, desired sodium: 125 mEq/L, measured sodium: 120 mEq/L, weight: 10 kg sodium deficit: 5 × 10 × 0.6 = 30 mEq. 30 mEq is administered over 48 hours.

Hypovolemic Hyponatremia

Water and sodium are replaced using isotonic fluid viz. NS based on the severity of dehydration (Flowchart 13.5)).

Isovolemic Hyponatremia

Restrict NS to two-thirds maintenance.

Hypervolemic Hyponatremia

Sodium and water are restricted.

- Hyponatremia is clinically associated with syndrome of inappropriate ADH secretion (SIADH).

Causes

Meningitis, head trauma, spinal or intracranial surgery, near fatal asthma, pneumonia, tuberculosis, carbamazepine and vincristine cyclophosphamide.

Diagnostic criteria for SIADH

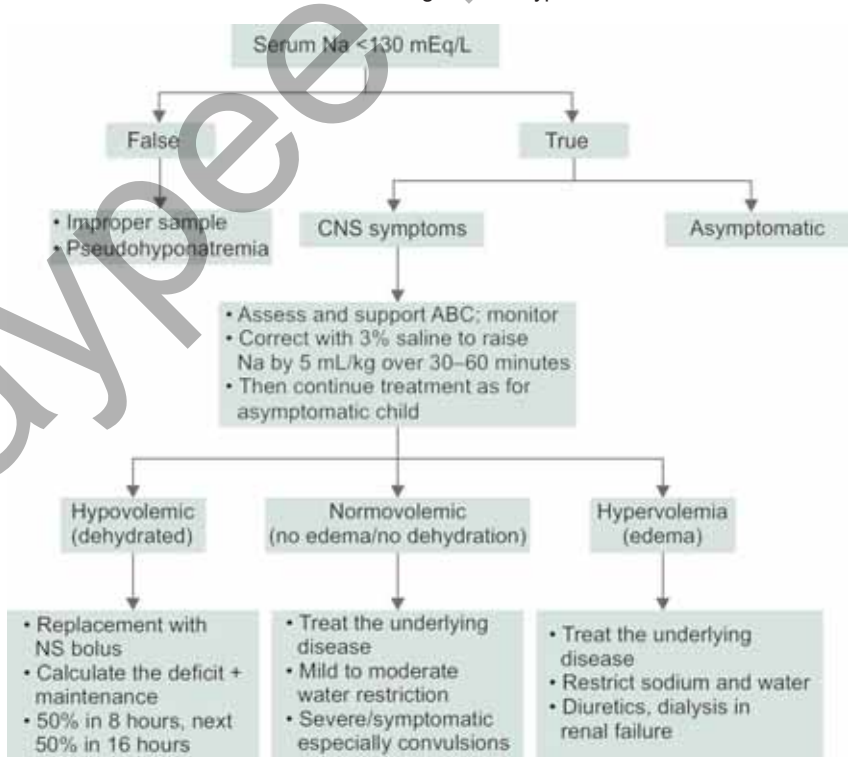
- Hyponatremia
- No dehydration, no edema
- Normal renal, hepatic, adrenal and thyroid function
- Urine osmolality >100 mOsm (urine is concentrated more than serum osmolality)
- Urine sodium is increased (20–40 mEq/L).

Hyponatremia with seizures: Restrict fluids, 3% saline and diuretics.

Asymptomatic hyponatremia: Restrict fluid.

- *Hyponatremia, seizures with shock:* Correction of shock with RL or NS corrects sodium deficit.
- *Hyponatremia, seizures with renal failure:* Dialysis and 3% saline.

Flowchart 13.5: Management of hyponatremia.



(ABC: airway-breathing-circulation; NS: normal saline)

- *Hyponatremia with adrenal insufficiency:* Hydrocortisone.

HYPERNATREMIA

A sodium concentration more than 145 mmol/L is defined as hypernatremia. Hypernatremia is relatively less common than hyponatremia.

Causes

Osmolality and serum sodium are maintained at optimum level by two mechanisms: (1) thirst and (2) vasopressin. Young infants who cannot express thirst, have access to water or have depressed level of consciousness are vulnerable. Hypernatremia occurs as result of water deficit in relation to body's sodium content. Diabetes insipidus (DI), that characterizes this condition, results disproportionate loss of fluids in comparison to sodium. It can also occur when excessive sodium has been ingested as seen in salt poisoning (rare).

- *Hypotonic fluid loss (disproportionately more water is lost than electrolytes):*
  - Vomiting—water loss is associated with reduced intake of water
  - Diarrhea, in association with obstructive uropathy or renal tubular dysfunction.
- *Sodium excess:*
  - Concentrated formula feeds or oral rehydration solution
  - Salt poisoning—unintentional, addition of salt instead of sugar
  - Mineralocorticoid excess—hyperaldosteronism.
- *Electrolyte free water loss:* DI, reduced access to water, mentally retarded children, depressed level of consciousness or defective thirst mechanism. Hypernatremia in the neonate is a marker of either, reduced breast milk or lactation failure.

