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SECOND EDITION

ILLUSTRATED TEXTBOOK OF PEDIATRICS



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Foreword
MR Khan

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3 Fluid and Electrolyte Balance and Its Disorders

INTRAVENOUS FLUID AND ELECTROLYTE

Ideally fluid, electrolyte and calorie replacement should be given by enteral routes. Maintenance intravenous (IV) fluids do not provide adequate calories, proteins, fat, minerals and vitamins. A patient receiving maintenance IV fluid is receiving inadequate calories and will loss 0.5–1% of body weight each day. It is imperative that the patient does not remain on maintenance therapy indefinitely. This is, however, typically not problematic for a patient receiving IV fluid for a few days. IV fluid should only be given when enteric fluid is not possible or appropriate. This chapter mostly deals with IV fluid and electrolytes involvement assuming infants and children are unable to take oral and enteral fluids.

General Points

Water

- Approximately 80% of the body weight at birth is of water
- This is reduced to 60% by 1 year of age
- Approximately two-thirds is intracellular and one-third is extracellular. Extracellular fluid (ECF) is proportionately greater in neonates (initially two-thirds) and falls as the child grows and cells increase in number
- Adipose tissue stores less water, so postpubertal girls have lower total body water content than boys and prepubertal girls.

Electrolytes

- Within the intracellular compartment:
  - Potassium (K^+) is the principal cation
  - Phosphate (PO_4) and proteins are the main anions.
- Within the extracellular compartment:
  - Sodium (Na^+) is the principal cation
  - Bicarbonate (HCO_3^-) and chloride (Cl^-) are the main anions.

Daily Requirements

Fluid Requirements

Fluid needs can be calculated with the weight of the child using the calculations depicted in Table 1.

Table 1: Daily fluid requirement calculation

Child's weight (kg)	"Maintenance" (mL/24 h)
2–10	100 mL/kg
10–20	1,000 mL plus 50 mL/kg for each kg >10 kg
>20	1,500 mL plus 20 mL/kg for each kg >20 kg
Maximum amounts: Males: 2,500 mL/24 h; females: 2,000 mL/24 h	

- This guide amount is reduced:
  - In neonates: 50 mL/kg/day on day 1 of life, 75 mL/kg/day on day 2
  - After major surgery
  - If child is intubated on humidified ventilation (25–50% reduction)
  - Where fluid is being retained (cardiac failure or renal impairment)
- The amount is increased in the following conditions:
  - With abnormal skin
  - Who are pyrexial or on phototherapy (25% increase)
  - With tachypnea or on nonhumidified mask ventilation.

Electrolyte Requirements

Daily electrolyte requirements have been summarized in Table 2.

Table 2: Daily electrolyte requirements (mmol/kg/day)

Na^+	2–4
K^+	2–3
Ca^{2+}	1
Mg^{2+}	1
Cl^-	3–5
PO_4^{3-}	2–3

- Potassium concentrations should not normally exceed 40 mmol/L for peripherally administered solutions (risk of extravasation injury)
- Avoid giving calcium via peripheral lines unless absolutely essential. Monitor peripheral cannulation sites carefully where calcium is added to fluid as skin burns can result from extravasation.

Fluid Replacement

Ideally all fluids, electrolytes and calorie replacements should be given by the enteral route, and IV fluids should only be prescribed when this is not possible or appropriate (Table 3).

Osmolality and Tonicity

- Osmolality is a measure of the solute concentration, or the number of solute particles present in solution and is independent of the size or mass of the particles
- Tonicity is the effective osmolality and is equal to the sum of the concentrations of all the solutes that have the capacity to exert an osmotic force across a membrane
- Solutes that penetrate freely across a membrane do not exert an osmotic force across it
- When infusing 5% glucose, for instance, the glucose is taken up and metabolized by cells, leaving only electrolyte-free water. Therefore, glucose does not exert significant sustained osmotic force across a membrane. In contrast, sodium exerts more and significant sustained osmotic force across membrane.

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Table 3: Composition of commonly used IV fluids

	Serum	0.9% saline	0.9% saline/g5w	0.45% saline	0.45% saline/ g5w	0.18% saline/g4w	HS
Na^+	135–145	154	154	77	77	31	131
K^+	3.5–5						5
Ca^{2+}	2.2–2.6						2
Cl^-	95–105	154	154	77	77	31	111
HCO_3^-	24–32						29*
Na^+/Cl^-	1.28–1.45:1	1:1	1:1	1:1	1:1	1:1	1.18:1
Glucose	4–6		50 g/L		50 g/L	40 g/L	
Osmolality (mOsm/kg)	275–295	286	580	142	432	355	276
Calorie/L	-	0	200	0	200	160	
Tonicity		Iso	Iso	Hypo	Hypo	V. hypo	Iso

All values in mmol/L, except Na^+/Cl^- (ratio); osmolality (mOsm/kg); g5w, 5% glucose; g4w, 4% glucose; HS, Hartmann's solution; *As lactate; Iso, isotonic; Hypo, hypotonic; V. hypo, very hypotonic

How to Calculate Osmolality and Tonicity

Plasma osmolality can be estimated by calculation based on the following formula:

Osmolality = $2 \times [Na^+] + [glucose]/18 + BUN/2.8$.

If the calculated osmolality is less than the laboratory measured osmolality, then there is another osmotically active substance present, i.e. sugar, mannitol, etc.

Glucose and blood urea nitrogen (BUN) are measured in mg/dL. Division of this value by 18 and 2.8 as shown converts units into mmol/L.

Effective Osmolality also called Tonicity

As mentioned earlier, solutes that penetrate free across a membrane do not exert an osmotic force across it. Urea usually contributes little to the plasma osmolality. Urea is not confined to the extracellular space because it readily crosses the cell membrane and intracellular concentration approximately equals its extracellular concentration. Therefore it is the effective osmolality which determines the osmotic force that is mediating the shift of water between ECF and ICF. The effective osmolality also called tonicity, therefore, can be calculated as follows:

Effective osmolality = $2 \times [Na^+] + [glucose]/18$.

Few Points on Intravenous Solution

Osmolality

A normal plasma osmolality is 285–295 mOsm/kg. Infusing an IV solution peripherally with a much lower osmolality can cause water to move into red blood cells causing hemolysis. This IV fluid is generally designed to have an osmolality that is either close to 285 or greater. Higher osmolality does not cause problem. Half strength normal saline (0.45% saline) or 0.2 normal saline has lower osmolality. However, if given with 5% dextrose, osmolality can be increased significantly. 0.2 normal saline has osmolality of 68, but 5% dextrose with 0.2 normal saline has osmolality of 472. Therefore, 0.45% saline or 0.2% saline should not be used alone as IV fluid.

