Fluid and Electrolyte Balance: Concepts and Vocabulary

Body Fluid Compartments:

- The major body constituent is **water.**
- **Intracellular fluid (ICF).**
- **Extracellular fluid (ECF).**
- ECF can be further subdivided into: plasma & interstitial fluid.
- Insensible loss can be thought of as surface evaporation.
- Intracellular fluid loss causes cellular dysfunction.
- Loss of blood leads to circulatory collapse.
- The term 'dehydration' simply means that fluid loss has occurred from body compartments.
- Over-hydration occurs when fluid accumulates in body compartments.
- Clinical assessments of skin turgor, eye-ball tension and the mucous membranes are not always reliable.

Electrolytes:

Sodium (Na^+) is the principal extracellular cation, and **potassium (K^+)** is the principal intracellular cation. Inside cells the main anions are **protein and phosphate**, whereas in the ECF **chloride (Cl^-)** and **bicarbonate ($HCO3^-$)** predominate. Urea and creatinine concentrations provide an indication of renal function, increased concentrations in the blood indicates a decreased glomerular filtration rate.

Concentration:

Remember that a concentration is a ratio of two variables: the amount of solute (e.g. sodium), and the amount of water.

Osmolality:

While the concentration of substances may vary in the different body fluids, the overall number of solute particles, the osmolality, is identical.

- Osmotic pressure must always be the same on both sides of a cell membrane.
- The osmolality of the ICF is *normally* the same as the ECF.
- The two compartments contain isotonic solutions.
- In man, the osmolality is around 285 mmol/kg.
- Serum osmolality = 2 x serum (sodium)
  (mmol/kg) (mmol/l)

Oncotic pressure:

The barrier between the intravascular and interstitial compartments is the capillary membrane. Plasma proteins exert a colloid osmotic pressure, known as oncotic pressure. Humans deprived of fluids die after a few days from circulatory collapse as a result of the reduction in the total body water.

Water:

- Water intake.
- Water losses changes in the volume of urine produced.
- Water excretion by the kidney is very tightly controlled by antidiuretic hormone, ADH.
- The body is also continually losing water through the skin as perspiration, and from the lungs during respiration.
- Water may also be lost in disease (ex. diarrhea, vomiting).

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AVP and the regulation of osmolality:
Secretion of AVP from the posterior pituitary gland. A rising osmolality promotes the secretion of AVP, a declining osmolality switches the secretion off. AVP causes water to be retained by the kidneys.

Sodium (Na^+):

- A quarter of the body sodium is termed non-exchangeable, which means it is incorporated into tissues such as bone and has a slow turnover rate.
- The sodium concentration is tightly regulated (around 140 mmol/l).
- Urinary sodium output is regulated by two hormones :
  - Aldosterone.
  - Atrial natriuretic peptide.

Aldosterone:
Aldosterone decreases urinary sodium excretion by increasing sodium reabsorption in the renal tubules. Specialized cells in the juxtaglomerular apparatus of the nephron sense decreases in blood pressure and secrete renin, the first step in a sequence of events that leads to the secretion of aldosterone by the glomerular zone of the adrenal cortex.

Atrial natriuretic peptide:
Secreted by the cardiocytes of the right atrium of the heart. It increases urinary sodium excretion.

Hyponatremia:
Hyponatremia is defined as a serum sodium concentration below the reference interval of 135-145 mmol/l.

Development of Hyponatremia:

- Hyponatremia can arise either because of loss of sodium ions or because of retention of water.
- When significant sodium depletion occurs, water is lost with it.
- Water retention: retention of water in the body compartments dilutes the constituents of the extracellular space including sodium, causing Hyponatremia.

Water retention:

- Water retention usually results from impaired water excretion and rarely from increasing intake (compulsive water drinking).
- Most patients who suffer from Hyponatremia due to water retention have the so-called syndrome of inappropriate antidiuresis (SIAD). SIAD results from the inappropriate secretion of AVP.

Sodium loss:

- Either from the gastrointestinal tract or in urine.
- Gastrointestinal losses commonly include those from vomiting and diarrhea.
- Urinary loss may result from mineralocorticoid deficiency (especially in aldosterone secretion).
- Initially in all of the above situations, sodium loss is accompanied by water loss and the serum sodium concentration remains normal. As sodium and water loss continue, the reduction in ECF and blood volume stimulates AVP secretion non-osmotically, overriding the osmotic control mechanism.The increase in AVP secretion causes water retention and thus patients become Hyponatraemic.