Pathophysiology

Increased sodium content in the ECF compartment leads to movement of water from ICF. The resultant alteration in intracellular tonicity and osmolality in the neuronal cell leads to shrinking of cell or cellular dehydration. The brain is most vulnerable to changes in sodium concentration. Hypernatremia leads to disturbances of consciousness. Rarely, it can cause tearing of the blood vessels and intracranial bleed.

Neurons, however, have an inherent capacity to reduce cell shrinkage. They produce osmotically active substances called "idiogenic" osmoles. Currently, these moieties have been identified as taurine and other amino acids. This protective mechanism, takes time to evolve. Hence treatment guidelines advise gradual reduction or elevation of serum sodium (not more than 0.5 mmol/hour).

Rapid reduction in serum sodium in the presence of undisposed idiogenic osmoles can cause reversal of movement of water into the cells from the ECF resulting in cerebral edema during therapy.

Clinical Features

Central nervous system: Lethargy, restlessness, high pitched cry, features of intracranial bleed, and convulsion and loss of consciousness.

Volume status: Signs of dehydration are *not* evident. Skin may feel doughy.

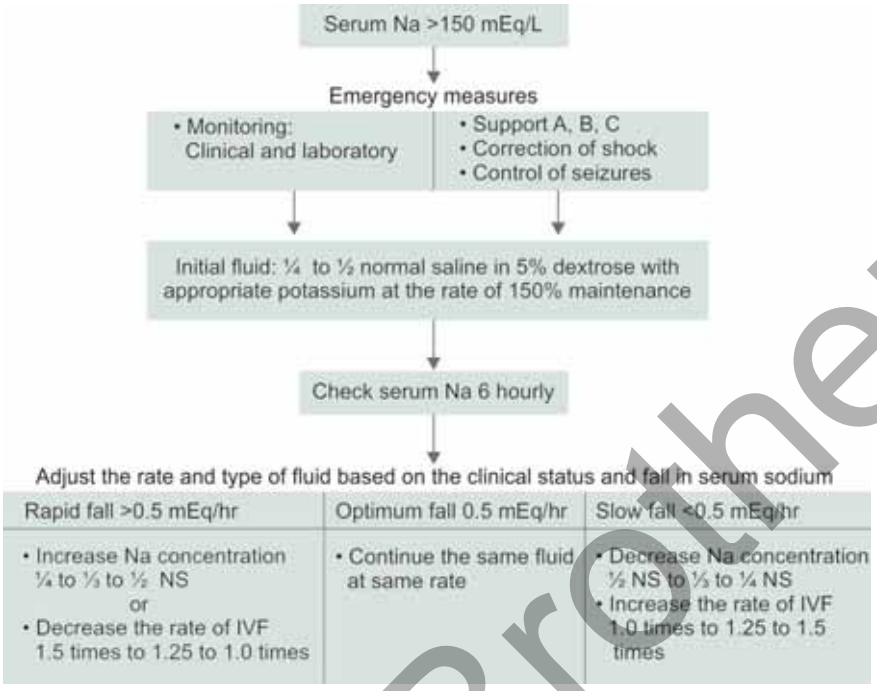
Laboratory Investigations

- Hypocalcemia and hyperglycemia are two common problems
- Neuroimaging is indicated to find out whether the child has developed intracranial hemorrhage. It is also performed to exclude other causes of altered consciousness and seizures.

Management

- Seizures control (refer to chapter on "Emergency").
- Correction of shock if identified. RL, is avoided since it is relatively hypo-osmolar in comparison to NS.
- After the correction of shock, hypernatremia is treated with half NS at a rate of 25–50% more than the maintenance rate (deficit correction + maintenance).
- Estimation of serum sodium every 4–6 hourly is vital in titrating fluid rate or sodium concentration of the IV fluid.
- *Treat the underlying cause:* Treat DI, salt poisoning, coexisting hypocalcemia. Hyperglycemia does not require correction.
- If seizures occur during treatment of hypernatremia, 3% NaCl is infused at the rate of 5 mL/kg over 1 hour. This will raise sodium by 5 mEq/L.

Flowchart 13.6: Approach to hypernatremia.



Management of Hypernatremia with Edema and Salt Excess (Flowchart 13.6)

Salt poisoning leads to hypervolemic hypernatremia. Diuretics and replacement of urine output with hypotonic fluid such as ¼ NS, is curative. If renal failure exists, dialysis is necessary.

HYPOKALEMIA

Definition

Normal serum level is 3.5–4.5 mEq/L (Fig. 13.11).

Serum potassium level less than 3.5 mEq/L is defined as hypokalemia.