On the other hand, higher osmolality (more than plasma osmolality) does not usually create problem. Like 5% dextrose of 0.2 normal saline has osmolality of 472 due to added glucose. But glucose contributes little to osmolality; as mentioned earlier, glucose is taken up and metabolized by cells leaving only electrolyte in serum.

However, in exceptional conditions, it can create problem, for example, in hyperglycemia and in diabetic ketoacidosis (DKA). In such conditions, there will be a shift of water from intracellular to extracellular space. This shift of water in the extracellular space causes dilutional hyponatremia despite elevated plasma osmolality.

Tonicity

Isotonic solution should not only be used for acute volume replacement but also as maintenance solution. Isotonic solution containing glucose, like 5% dextrose in normal saline, is preferred to isotonic infusion without glucose (Hartmann's solution). 0.9% saline (Na^+ = 154 mmol/L) containing 5% glucose is an ideal isotonic solution for infusion. Hypotonic solutions are 5% dextrose or 10% dextrose or 5% glucose + 0.45% normal saline or 4% glucose with 0.18% saline. They are currently not recommended for IV maintenance to avoid risk of hyponatremia.

Hyponatremia and Its Consequences Associated with Hypotonic IV Infusion

Sodium chloride 0.45% (Na^+ = 77 mmol/L) with 5% glucose or 0.18% saline (Na^+ = 31 mmol/L) with glucose not only containing less sodium as compared to 0.9% (Na^+ = 154 mmol/L) saline with or without glucose but also aggravating dilutional hyponatremia which may occur when given in unrecognized stressful clinical conditions like respiratory disease, dehydration, intracranial lesions like meningitis, etc. due to inappropriate Antidiuretic Hormone (ADH) secretion in such conditions. Hyponatremia may cause acute neurological problems with significant morbidity and mortality.

KCl Administration

20 mEq/L of KCl should be given in maintenance fluid to prevent hypokalemia.

Managing Fluids and Electrolytes

- 0.9% saline or 0.9% saline with 5% glucose has become the fluid of choice for maintenance and deficit replacement in most cases.
- Hartmann's solution although isotonic but does not contain glucose, therefore, not ideal for IV maintenance solution

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- Previous use of 0.45% saline with 5% glucose (half normal with glucose) or 0.18% saline with 4% glucose (quarter normal with glucose) has been declined with concerns about hyponatremia
- Preterm and newborn infants may be an exception to this rule as they have limited ability to excrete Na^+. They may be managed with 10% glucose, added electrolytes and regular monitoring
- However, many centers have not yet adopted the practice of giving isotonic infusion (0.9% saline with 5% glucose) as maintenance fluid
- If 0.45% saline with 5% glucose or 0.18% solution with 4% glucose has to be given, it should be infused at reduced rate to avoid risk of water intoxication
- Glucose content of fluid can be adjusted to the needs of the child
- 20 mEq/L of KCl can be administered to prevent hypokalemia associated with IV infusion.

Fractional Excretion of Water and Sodium

- The fractional excretion of water (FEH_2O) is the fraction of the glomerular filtrate volume that appears as urine, expressed as a percentage
- The fractional excretion of sodium ($FENa^+$) is the fraction of the sodium filtered by the glomeruli which appears in the urine, expressed as a percentage
- Values depend upon water intake, ADH levels, the renin/angiotensin system, renal health and maturation, medications and other factors
- Children with healthy kidneys can lower both FE values to less than 1%
- In renal hypoperfusion, the $FENa^+$ is less than 1%
- In intrinsic acute renal failure (ARF), it is greater than 2.5%
- Recent use of diuretics makes interpretation of FE values difficult
- Together, urinary sodium concentration, urinary osmolality, $FENa^+$ and FEH_2O can be interpreted to understand a patient's fluid and electrolyte status
- Urinary sodium concentration cannot be interpreted alone, as sodium and water excretion may vary together or independently.

Calculation of FEH_2O and $FENa^+$

$FEH_2O = (PCr/UCr) \times 100$
$FENa = [(UNa/PNa) \times (PCr/UCr)] \times 100$
PNa = Plasma sodium, mmol/L
UNa = Urine sodium, mmol/L
PCr = Plasma creatinine, µmol/L
UCr = Urine creatinine, µmol/L
(UCr often reported in mmol/L, may need multiplication by 1,000).
The fractional excretion values are expressed as a percentage.

Intravenous Fluids—a Few Points and Some Fallacies and Myths

Sick infants and small children are particularly susceptible to increased ADH secretion as part of the "stress response"

- This is not "inappropriate" ADH syndrome but an evolutionary physiological response to real or potential hypovolemia (e.g. following surgery or trauma), when the body needs to retain fluid
- Obviously evolution did not anticipate the advent of IV fluid therapy. Combined *increased* ADH secretion and prescribed hypotonic IV maintenance fluid result in iatrogenic hyponatremia which becomes a major problem with a significant associated mortality.

Very hypotonic; hypotonic; isotonic; or hypertonic fluids as IV maintenance and their possible consequences

- Very hypotonic fluids are associated with hyponatremia whereas the moderately hypotonic and the isotonic fluids appear to be safer
- Isotonic solutions should be prescribed for "maintenance fluids" in most cases
- If hypotonic fluids are prescribed, the child should be reviewed regularly, particularly if they are, or have recently been sick.

The frequently used formulae for IV fluid prescription may in fact be an overestimation of actual requirement

- This calculation is based on calorie consumption (1 mL of fluid for every kCal consumed) which itself is based on the child's weight
- 80% of energy expenditure occurs in the major organs which account for less than 10% of the body weight. The assumption that increase in weight results in a direct proportional increase in energy expenditure is incorrect. Energy expenditure and hence fluid requirements may be overestimated, particularly in obese children
- Sick children consume significantly less energy than healthy children due to inactivity.

All these factors have implications for hospitalized children:

- Intravenous maintenance should be isotonic (preferably 0.9% saline with 5% glucose or Hartmann's solution/ Ringer's lactate) or moderately hypotonic (0.45% saline with glucose 2.5% or 5%)
- All very hypotonic IV fluids are restricted to specialized use only
- All additional losses, e.g. Nasogastric (NG) losses or excessive diarrhea/stoma losses should be replaced with 0.9% saline
- The volume of maintenance fluid prescribed should be restricted in all cases where ADH secretion is raised [e.g. postoperatively, central nervous system (CNS) and lung pathology]. Prescribe 66–75% of full maintenance (based on body weight).