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Pseudohyponatraemia:

- A patient with a sodium concentration of 110 mmol/l who is completely asymptomatic
- A normal serum osmolality in a patient with severe Hyponatraemia, strongly suggestive of Pseudohyponatraemia.

Severity:

- Depends on the serum sodium concentration.
- How quickly the sodium concentration has fallen from normal to its current level?
- The presence of signs or symptoms attributable to hyponatraemia.
- Many experienced clinicians use a concentration of 120 mmol/l as a threshold in trying to assess risk.
- A patient whose serum sodium falls from 145 to 125 mmol/l is 24 hours may be at great risk.

Symptoms of hyponatremia:

- Nausea
- Malaise
- Headache
- Lethargy
- Reduced level of consciousness
- Seizures
- Coma
- Focal neurological signs are not usually seen until the sodium concentration is less than 110 to 115 mmol/l

Clinical examination of dehydration:

- Increased pulse
- Dry mucous membranes
- Soft/sunken eyeballs
- Decreased skin turgor
- Decreased consciousness
- Decreased urine output
- Decrease in blood pressure

- These signs should always be looked for; in Hyponatraemic patients they are diagnostic of sodium depletion.
- In the early phases of sodium depletion postural hypotension may be the only sign.

Biochemistry:

However, both sodium depletion and SIAD produce a serum osmolality reflecting hyponatraemia, and a high urine osmolality reflecting AVP secretion. Urinary sodium excretion is often increased in SIAD.

Edema:

- Edema is an accumulation of fluid in the interstitial compartment.
- Looking for pitting in the lower extremities of ambulant patients or in the sacral area of recumbent patients.
- It arises from a reduced effective circulating blood volume, due either to heart failure or hypoalbuminaemia (low concentration of serum protein).

Treatment:

- Hypovolaemic patients are sodium depleted and should be given sodium. More aggressive treatment (usually requiring hypertonic saline) may be indicated if symptoms attributable to hyponatraemia are present, or the sodium concentration is less than 110 mmol/l

Clinical note:
The use of oral glucose and salt solutions to correct sodium depletion in infective diarrhea is one of the major therapeutic advances of the last century and is life saving, particularly in developing countries. Family practitioners, nurses and even parent are able to treat sodium depletion using there oral salt solutions, without making biochemical measurements.

Hypernatremia:
Hypernatremia is an increase in the serum sodium concentration above the reference interval of 135-145 mmol/l.

Water loss:

- Pure water loss may arise from decreased intake or excessive loss.
- Severe Hypernatremia due to poor intake is most often seen in elderly patients.
- Less commonly there is failure of AVP secretion or action, resulting in water loss and Hypernatremia. This is called diabetes insipidus; it is described as central if it results from failure of AVP secretion, or nephrogenic if the renal tubules do not respond to AVP.
- However, loss in body fluids because of vomiting or diarrhea usually result in Hyponatremia.

Sodium gain:

- Thirdly, infants are susceptible to Hypernatraemia if given high-sodium feeds.
- (Conn's syndrome), where there is excessive aldosterone secretion and consequent sodium retention by the renal tubules.

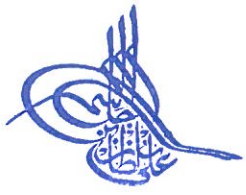
Clinical features:
If the patient has obvious clinical feature of dehydration it is likely that the ECF volume is reduced

Treatment:

- Patient with Hypernatraemia due to pure water loss need free water; this may be given orally, or intravenously as 5% dextrose.
- The sodium overload can be treated with diuretics and the natriuresis replaced with water.

Other osmolality disorders:

- A high plasma osmolality may sometimes be encountered for reasons other than Hypernatraemia. Causes include:
  - Increased urea in renal disease
  - Hyperglycaemia in diabetes mellitus
  - The presence of ethanol or some other ingested substance
- And an increasing ECF concentration causes water to move out of cells and leads to intracellular dehydration.



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Clinical note:

Patients often become Hypernatremic because they are unable to complain of being thirsty. The comatose patient is a good example. He or she will be unable to communicate his/her needs, yet insensible losses of water will continue from lungs/skin and need to be replaced.

Potassium metabolism:

- Almost all is inside cells.
- Urinary potassium excretion rises in response to increased intake.
- The most important factor that regulates potassium excretion in the urine is the plasma potassium concentration.