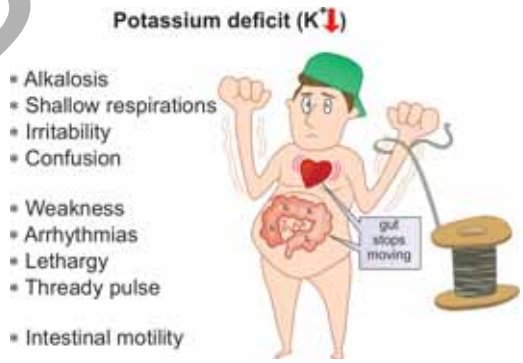
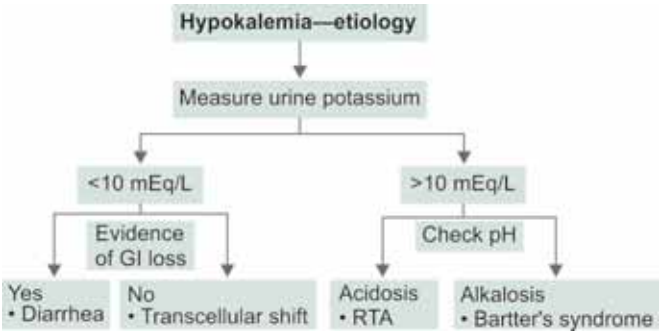


Fig. 13.11: Signs and symptoms of hypokalemia.

Causes (Flowchart 13.7)

1. *Reduced intake:* Malnutrition and potassium free intravenous fluid.
2. *Gastrointestinal loss:* Vomiting, diarrhea and laxative abuse.
3. *Renal loss:*
   - Metabolic acidosis: Renal tubular acidosis
   - Metabolic alkalosis and normal BP—Bartter's syndrome
   - Metabolic alkalosis and normal or low BP—diuretic therapy
   - Metabolic alkalosis and hypertension—mineralocorticoid excess
4. *Transcellular shifts:* Shift of potassium from ECF to ICF:
   - Alkalosis
   - Insulin treatment of DKA
   - β2 agonist in the treatment of asthma
   - Familial hypokalemic periodic paralysis
   - Rodenticide poisoning.

Flowchart 13.7: Causes of hypokalemia.



(RTA: renal tubular acidosis; GI: gastrointestinal)

Clinical Features

- *Mild hypokalemia:* Fatigue and myalgia, abdominal distention due to paralytic ileus and phantom hernia.
- *Severe hypokalemia:* Weakness of skeletal and smooth muscle function leading to hypotonia, head lag and frog leg posturing and rarely respiratory paralysis.
- *Severe life-threatening hypokalemia:* Bradycardia and cardiac arrhythmias.
- *Persistent hypokalemia:* Alkalosis is caused by increased excretion of chloride.

Investigations

- Many biochemical derangements alter potassium balance. Hence investigations must include serum electrolytes, chloride, sugar, urea, creatinine, urine electrolytes, arterial blood gas (ABG), anion gap estimation and ultrasonography (USG) of abdomen.
  - Urine potassium level less than 20 mEq/L: Nonrenal loss
  - Urine potassium level higher than 40 mEq/L: Renal loss.
- ECG changes: Prolongation of PR interval, reduction in T wave amplitude or flattening or inversion, ST depression and appearance of U wave (Fig. 13.12).