PICU patients have even more reasons to restrict IV fluid prescriptions

- Children are extremely inactive (sedated and paralyzed)
- Mechanical ventilation reduces work of breathing and therefore calorie consumption
- Insensible loss is reduced through humidification of inspired gases
- Antidiuretic hormone secretion is induced by positive pressure ventilation, lung pathology, CNS insults, and other common situations in pediatric intensive care unit (PICU)
- This means that children on ventilators should be restricted to 50–75% of normal IV maintenance fluids (calculations based on their weight).

Note: This does not apply to children with large fluid requirements, e.g. burns, sepsis, etc. or to NG feeding prescriptions.

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Fluid Deficits and Rehydration

- Shock states or ongoing losses of body fluids should be treated initially with normal saline, plasma expanding colloids or 4.5% human albumin solution. Further losses may necessitate the use of blood products
- A fluid deficit is estimated by weight or clinical examination, and the deficit added into maintenance fluids for replacement at a rate which is governed by the extent of the deficit and its cause
- Fluid deficit is most accurately gauged by a change in weight
- Where weighing is impossible or no recent weight is available for comparison, a clinical estimate of dehydration may be made
- This is expressed as the number of mL of water lost per 100 g of body weight or the percentage of dehydration
- Rates of rehydration need careful recalculation and adjustment in each child.

Clinical Estimation of Dehydration

- *Five percent dehydration*: Reduced skin turgor, sunken eyes and/or fontanel, dry mucous membranes, decreased peripheral perfusion, irritability, oliguria, pyrexia
- *Ten percent dehydration*: Lax skin, poor perfusion, drowziness, anuria
- Using this estimate, the child's fluid deficit can then be calculated:

Deficit in liters = [Weight (kg) × % (Dehydration)]/100.
A 5% dehydration in 10 kg child is:

$$\text{Deficit in liters} = \frac{10 \times 5}{100} = 0.5 \text{ L or } 500 \text{ mL}.$$

Rate of Deficit Replacement

- Shock needs to be addressed swiftly (20 mL/kg of 0.9% sodium chloride)
- Severe dehydration (10% or more) should be corrected quickly:
  - In infant less than 12 months, 100 mL/kg Ringer's lactate, cholera saline or 0.9% sodium chloride should be given in 6 hours of which 30 mL/kg in first hour followed 70 mL/kg in next 5 hours
  - In child of 12 months to 5 years with severe dehydration is corrected in 3 hours by giving 100 mL/kg fluid of which 30 mL/kg is given in 30 minutes followed by 70 mL/kg in next 2½ hours.
- In moderate dehydration (some dehydration 5–9%), fluid deficit in liter [weight (kg) × % (dehydration)]/100 is given over 6 hours (approximately 75 mL/kg)
- In all above cases, enteral fluid (or saline) can be given orally or by NG tube, if facilities for IV are not available or not appropriate
- In hypernatremia and hyponatremia, 48–72 hours may be needed for deficit replacement. This is necessary to avoid neurological complications caused by rapid changes in serum Na^+. However, severe hyponatremia will require quick correction.

Severe Dehydration

- Children who are greater than or equal to 10% dehydrated are prone to complications such as renal and cerebral venous sinus thrombosis
- These complications should be remembered when the expected responses in urine output or conscious level are not obtained with fluid therapy
- Appropriate further examination and investigation should be undertaken.

Colloids and Its Advantage in Clinical Practice

Colloids are aqueous electrolyte solutions containing large molecules-proteins in the case of human albumin solution (molecular weight of 68,000 Daltons) and gelatins in gelofusine and hemaccel (molecular weight of 30,000 Daltons). The large molecules leak slowly from the circulation through the capillary endothelium and therefore these solutions exert a colloid osmotic (oncotic) pressure. The advantage of colloids is that they stay in the circulation longer and thus less is needed to resuscitate the intravascular space when compared to crystalloids. In theory, they should also cause less edema. Best use in clinical practice is in dengue shock syndrome refractory to crystalloid solution infusion.

Human albumin solution 4.5% is a heat-sterilized human blood by-product that has a half-life of 16 hours in the circulation. Gelofusine and hemaccel persist for less time in the circulation but for longer than crystalloids. All three solutions contain plasma-like concentrations of sodium and chloride. Larger molecular solutions such as hespan and pentaspan as well as dextran should be avoided.

Crystalloids versus Colloids

Whilst it is standard practice to prescribe maintenance IV therapy with crystalloid solutions, there is controversy regarding the suitability of colloids in resuscitating patients with hypovolemia and sepsis. Proponents of colloid point to their efficiency (less required for the same effect) and physiological effect. Opponents point to their cost (colloids are considerably more expensive) and lack of evidence base. However, most intensivists use a combination of both depending on the situation—often starting resuscitation with IV crystalloids, e.g. at 20 mL/kg, and introducing colloids if more aggressive resuscitation is required (5–10 mL/kg). Postoperative cardiac patients are often treated with colloid IV and many intensivists use human albumin for meningococcal sepsis.

Monitoring Fluid Therapy

The key to effective and safe practice in fluid and electrolyte therapy is careful documentation and regular monitoring of the following:

Clinical Parameters

- *Weight:* Remember individual weighing-scales differ in accuracy
- *Fluid balance:* Intakes-outputs all need to be charted
- *Perfusion state:* pulse and central capillary refill time
- *Conscious level:* Glasgow Coma Score (GCS) should not drop
- Hydration state
- Cannulation sites.

Laboratory Parameters

- *Serum electrolytes:* Especially where supplements are given
- *Serum urea and creatinine:* Rising values alert to renal impairment
- *Hematocrit:* A marker of intravascular status, if no bleeding

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- Blood glucose
- *Urine electrolytes:* Urine Na^+ and Cl^- less than 20 mmol/L in hypovolemia.

Fluid Losses

- Losses incurred such as hemorrhage, and capillary leak.
- Losses from the gastrointestinal (GI) tract will depend on their origin and may require specific replacement intravenously (Table 4).

Table 4: Composition of gastrointestinal (GI) fluids (mmol/L)

Fluid	Sodium	Potassium	Chloride
Gastric	20–80	5–20	100–150
Pancreatic	120–140	5–15	40–80
Biliary/upper small intestine	100–140	5–15	80–130
Ileostomy	40–140	5–15	20–120
Diarrheal	10–90	10–80	10–110

- Fluid balance fluctuates rapidly and widely in seriously ill patients
- Carefully measured input (IV and enteral) and output (urine, drain losses, GI losses) values must be recorded hourly
- Insensible losses are often overlooked:
  - Important as children have a ↑surface area to body weight ratio
  - Come from skin and respiratory tract
  - Vary with the patient's temperature, dimensions and ventilation (humidification of inspired gases ↓respiratory losses by 80%)
- 10–30 mL/kg/day (300 mL/m^2/day) is a reasonable estimate of insensible losses

ELECTROLYTE IMBALANCE

Hyponatremia: Na^+ <130 mmol/L

- Causes are as follows:
  - *Sodium loss*: Diarrhea particularly associated with cholera, burns, adrenal failure including Congenital Adrenal Hyperplasia (CAH), hypothyroidism; thiazide diuretics.
  - *Water gain*: Syndrome of inappropriate ADH secretion (SIADH); edema, e.g. congestive cardiac failure (CCF), liver failure; oliguric renal failure, e.g. acquired excessive IV rehydration; psychogenic polydipsia (very dilute urine and dilute plasma).
- Syndrome of inappropriate secretion of ADH:
  - Low serum sodium (and often urea and creatinine)
  - Plasma osmolarity less than 280 mOsmol/kg
  - Inappropriately concentrated urine greater than 100 mOsmol/kg
  - Elevated urine sodium greater than 20 mEq/L.