Serum potassium:

- The small fraction (2%) of the total body potassium that is in the extracellular compartment is distributed proportionately between the interstitial and plasma spaces. Serum potassium concentration does not vary appreciably in response to water loss or retention.
- Cellular uptake of potassium is stimulated by insulin.
- Of particular importance is the reciprocal relationship between potassium and hydrogen ions. Many hydrogen ions are buffered inside cells. As the concentration of hydrogen ions increases with the development of acidosis, potassium ions are displaced from the cell in order to maintain electroneutrality.
- Despite its low concentration in the ECF, potassium determines the resting membrane potential of cells.

Hyperkalemia:

- Hyperkalemia causes muscle weakness that may be preceded by paraesthesiae.
- Above 7.0 mmol/l there is a serious risk of cardiac arrest. However, the ECG changes in hyperkalemia may mimic other conditions such as myocardial infarction.

Causes of hyperkalemia:

- Renal failure.
- Mineralocorticoid deficiency in Addison's disease or in patients receiving aldosterone antagonists: increases the total body potassium.
- Acidosis: Hyperkalemia results from a redistribution of potassium from the intracellular to the extracellular fluid space.
- Potassium release from damaged cells. Because of the very high potassium concentration inside cells, cells damage can give rise to a very high serum potassium

Pseudohyperkalemia:

- Pseudohyperkalemia refers to elevation in the measured potassium concentration resulting from potassium movement out of cells during or after the drawing of the blood specimen.
- The commonest cause is hemolysis.
- This can occur due to mechanical trauma during venepuncture.
- Pseudohyperkalemia should be suspected when there is no apparent cause for the hyperkalemia, and there are no ECG changes reflecting altered cardiac muscle contractility.

Treatment:

- The infusion of insulin and glucose to move potassium ions into cells.

- Dialysis is frequently necessary to treat severe hyperkalemia.
- When there is a slow rise in the plasma potassium this may be stopped or reversed by oral administration of a cation Resonium A.

Hypokalemia:

The main clinical effects of hypokalemia are:

- Severe muscle weakness
- Hyporeflexia
- Cardiac arrhythmias

The causes of hypokalemia include:

- Gastrointestinal losses (diarrhea).
- Renal losses – administration of diuretics or increased aldosterone production
- Drug-induced
- Alkalosis. An alkalosis may cause a shift of potassium from the ECF to ICF

Treatment:

- Potassium salts given prophylactically in an enteric coating.
- Severe potassium depletion often has to be treated by intravenous potassium.
- Intravenous potassium should not be given faster than 20 mmol/hour except in extreme cause and under ECG monitoring.

Intravenous fluid therapy:

Does this patient need IV fluid?

- The easiest and best way to give fluids is orally.
- However, patients may be unable to take fluids orally e.g. because the patient is comatosed, or has undergone major surgery, or is vomiting.
- Fluid depletion or electrolyte disturbance (more rapid than could easily be achieved orally).

Which IV fluids should be given ?

- Plasma, whole blood, or plasma expanders:
  - These replace deficits in the vascular compartment only.
  - They are indicated where there is a reduction in the blood volume due to blood loss from whatever cause.
- Isotonic sodium chloride (0.9% NaCl):
  - Is indicated where there is a reduced ECF volume as, for example in sodium depletion.
- Water:
  - If pure water were infused it would heamolyse blood cells as it enters the vein.
  - Water should instead be given as 5% dextrose (glucose), which, like 0.9% saline, is isotonic with plasma initially.
  - Five per cent dextrose is, therefore, designed to replace deficits in total body water e.g. in most hypernatraemic patients.

How much fluid should be given?

- Even where there is a severe deficit of water or sodium, it is important not to replace too quickly If complications of over-rapid correction are to be avoided.

- Most textbooks quote a water throughput of between 2, and 3 l daily, a sodium throughput of 100 to 200 mmol/day, and a potassium throughput that varies from 20 to 200 mmol/day.

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How quickly should the fluids be given?

A patient with trauma-induced diabetes insipidus can lose as much as 15 l urine daily (for example)

- Perioperative patient:
  Appropriate daily regimen should include between 2.0 and 3.0 l isotonic fluids, of which 1.0 l should be 0.9& saline with potassium supplement.

- Hyponatraemia:
  Patients with severe Hyponatraemia are vulnerable to demyelination if the serum sodium is raised acutely.