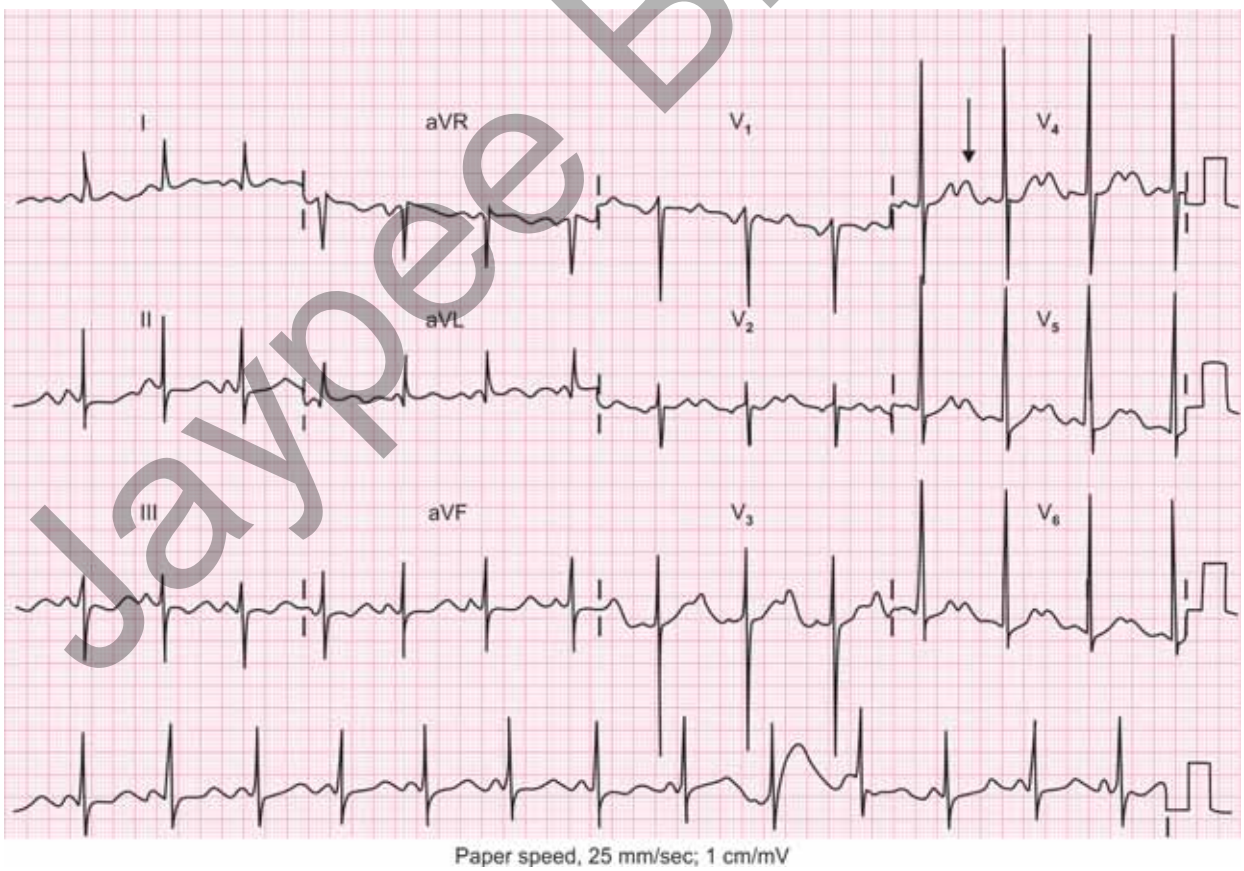


Fig. 13.12: Electrocardiogram (ECG) changes in hypokalemia (arrow indicates U wave).

Differential Diagnosis

Conditions that mimic hypokalemia include acute flaccid paralysis such as Guillain-Barre syndrome.

Hypokalemia never occurs alone and is always associated with a systemic illness.

Treatment

Severe hypokalemia: Serum level <2.5 mEq/L. Associated with paralysis and cardiac arrhythmia

- Rapid correction by infusing potassium (0.3–0.5 mEq/kg/h) over a period of 2–3 hours under cardiorespiratory monitoring.
- Potassium is diluted in NS.
- Hyperkalemia can complicate therapy.

Moderate hypokalemia: Serum level ranges between 2.5 mEq/L and 3.0 mEq/L. No cardiac arrhythmia or bradycardia or paralysis

- Potassium is added to the maintenance fluid. It is increased to 40 mEq/L.
- Serum potassium estimation is repeated after 8–12 hours.
- Addition of 5 mL of potassium chloride to 500 mL of maintenance fluid (D5 ½ NS) will result in a concentration of 20 mEq/L of potassium. Addition of 10 mL will increase potassium content to 40 mEq/L.
- Serum levels are verified every 12 hours and replacement is titrated.

Mild hypokalemia: Serum level ranges between 3.0 mEq/L and less than 3.5 mEq/L. No cardiac arrhythmia or paralysis

- Oral potassium chloride solution or dietary supplements such as orange juice or coconut water is advised. If dietary supplements fail to hypokalemia, *is corrected using* oral potassium chloride solution. Standard oral KCl solution 15 mL contains 20 mEq of potassium.
- Potassium citrate solution is used when acidosis is associated with hypokalemia.

General Principles

- Oral replacement is recommended in the absence of paralysis or arrhythmia.
- No formula is available to calculate the potassium replacement. Total concentration should not exceed 40 mEq/L to avoid the risk of phlebitis and pain. Higher concentration is infused through the central venous catheter under ECG monitoring.
- Potassium solutions cannot be given rapidly. It is administered as a dilute solution preferably in saline.

Prior to infusing potassium, urine output and renal function should be checked.

- If possible, offending drug should be stopped if the primary illness permits.
- The primary disease is treated concurrently (control of diarrhea, treatment of RTA or Bartter's syndrome).

Once the serum level is increased to more than 3.0, and the child can retain oral intake, oral supplementation can be initiated.

Potassium Preparations

- Injection potassium chloride 15% 10 mL = 1.5 g KCl = 20 mEq; 1 mL= 2 mEq.
- *Syrup Potchlor*

  Potassium chloride solution: 5 mL= 1.33 mEq/mL.

HYPERKALEMIA (FIG. 13.13)

Potassium plays a major role in regulating electrical activity (Fig. 13.14).