Causes of Hyponatremia in PICU (Table 5)

- Causes of hyponatremia can be grouped by assess-ment of ECF volume status and urine Na^+
- Measurement of FEH_2O and $FENa^+$, and urinary osmolality help in the diagnosis
- More than one cause may coexist [e.g. SIADH and cerebral salt wasting (CSW) in head injury]
- SIADH.

Clinical Features

May complain of nausea, headache, lethargy with progression to confusion. Seizures occur if sodium falls below 120 mmol/L.

- Evidence of shock associated with dehydration in diarrheal disease, adrenal insufficiency including CAH. Other clinical features depend on the cause.

Management of Hyponatremia

- Assess the hydration status
- Evidence of adrenal insufficiency such as hyper-pigmentation, ambiguous genitalia.

Investigations

- Blood-urea and electrolytes, glucose, plasma osmolality (normal = 275–295 mOsm/kg)
- Urine-dipstick (expected specific gravity >1.005) and osmolality (expected >750 mOsm/kg); 24-hour urinary collection for urinary sodium.

Treatment

- Depends on specific cause and severity of hyponatremia
- Hyponatremia management associated with diarrhea and dehydration (discussed in chapter Gastroenterology)
- Acute symptomatic hyponatremia, for example, associated with convulsion or shock should be corrected quickly
- Patients who have symptomatic hyponatremia like convulsion, sodium depletion should be corrected quickly as follows: Use 3% sodium chloride, (513 mmol/L or 0.5 mmol/mL). The exact sodium deficit can be calculated (assessing assumed volume of distribution of Na^+ of 60% body weight) using the classical formula
  Na^+ deficit in mmol = Weight (kg) × 0.6 × (125 – Current plasma Na^+). For example, in a 10 kg weight child with hyponatremic convulsion with plasma sodium of 120 mmol/L, the Na^+ deficit to be corrected:
  Na^+ deficit = 10 × 0.6 × 5 = 30 mmol.
  1 mmol of Na^+ is present in 2 mL of 3% Na^+ chloride. 30 mL of Na^+ is present in 60 mL of 3% Na^+ chloride. Therefore, if given quickly within 4 hours, give 3% Na^+ chloride at 15 mL/hour.
  (Discuss with PICU personnel if convulsion occurs with hyponatremia).

Hyponatremia associated with hypovolemic shock—20 mL/kg, 3% sodium chloride should be given quickly (within 4 hours):

Table 5: Causes of hyponatremia in PICU

ECF volume status	Ur Na^+ <20 mmol/L	Ur Na^+ >20 mmol/L
Normal	-	H_2O excess, SIADH, NSAID use, hypoadrenal/thyroid
Increased (edema)	Capillary leak syndrome, low cardiac output, hepatic failure	ARF
Decreased	3rd space loss, GI loss, burns	Polyuric phase of ARF, osmotic diuresis, CSW

Abbreviations: PICU, pediatric intensive care unit; ECF, extracellular fluid; Ur, urine; SIADH, syndrome of inappropriate ADH secretion; NSAID, nonsteroidal anti-inflammatory drug; GI, gastrointestinal; ARF, acute renal failure; CSW, cerebral salt waiting.

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- If symptoms are severe that is if child is fitting, give 4 mL/kg 3% sodium chloride over 30 minutes and repeat. If symptoms persist, give 2 mL/kg over 15 minutes and repeat if symptoms persist
- Stop at plasma sodium of 125 mmol/L, or if sodium is increased greater than 0.5 mmol/hour
- Hyponatremia associated with diarrhea with nonsevere dehydration should be preferably corrected by sodium containing Oral Rehydration Salts (ORS)
- Hyponatremia associated with diarrhea due to cholera should preferably be corrected by previous higher sodium containing ORS (sodium of 90 mmol/L) rather currently used hypo-osmolar low sodium (sodium 75 mmol/L) containing ORS.

Treatment of chronic hyponatremia: The neurons adjust to the lower serum osmolality by lowering their own osmolality. Cerebral edema is less severe, but it is the rapid treatment that can cause mortality or central pontine myelinolysis. In such condition, the sodium requirement is determined as follows:

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

If desired sodium is 125 and present sodium is 120 and child weight is 10 kg then sodium deficit is: 5 × 10 × 0.6 = 30 mEq.

It should be corrected slowly over 48 hours.

Treatment of hyponatremia in other conditions:

In PICU:

- Best dealt by taking preventive measures—restrict intake to 50–75% of normal maintenance for patients on positive pressure ventilation and receiving humidified gases
- If hyponatremia is due to water retention (i.e. SIADH), restrict fluid to 25–50% of normal maintenance rather than increased sodium supply
- With severe (or symptomatic) hyponatremia, less than 125 mmol/L fluid restriction alone would take too long to rescue the patient from dangerously low sodium concentrations. Use 3% saline (i.e. 523 mmol/L or 0.5 mmol/mL):
  - The exact sodium deficit can be calculated (assuming a volume of distribution of Na^+ of 60% body weight) using the classic formula:

Na^+ deficit (in mmol) = Weight (kg) × 0.6 × (125 – Plasma Na^+).

Further Management

- Take a detailed history focusing on precipitating factors—water excess, chest infection, habitual juice drinking and head injury
- Examine the child with particular attention to hydration state, perfusion and neurological system.

Hypernatremia: Na^+ >150 mmol/L

- Results from either:
  - Water deficiency (more than sodium loss)
  - Sodium excess (more than water gain).

Causes (Table 6)

- Loss of water (more than sodium):
  - Gastrointestinal water loss (osmotic diarrhea, gastroenteritis in bottle fed infant)
  - Insensible water loss (fever, hyperventilation, burns)

Table 6: Causes of hypernatremia in PICU (all figures in mmol/L)

Urine sodium	Urine osmolality		
	<800	Variable	>800
< 20	-	-	GI water loss
Variable	Diabetes insipidus	-	↓Water intake
>20	Hyperglycemia	-	-
>75	-	↑Na^+ intake	-

  - Diuretic therapy (osmotic diuretic, loop diuretic)
  - Hyperglycemia (osmotic diuretic)
  - Renal disease
  - Diabetes insipidus
- Sodium excess (more than water):
  - Excess ingestion—including salt poisoning, iatrogenic (IV fluids with high sodium or salt containing drugs or hyperosmolar oral saline). Currently low osmolar ORS is used in diarrhea to prevent hypernatremia
  - Near-drowning (seawater)
  - Cushing's syndrome
  - Conn's syndrome.

Clinical Features

- Targeted history focusing on causes (diarrhea and vomiting, head injury, thirst and polyuria, milk formula use) and any neurological symptoms
- Weakness, confusion progressing to seizure, coma
- Clinical signs of dehydration may be masked as ECF volume is protected by the high Na^+.

Investigations

- Blood-urea and electrolytes, glucose, plasma osmolality (normal = 275–295 mOsm/kg)
- Urine-dipstick and osmolality. If less than 750 mOsm/kg, concurrent with plasma osmolality greater than 295 mOsm/kg, this is diagnostic of diabetes insipidus.

Management

- Cautious rehydration using ORS or with 0.9% saline or 0.45% IV saline. Aim for a slow fall of Na^+ at 0.25 mmol/L/hour.
- Rehydration over 48 hours, using ORS or IV fluids. For example, for 12 kg child with 5% dehydration:
  - Maintenance fluids in 48 hours = 1,100 mL/24 hour (up to 10 kg, 100 mL/kg and 50 mL/kg for each kg after 10 kg)
  - Fluid deficit = 5% of 12 kg = 600 mL or 0.6 L (50 × 12 in mL or weight in kg × percentage of dehydration/100 in liter)
  - Replace 2-days maintenance with deficit over 48 hours: (1,100 + 1,100 + 600 = 2,800 mL in 48 hours, i.e. 58.3 mL/hour) at 14 drops/minute in normal drop or 56/minute in microdrop (1 mL = 15 drop or 60 microdrop). One drop in normal burette in IV infusion = 4 drops in microburette. If the child is taking some fluid orally then adjust IV fluid accordingly (see also in Gastroenterology chapter).
- Check urea and electrolyte every 4 hours. Aim to reduce sodium by no more than 10 mmol/L every 24 hours.
- Specific replacement of ADH is indicated for central or neurogenic diabetes insipidus.
- Resistance to replacement therapy suggests a nephrogenic cause.

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Hypokalemia: Serum K^+ <3.5 mmol/L

- Ensure that sample is the not contaminated.
- If K^+ is less than 2.5 mmol/L, the Electrocardiogram (ECG) changes normally evident (ST depression + U waves).

Causes

- Inadequate intake (typically in PICU patient in IV fluids).
- Potassium loss:
  - Gastrointestinal (diarrhea most important cause)
  - Diuretic therapy
  - Hyperglycemia (osmotic diuretic)
  - Renal disease
  - Low Mg^{2+} can cause hypokalemia and hypocalcemia.
  - Cushing's syndrome
  - Conn's syndrome
- Movement of potassium into cells:
  - Acidosis correction
  - Insulin use
  - Beta-2 (β_2) agonist use.

Clinical Features

- Weakness, cramps, hypotonia, abdominal distension due to weakness of abdominal muscle and ileus (decreased peristalsis), hyporeflexia and lethargy.

Investigations

- Blood-urea electrolyte
- ECG provides better index of tissue potassium status of body than serum potassium level (ST depression, increase QT interval, presence of U wave) (Fig. 1)
- Blood gas analysis.

Management

- Assess for alkalosis associated with hypokalemia by blood gas analysis. Where present, this should also be treated—KCl therapy may be used as a source of both K^+ and Cl^- in the setting of diuretic-induced alkalosis
- Assess need for urgent correction of deficit:
  - Is the child symptomatic (cardiac arrhythmia, severe muscle weakness or illness)?
  - Or at risk (congenital heart disease, myopathy, severe illness)?
- Urgent replacement—use high strength KCl via a central line in aliquots of 0.5 mmol/kg over 1 hour then recheck K^+.

Injection of magnesium sulfate: In refractory hypokalemia as found in severe malnutrition with abdominal distension, injection of 50% magnesium sulfate intramuscular (IM) can improve hypokalemic status (0.3 mL/kg IM on 1st day and 0.1 mL/kg on 2nd and 3rd day in severe malnutrition).

Do not give boluses of potassium solutions.

- Nonurgent enteral supplements (preferred) and/or parenteral replacement by peripheral IV with K^+ additives in maintenance fluid (maximum concentration 40 mmol/L)
- Elucidate cause by considering depletion (diet, renal or gut loss) or redistribution (alkalosis, thyrotoxicosis and familial paralysis).

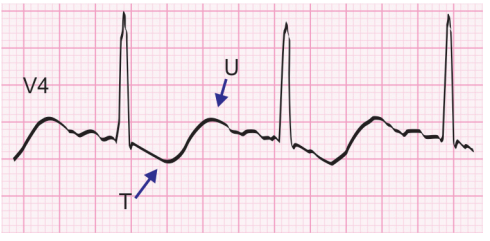

Fig. 1: ECG changes in hypokalemia

Hyperkalemia: Serum K^+ >5.5 mmol/L

- Normal ranges for K^+ are higher in infants who also tolerate hyperkalemia better than older children
- Ensure that the sample is not hemolyzed
- Stop exogenous administration of K^+
- Elucidate causes: Consider sources of and evaluate renal function.

Causes

- Excess K^+ [Total Parenteral Nutrition (TPN), blood transfusion, supplements]
- Acute renal failure
- Cell destruction:
  - Tumor lysis syndrome
  - Rhabdomyolysis
  - Acute burns
  - Trauma—crush injuries
- Congenial adrenal hyperplasia
- Addison's disease
- Drugs
- Movement of potassium out of cells
- Hemolysis during/after sampling—artifactually high K^+
- Acidosis
- Insulin deficiency (DKA) but total body sodium often depleted.

Clinical Features

- Tingling, numbness
- Cardiac arrhythmia (ectopic beat, drop beat, asystole).

Investigations

- Blood, electrolyte, urea and creatinine
- Hyperkalemia necessitates ECG monitoring—if K^+ greater than 6.5 mmol/L—ECG changes normally evident (long PR, tall peaked T waves and wide QRS) (Figs 2A and B)
- Urgent blood glucose and arterial blood gas (ABG).
- If evidence of increased tissue destruction, order for creatine kinase (CK), uric acid, PO_4, calcium.