Potassium and sodium are needed to maintain the membrane potential across the cell membrane (Fig. 13.15).

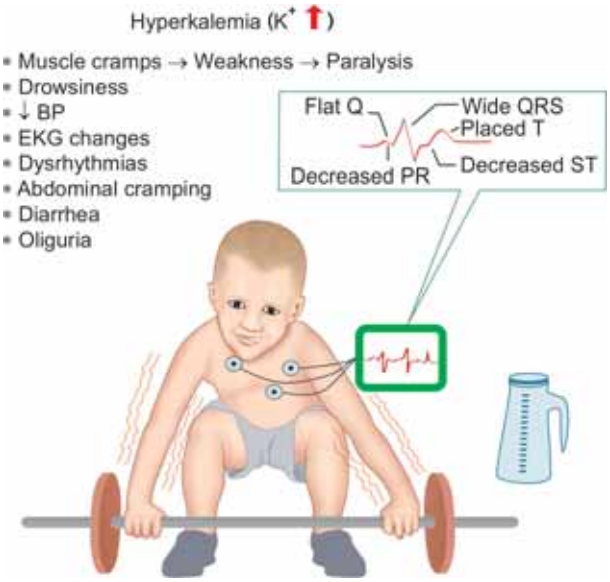
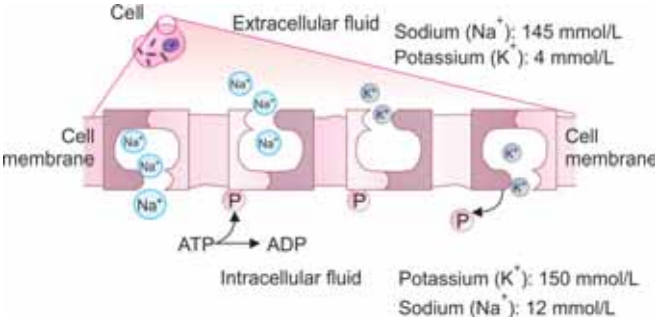


Fig. 13.13: Signs and symptoms of hyperkalemia.



The concentration differences between K^+ and Na^+ across cell membranes create an electrochemical gradient known as the membrane potential. Adenosine triphosphate (ATP) maintaining the membrane potential.

Fig. 13.14: A simplified model of the sodium (Na^+)-potassium (K^+) ATPase pump.

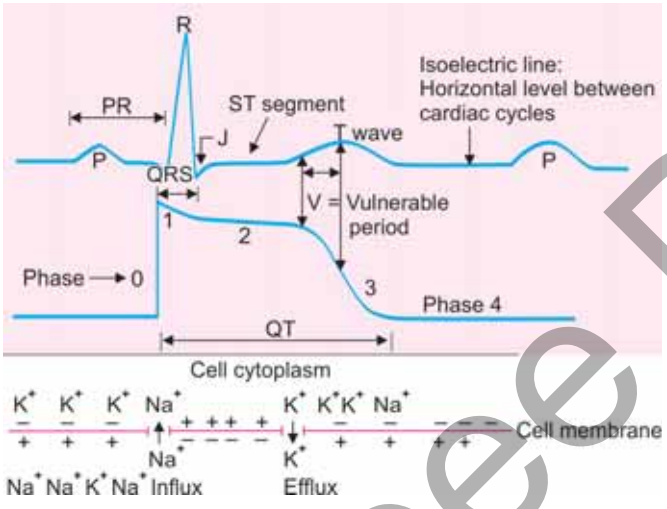


Fig. 13.15: Sodium influx, potassium efflux, the action potential and the electrocardiogram.

Disturbances in potassium content can cause life-threatening arrhythmias.

The normal range of potassium is 3.5–5 mEq/L.

Serum potassium level greater than 5.5 mEq/L is termed as hyperkalemia.

Causes (Fig. 13.16)

Spurious Hyperkalemia

Spurious (false) hyperkalemia occur when the collected blood sample undergoes hemolysis. Since potassium is the predominant cation within the ICF compartment (cells), lysis of the cells leads to release of potassium.

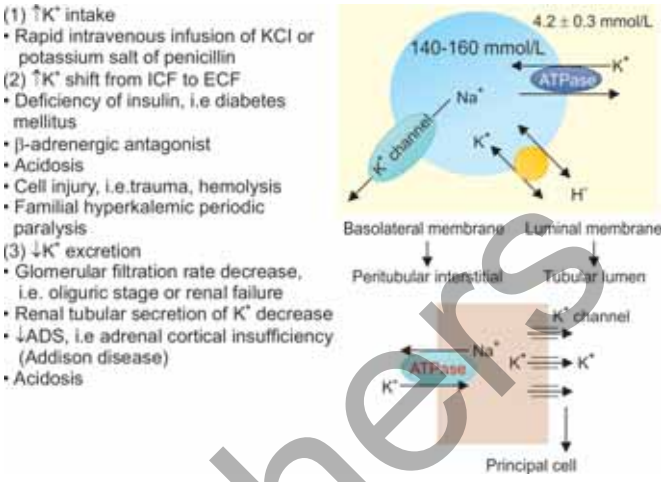


Fig. 13.16: Hyperkalemia—etiology and pathogenesis.