Treatment

- Stop all IV and oral fluid containing K^+
- If arrhythmia present, give calcium gluconate 10%; 0.5–1 mL/kg over 5–10 minutes.

If no arrhythmia, treatment can be combined as follows:

- Nebulize salbutamol 2.5 mg for less than 5 years old children, 5 mg to greater than 5 years old children or with salbutamol IV 4 µg/kg over 10 minutes (shifts K^+ into cells)
- Sodium bicarbonate 8.4%; 1–2 mmol (mL)/kg over 30 minutes
- Glucose and insulin:

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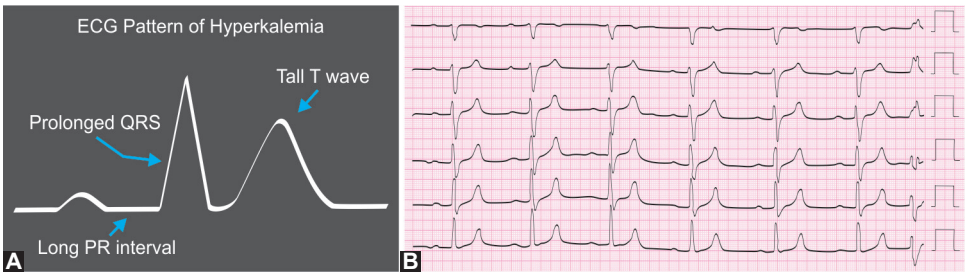


Figs 2A and B: (A and B) ECG changes of hyperkalemia (prolonged PR interval, broad QRS and tall peaked T wave)

- Bolus 0.1 units/kg insulin with 2 mL/kg of 50% glucose
  or
- Infuse 1 g/kg/hour glucose (i.e. 5 mL/kg/hour-20% glucose or 10 mL/kg/hour-10% glucose) with insulin infusion 0.1 unit/kg/hour
- Monitor blood-glucose every 15 minutes for first hour, then hourly thereafter
- Watch for late hypoglycemia

- Calcium resonium 1 g/kg orally or rectally with oral lactulose.

Calcium, Magnesium and Phosphate

These ions affect cardiac function and need to be monitored closely in the perioperative period of cardiac surgical cases. They are also important for skeletal system repair and growth.

Plasma Forms of Calcium and their Clinical Significance

Three main fractions:
1. Ionized (50%)
2. Albumin bound (40%)
3. Complex to phosphate citrate, etc (5–15%).

- Total Ca^{2+} lab values, corrected for albumin, should be 2.0 mmol/L
- Ionized Ca^{2+} (normal = 1.0–1.5 mmol/L) is not age or albumin dependent. It is affected by acid-base status (↑ by acidosis and ↓ by alkalosis).

Use ionized Ca^{2+} as the guide to treatment rather than total or corrected total calcium—threshold for IV calcium supplementation should be less than 0.7 unless symptomatic.

Hypocalcemia

Causes

Neonatal:
- Prematurity, infant of diabetic mother, RDS, birth asphyxia
- Transient neonatal hypoparathyroidism
- Maternal osteomalacia
- Maternal hyperparathyroidism
- Persistent idiopathic hypoparathyroidism
- DiGeorge syndrome
- Congenital hypomagnesemia with secondary hypocalcemia.

Older children:
- Vitamin D deficiency:
  - Nutritional rickets
  - Anticonvulsants
  - Renal rickets, ARF

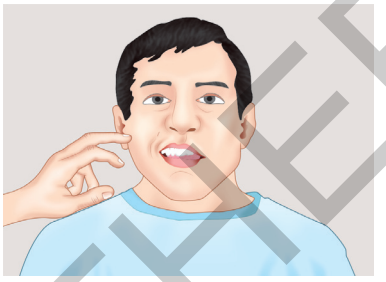
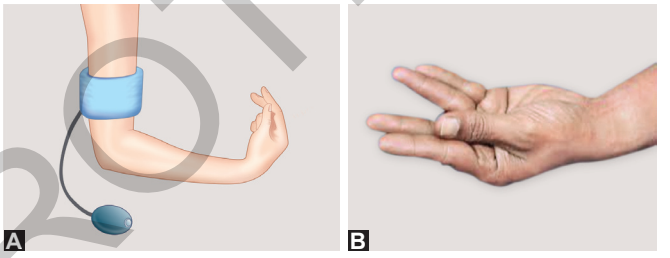


Fig. 3: Eliciting Chvostek's sign



Figs 4A and B: Carpal spasm following inflation of sphygmomanometer

  - Vitamin D dependent rickets
  - Nephrotic syndrome
- Hypomagnesemia
- Acute pancreatitis.

Clinical Features

- Likely to be triggered as a result of a convulsion or the presence of tetany, the clinical hallmark of hypocalcemia.
- Difficult to give an exact figure at which emergency intervention is required but serum calcium concentrations below 1.7 mmol/L (ionized calcium < 0.7 mmol/L) are likely to be associated with problems. Clinical presentation includes the following:
  - *Neuromuscular*: Paresthesia, tetany, convulsion, myopathy, laryngospasm (hypocalcemic stridor)
  - Positive Chvostek's sign characterized by twitching of the facial muscle in response to gentle tapping over facial nerve anterior to earlobe (Fig. 3).
  - *Positive Trousseau's sign*: Carpal spasm following inflation of sphygmomanometer over upper arm above systolic BP (Figs 4A and B).

Management

- Get adequate venous access in a large vein. IV calcium is very corrosive and may cause serious extra—vasation injury
- Commence slow IV injection of 10% calcium gluconate (1–2 mL/kg equivalent to 9–18 mg elemental calcium/kg or 0.225–0.45 mmol/kg) over 3 hours with ECG monitoring
- Repeat every 6–8 hours whilst symptoms persist
- May need longer continuous infusion as total body calcium may be low

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- Maintenance therapy with oral calcium (50–75 mg/kg/day of elemental calcium) and at the same time, start treatment with oral vitamin D (calciferol) 1,500 IU/day in neonates or 3,000 IU/day in older children
- Do not use 1α-hydroxyl-cholecalciferol at this stage or until definitive diagnosis has been made
- Low Mg^{2+} can cause hypocalcemia. Treatment with Mg^{2+} to prevent recurrence of hypocalcemia is done. Dose 0.2 mL/kg of 50% magnesium sulfate IV over 30 minutes. Repeat, if necessary.

Calcium chloride and gluconate formulations contain different amounts of Ca^{2+}. Both are irritant to veins (gluconate milder).

Hypercalcemia

Causes

- Hyperparathyroidism (primary, tertiary but not secondary)
- Idiopathic infantile hypercalcemia
- Vitamin D intoxication
- Vitamin A intoxication
- Milk alkali syndrome
- Prolonged immobilization (after fracture of long bones).