Alteration in potassium levels causes paralysis of skeletal muscles, paralytic ileus and cardiac arrhythmias!

These are usually nonspecific, comprising of muscle weakness, tetany, and paresthesia and ascending paralysis. Serious cardiac toxicity can occur even before the appearance of these nonspecific symptoms.

Investigation and Monitoring

Serum electrolytes, ABG, urea, creatinine are necessary to evaluate cause of hyperkalemia. However, investigation reports should not delay the initiation of therapy. ECG monitoring is mandatory till serum potassium decreases to safer levels.

ECG changes: Peaked T wave, prolonged P-R interval, ST segment depression and wide QRS complex (Fig. 13.17 and Table 13.10).

Therapy

Serum potassium level more than 5.5 mEq/L is a medical emergency. Treatment is instituted without delay.

Intravenous Calcium Gluconate

0.5–1.0 mL/kg of calcium gluconate, is diluted with equal quantity of 5% dextrose and given as a slow IV for over 10 minutes. Cardiac monitoring is essential during and after the infusion. The infusion is stopped if bradycardia

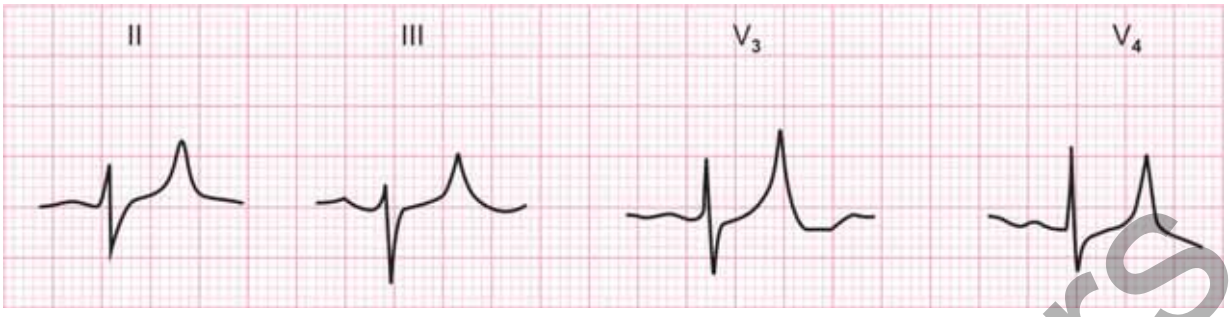


Fig. 13.17: Electrocardiogram (ECG) changes in hyperkalemia.

is noted. Although, calcium therapy does not alter serum "K" level, it acts by protecting the myocardium from the toxic effects of hyperkalemia.

Intravenous Sodium Bicarbonate (8.4%)

Sodium bicarbonate (8.4%), 1–2 mEq/kg is diluted with equal volume of 5% GDW and administered over 10 minutes. To avoid precipitation the IV site is flushed between the calcium and bicarbonate infusion.

Nebulized Salbutamol

This is given in the usual dose as for asthma and can be repeated hourly. Salbutamol respiratory solution 1.25 mg for less than 1 year, 2.5 mg between 1 year and 5 years and 5 mg above 5 years.

Insulin and Dextrose

The combination of insulin and glucose works within 30 minutes. The addition of six units of short-acting insulin to 100 mL of 25% dextrose is infused at the rate of 2 mL/kg as a slow IV over 1 hour. Blood sugar is monitored.

Furosemide

It is administered, at the dose of 1–2 mg/kg IV, provided renal function is normal and perfusion is adequate.

Kayexalate

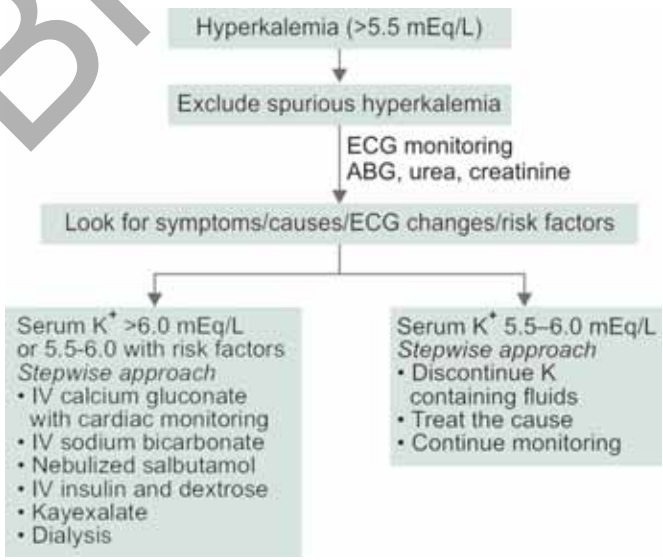
Kayexalate (1 g/kg/dose) is given either orally or rectally. The onset of action takes several hours to take effect. It may be repeated. Although, it is considered very useful in the management of hyperkalemia, it is expensive and carries the risk of sodium overload.

Dialysis, if potassium levels rise rapidly despite these measure, dialysis is an option. The latter is particularly useful if renal failure is causative.

If hyperkalemia is secondary to adrenal insufficiency, hydrocortisone 10 mg/kg IV is initiated.

Management (Flowchart 13.8)

Flowchart 13.8: Approach to hyperkalemia.



HYPOCALCEMIA

Definition

Normal serum calcium level is 9–11 mg/dL. Total calcium less than 7.5 mg/dL or ionized calcium level less than 4 mg or 1 mmol/L needs therapy. Hypoalbuminemia, may lead to reduction in total calcium levels, however, ionized calcium level may be normal. Hence measuring ionized calcium is important in such states. Ionized calcium may also be low in alkalosis.

Causes

Neonatal Period

Early onset (within first 3 days of life): Prematurity, infant of diabetic mothers.

Late onset: At the end of first week of life.
High phosphate intake due to undiluted cow milk feeding, hypomagnesemia, and maternal vitamin D deficiency states are causative.

Children

- Infants: DiGeorge syndrome, maternal vitamin D deficiency, cow milk feeding, magnesium deficiency
- Hypoparathyroidism
- Pseudohypoparathyroidism
- Renal failure
- Vitamin D-dependent rickets type 1
- IV bicarbonate therapy/citrate products
- Acute pancreatitis.

Clinical Features

Muscular pain and cramps progress to numbness and tickling sensation in the hands and feet. A positive Chvostek's or Trousseau's sign, laryngeal and carpopedal spasms are other classical signs of hypocalcemia. Neonates, manifest with jitteriness, multifocal clonic convulsions and rarely ECG abnormalities, dysrhythmias or heart failure (Table 13.10). Long standing hypocalcemia can manifest with late and irregular teeth eruption.

Table 13.10: Electrocardiogram (ECG) manifestation of electrolyte imbalances.

PR interval	*Short*	*Prolonged*
	(Think pre-excitation syndromes such as Wolff-Parkinson-White)	High K Low Ca
QRS duration	Narrow	Wide (>100 msec)
	Low K Low Ca Normal	High K High Ca
QTc interval	Short (<350 msec)	Prolonged (>440 msec)
	High Ca	Low K Low Ca
ST segment	Depressed	Elevated
	Low K High Ca	High K
T wave	Peaked/tall	Flattened
	High K	Low K
U wave	Absent	Present
	Normal	Low K Low Ca
Heart rate	Slow (Bradyarrhythmias, nodal block)	Fast (tachydysrhythmia)
	High K High Ca	Low K Low Ca
	Low	High
Ca	• QTc prolonged (hallmark) • U wave • Heart blocks, ventricular dysrhythmias, torsades de pointes	• QTc shortened (hallmark) • ST segment depression and shortening • QRS widening • Rare: Bradyarrhythmias, bundle branch blocks, high degree AV blocks
K	Early to late findings: • T wave: Decreased amplitude • T wave: Flat or inverted • ST segment depression • U wave • QTc prolonged (at risk for VT or torsades de pointes)	Early to late findings: • T wave: tall, then "peaked" (symmetrical) • P wave flattening • PR interval prolonged • QRS widening • Nodal blocks, escape beats • Sine wave: Fusion of QRS and T wave → VF or asystole

Mg derangements: Nonspecific ECG findings; often coexist with Ca derangements. Classic teaching: Low Mg level → QTc prolongation → torsades de pointes.

Treatment (Fig. 13.18 and Flowchart 13.9)

- Asymptomatic: Oral calcium is prescribed at the dose of 50 mg/kg day.
- Symptomatic hypocalcemia: Calcium is given intravenously in the dose of 1 mL/kg of calcium gluconate as a slow infusion over 30 minutes.
  - *Caution:*
    - Ensure patency of IV access, extravasation of calcium can cause necrosis.
    - Sudden push will precipitate bradycardia. Hence, it is diluted with D5 and infused over 20 minutes.
    - Calcium precipitates with bicarbonate. Combining it with bicarbonate containing solutions is avoided.
- 1 mL of calcium gluconate contains 9 mg/mL of calcium
- Refractory hypocalcemia occurs in:
  - Vitamin D deficiency
  - Phosphate loading due to undiluted cow milk feeding in newborn or early infancy
  - Renal failure
  - Hypoparathyroidism
  - Hypomagnesemia.
- *Check serum phosphate:* Normal levels are noted in vitamin D deficiency. Elevated levels are characteristic