Some syndromic conditions:

- Williams syndrome (Fig. 5)
- Hypophosphatasia.

Clinical Features

- Constipation
- Nephrocalcinosis (Figs 6A and B)



Fig 5: A child with Williams syndrome

- Failure to thrive
- Renal calculi
- Polyuria (nephrogenic diabetes insipidus)
- Metastatic calcification
- Supravalvular aortic stenosis in Williams syndrome.

Investigations

- Full blood count (FBC), blood film, serum calcium (total), phosphate and venous bicarbonate
- Blood urea, creatinine
- Ionized calcium
- Parathyroid hormone (PTH)
- Liver function test including albumin and alkaline phosphatase (ALP)
- ECG: Short QT
- Imaging: Ultrasound of kidney for nephrocalcinosis.

Other investigations: As indicated, e.g. formal glomerular filtration rate (GFR), blood gas, etc.

Treatment

- Volume expansion, e.g. 3 L/m^2/24 hour IV solution Loop diuretic (Furosemide 1 mg/kg IV 6–8 hourly)
- Corticosteroid in chronic hypocalcemia
- Calcitonin infusion, 5–10 units/kg IV followed by 4 units/kg IV
- Bisphosphonate, e.g. pamidronate infusion in hypercalcemia of malignancy.

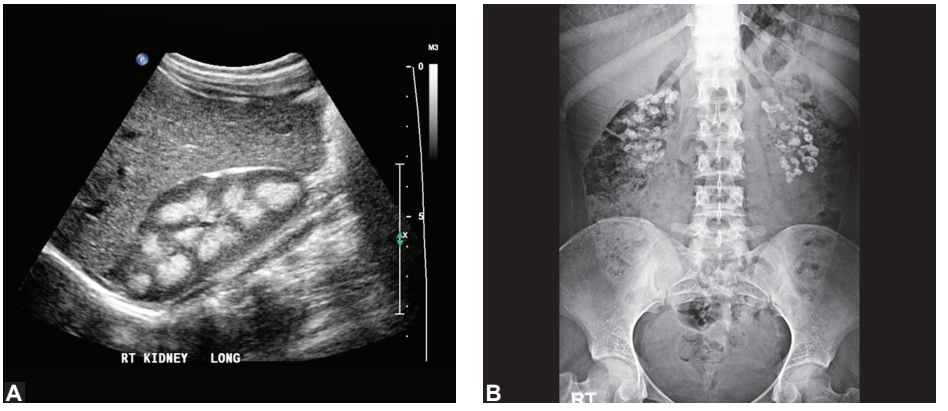
Magnesium: State of magnesium in plasma and their clinical significance

- Normal plasma Mg^{2+} (0.7–1.0 mmol/L)
- Mg^{2+} and Ca^{2+} absorption are linked
- Low Mg^{2+} levels are proarrhythmic
- Ionized fraction is the active fraction (like calcium) but not routinely measured in most centers.

Hypomagnesemia

Magnesium less than 0.6 mmol/L. Clinically concerning when it is under 0.3 mmol/L as it impairs the release of PTH, resulting in hypocalcemia. Secondary hypokalemia can also arise, especially with renal disease. Symptoms of isolated hypomagnesemia resemble those of hypocalcemia, but arrhythmias are unlikely if there are no cardiac anomalies.

Intravenous magnesium sulfate is given in infusion in the management of acute severe asthma as bronchodilator, but it



Figs 6A and B: Ultrasonogram of abdomen and plain X-ray abdomen showing nephrocalcinosis respectively

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is not clear whether such patients are magnesium deficient or not.

Causes

- *Decrease intake:* Severe malnutrition, particularly associated with hypokalemia.
- *Gastrointestinal loss:* Vomiting, diarrhea, protracted inanition, malabsorption conditions, e.g. celiac, cystic fibrosis (CF), inflammatory bowel disease (IBD).
- *Renal loss:* tubular disease, e.g. nephritis, Bartter's, Gitelman's; osmotically active agents, e.g. glucose, mannitol; medications, e.g. diuretics (loop and thiazides); nephrotoxic, e.g. amphotericin, cyclosporine.
- Acute renal failure (polyuric phase).
- Endocrine causes, i.e. hypoparathyroidism and hyperthyroidism.

Clinical Features

- Resembles those of hypocalcemia, but arrhythmia is unlikely
- Symptoms include the following:
  - Seizures, particularly in neonates
  - Stridor secondary to laryngospasm—may need intubation
  - Neuromuscular, e.g. tingling in hands, around mouth; carpopedal spasm and Chvostek's sign.

Investigation

ECG: Stinted T waves prolongation of ST.

Management

- If urgent, give 0.2 mmol/kg IV over 10 minutes (0.1 mL/kg of 50% magnesium sulfate diluted 5% with 0.9% saline)
- Neonates may be given 0.4 mmol/kg, appropriately diluted and given slowly
- In severe acute asthma:
  - 0.1 mL/kg in IV infusion for 20 minutes, followed by 0.06 mL/kg/hour until severity decreases
- In severe malnutrition:
  - 0.3 mL/kg IM on 1st day, followed 0.1 mL/kg/day on 2nd and 3rd day (useful in improving hypokalemic condition causing abdominal distension and feeding difficulty)
- Smaller doses of 0.1 mmol/kg are used in treating torsade de pointes [polymorphic ventricular tachycardia (VT)], pulmonary hypertension and acute severe asthma (nebulized or IV).

Hypermagnesemia

- Rarely seen except in renal failure and when given therapeutically
- Leads to muscle weakness and coma but not until very high levels are reached—often increase to 2–3 mmol/L in setting of Mg^{2+} treatment for pre-eclampsia, asthma, etc.

Low Phosphate Level

- Serum PO_4 varies with age and acid-base status (↑ in infancy and acidosis)
- Keeping level greater than 1.0 mmol/L helps cardiac function and bone growth
- Decrease phosphate results in poor bone mineralization causing osteopenia of prematurity in preterm babies, rickets in children and osteoporosis in adults
- Decrease in phosphate occurs in:
  - Nutritional rickets due to secondary hyperparathyroidism
  - Osteopenia of prematurity
  - Starvation/poor intake
  - Critical illness
  - Diuretic and renal replacement therapies
  - Parenteral nutrition
  - Hyperparathyroidism.

Clinical Features

- Decreased linear bone growth with pathological fracture
- Muscle weakness (rarely apparent clinically)
- Cardiac dysfunction.