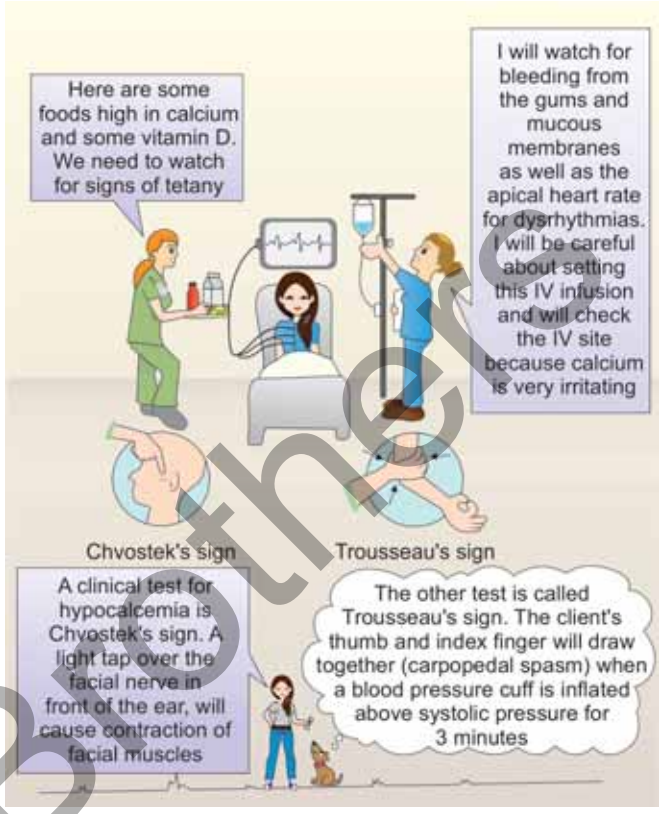
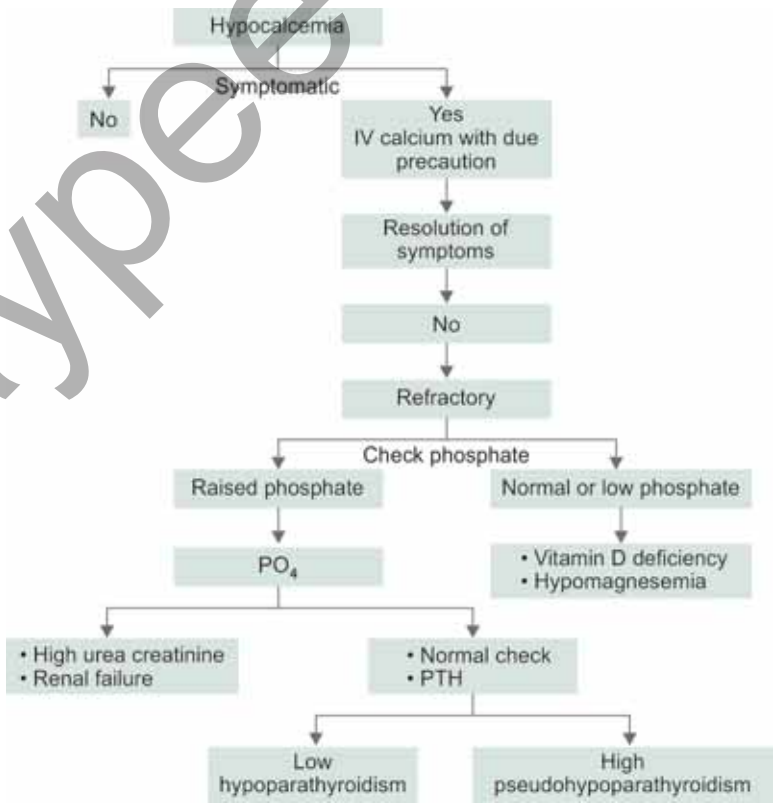


Fig. 13.18: Treatment of hypocalcemia.

Flowchart 13.9: Approach to hypocalcemia.



(PTH: parathyroid hormone)

in renal failure, phosphate loading due to undiluted cow milk feeding and hypoparathyroidism. In all these conditions, apart from treating the underlying conditions, vitamin D and calcium are given.

- Suspect hypomagnesemia, when hypocalcemia is refractory. Hypokalemia often coexists.

Key Points

1. Electrolytes are important for the normal functioning of the nerve, heart and muscle cells by maintain voltages across their cell membranes and carrying electrical impulses.
2. Kidneys work to keep the electrolyte concentrations in blood constant.
3. If electrolytes are less than normal, they should be replaced.
4. Sodium levels affect brain function.
5. Calcium levels affect the heart predominantly.
6. Potassium levels impact the function of the skeletal, smooth and cardiac muscle.

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