Treatment

- Low levels are treated with 0.5 mmol/kg over 10 hours (over 1 hour if urgent) as 1 mL/kg of 13.6% potassium acid phosphate diluted × 10 with 0.9% saline (this also gives some dose of potassium)
- In osteopenia of prematurity, oral supplementation is given: Prescribe phosphate (joulic solution) in Very Low Birth Weight (VLBW) less than 1,500 g baby with ALP greater than 500 IU/L and plasma phosphate less than 1.8 mmol/L. In a breastfeed baby, 1.5 mmol/kg/day orally for 6 months corrected age.

High Phosphate Level

Causes

Causes of decreased excretion:

- Renal insufficiency (most important cause)
- Hypoparathyroidism or pseudohypoparathyroidism
- Increased intake:
  - Cow's milk intake
  - Vitamin D intoxication.

Other causes:

- Tumor lysis syndrome
- Acute hemolysis
- Rhabdomyolysis.

Clinical features of hypocalcemia (↑ phosphate precipitates calcium as calcium phosphate salts).

- Tetany, paresthesia, convulsion and muscle weakness.

Clinical features due to deposition of calcium phosphate salts in various organs

Eye:

- Photophobia, visual impairment.

Treatment

Treatment of underlying conditions: Treatment of renal failure, etc.

Phosphate binders: They are used to prevent intestinal calcium absorption.

Calcium carbonate and calcium acetate are most commonly used. Phosphate binders can decrease absorption 30–40% particularly if given before meal.

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Urea and Uric Acid

Urea is created to remove ammonia, a toxic by-product of protein catabolism that is not readily excreted. Uric acid is generated by purine metabolism, e.g. the breakdown of DNA. Uric acid is measured as urate, its salt form.

Both urea and uric acid are toxic if they accumulate.

Elevated urea is greater than 6 mmol/L.

Urea is soluble in water so is easily excreted by the kidney, thus high levels of urea seldom arise in isolation, and usually reflect impaired renal function, secondary to reduced GFR. Persistently high levels will cause itching and GI irritation, such as vomiting and nausea.

Causes

- Increased production. GI: Upper GI bleed, high protein diet; medications: Steroids, diuretics.
- Impaired excretion. Renal: prerenal (dehydration); renal (renal failure); postrenal (obstruction, e.g. urethral valve, stone).

Treatment

- Renal failure
- Assess hydration. Rehydrate, if necessary
- Review medication.

High Uric Acid

Urate is greater than 0.35 mmol/L. Uric acid becomes soluble in water at high concentration. Crystals are deposited in the kidney causing renal stones; in the joints causing gout and ultimately in the nerves with resultant neuropathies. Precipitation is exacerbated by dehydration or acidosis.

Causes

- Increased cell turnover: Malignancy, cyanotic heart disease, hereditary anemia and psoriasis.
- Associated syndromes: Lesch-Nyhan (X-linked, progressive developmental delay and spasticity, self-mutilation especially biting), Down's syndrome; glycogen storage disorders I, III, IV, V.

Treatment

- Increasing fluid intake IV rehydration
- Allopurinol 5 mg/kg/dose bd to qds PO (maximum 600 mg/day) to reduce production; probenecid 10 mg/kg/dose qds PO to increase excretion
- Consider alkalinization of urine.

Edema

Mechanisms of Edema

Movement of fluid across the capillary wall endothelium depends on:

- The nature of the molecule, i.e. its size, charge, water and fat solubility
- The balance of forces across the capillary wall
- The permeability of the capillary wall.

Water and water-soluble molecules (NaCl, glucose) pass across the capillary membrane with greater ease than large molecules such as proteins (albumin) or gelatins (gelofusine). This movement produces a low protein filtrate that is referred to as interstitial fluid. This is the ECF that sits in outside the vascular compartment.

The balance of forces affecting fluid distribution across the capillary depends on the balance between hydrostatic forces [capillary hydrostatic pressure (P_c)—interstitial fluid hydrostatic pressure (P_i)], forcing fluid out of the capillary and the osmotic forces [capillary colloid osmotic pressure (π_c)—interstitial fluid colloid osmotic pressure (π_i)], sucking fluid into the capillary. Thus:

$$\text{Flow of fluid} = k[(P_c - P_i) - \sigma(\pi_c - \pi_i)]$$

Where k is a filtration coefficient and σ is the coefficient that represents the permeability of the endothelium.

Capillary hydrostatic pressure falls from approximately 30 mm Hg at the arterial end (forcing fluid out of the capillary) to about 15 mm Hg at the venous end of the capillary. The pulmonary circulation has lower capillary pressures. These pressures are also slightly lower in infants and smaller children. Capillary colloid osmotic pressure (oncotic pressure) is generated by the "pull" of intravascular protein and is normally 25 mm Hg. Interstitial colloid osmotic pressure is about 10 mm Hg.

In health, the volume of fluid leaving the capillary exceeds that absorbed by a small amount. This excess is absorbed by the lymphatics.

When capillary filtration exceeds lymphatic drainage, there is an excess of interstitial fluid and edema that result. In intensive care unit (ICU), this is commonly seen in the peripheral subcutaneous tissues, the lungs (pulmonary edema), in the pleural space (effusion), and abdominal (ascites) and pericardial spaces. Edema may not only reflect disease severity but also impairs oxygen and nutrient delivery from the capillary to the cell.

From Starling's equation, we can see that edema may result from three causes:

1. Increased capillary pressure (P_c), e.g. in right heart failure, fluid overload or in dependent edema
2. Decreased colloid osmotic pressure (π_c); when there is a fall in plasma protein, e.g. malnutrition (↓intake), nephrotic syndrome (protein loss), hepatic failure (reduced synthesis).
3. Increased capillary permeability (↓σ); in diseases of inflammation circulating cytokines can cause massive and widespread capillary leak, e.g. dengue shock syndrome, meningococcemia, sepsis. In these conditions, the edema is usually high in protein content (>30 g/L), i.e. exudative type. Fluid may leak from capillary to body cavity, i.e. abdominal cavity causing ascites, pleural cavity causing pleural effusion.

Pulmonary Edema

Pulmonary edema occurs when pulmonary lymphatics become exhausted—initially interstitial edema occurs but this

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progreses to alveolar edema with accompanying reduction in compliance and intrapulmonary shunting. Classic signs are dyspnea and hypoxemia.

Pulmonary edema may be either:

- *Cardiogenic*: Myocardial dysfunction or inadequate emptying of left ventricle or atrium leads to subsequent increase in left ventricular end-diastolic pressure, left atrial pressure and pulmonary venous pressure resulting in increased pulmonary capillary pressure and pulmonary edema.
- *Noncardiogenic*: This is mainly due to increased capillary permeability and is seen in Acute Respiratory Distress Syndrome (ARDS) and pneumonia.

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