Perioperative electrolyte and fluid balance

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Appropriate fluid therapy is essential to protect organ function in the perioperative period. The physiological principles of fluid and electrolyte management are well described but a gap exists between knowledge and clinical practice. In this article, we will review fluid and electrolyte physiology, the stress response to surgery and hypovolaemia, and the consequences of electrolyte disturbances.

Fluid compartments

Total body water (TBW) is consistently 70% of the lean body weight across age and sex ranges but varies as a percentage of actual body weight between groups and individuals owing to varied deposition of adipose tissue, which contains less water than muscle. TBW is ~75% of total body weight in neonates, 70% in infants and 45% in the elderly. The average adult male (70 kg) has 60% of total body weight (42 litres) as water and an adult female (50 kg) has 55% (27 litres). This water is distributed between the extracellular (ECF) and intracellular (ICF) compartments.1

Extracellular fluid is, by definition, fluid outside the cells and comprises one-third (14 litres) of TBW. It is further subdivided into: (i) intravascular fluid (i.e. plasma), which fills the vascular system and, together with the red blood cells, constitutes the total blood volume, which is about 5% (3.5 litres) of body weight; and (ii) interstitial fluid, which is fluid outside the vascular system. It is mostly found in tissues adjacent to the microvascular circulation. More distant connective tissues, the so-called ‘third space’, remain relatively dry.

Intracellular fluid is contained within cells and comprises about two-thirds of TBW.

Transcellular fluids are fluids (total 0.6% of TBW) contained in body cavities, for example CSF, ocular, synovial, peritoneal and pleural fluids.

Electrolyte and water distribution

K⁺ and Na⁺ are the predominant cations in the ICF and ECF, respectively. Distribution and movement of water between the intracellular and interstitial spaces is governed by the osmotic forces created by differences in non-diffusible solute concentrations. The cell membrane separating the fluid compartments is selective and allows the free passage of water, but not solutes. Diffusion occurs by one of several mechanisms; directly through the lipid bilayer of the cell membrane, through protein channels within the membrane or by reversible binding to a carrier protein that can traverse the membrane (facilitated diffusion). Because of the charged lipid nature of the cell membrane, cations (such as Na⁺ and K⁺) cannot easily cross the membrane. These cations can diffuse only through specific voltage-dependent protein channels; thus, the cell transmembrane voltage potential (which is positive to the outside) created by the Na⁺-K⁺ pump is maintained. The solutions on each side of the cell membrane are therefore not identical and relative changes in osmolality between the intracellular and interstitial compartments result in a net water movement from the hypo-osmolar to the hyper-osmolar compartment.2

In contrast, the capillary endothelium is non-selective and freely permeable to both water and ions; plasma and interstitial fluids have similar solute compositions. Therefore, the major determinant of water flux is plasma protein concentration. Proteins do not normally pass out of the capillaries into the interstitium because of the tight intercellular junctions between adjacent endothelial cells. As a result, plasma proteins are the only osmotically active solutes in fluid exchange between plasma and interstitial fluid with albumin contributing 75% of the total colloid osmotic pressure (oncotic pressure). Compromising the integrity of the capillary membrane allows passage of albumin to the interstitial compartment and subsequent accumulation of tissue fluid.2

Key points

Understanding basic fluid and electrolyte physiology is essential to good perioperative fluid management.

The amount of Na⁺ in the extracellular fluid is the most important determinant of its volume.

Measurement of urine and plasma osmolalities helps in diagnosing electrolyte disturbances.

The characteristic response to anaesthesia and surgery is sodium and water retention.

Excess ADH secretion in the postoperative period is largely in response to hypovolaemia.
solute (i.e. Na\(^+\) and Cl\(^-\)). Because changes in Cl\(^-\) are mainly secondary to changes in Na\(^+\), the amount of Na\(^+\) in the ECF is the most important determinant of ECF volume; thus, the mechanisms that control Na\(^+\) balance are the major mechanisms defending ECF volume. However, the need to ensure optimal circulating volume is paramount and volume stimuli can override the osmotic regulation of vasopressin secretion. A rise in ECF volume inhibits vasopressin secretion, and a decline in ECF volume increases its secretion. In addition, expansion of ECF volume increases the secretion of natriuretic hormones, the most important being secretion of atrial natriuretic peptide (ANP) by the heart causing natriuresis and diuresis.\(^2\)

**Thirst and osmolality**

Thirst occurs in response to hypovolaemia (mediated via baroreceptors) and to changes in osmolarity detected by osmoreceptors in the hypothalamus. Drinking in response to thirst restores central circulating volume in hypovolaemia, ensures adequate hydration and allows additional fluid loss during the renal excretion of excess osmotic loads.

Total body osmolality is directly proportional to the total body sodium and potassium divided by TBW, so changes in osmolality occur when there is disproportionate change. When osmolality increases, the thirst mechanism is stimulated and vasopressin secretion (anti-diuretic hormone, ADH) is increased. Water is drunk and retained by the kidney, thus diluting the hypertonic plasma. Opposite effects occur when the plasma becomes hypotonic. Plasma osmolality ranges from 280 to 295 mosmol litre\(^{-1}\).\(^2\)

**The role of the kidney and renal sodium excretion**

While thirst and ADH control the intake and excretion of water, respectively, the electrolyte composition of urine is largely determined by renal mechanisms. Urinary sodium excretion is affected by changes in glomerular filtration rate (GFR) and tubular reabsorption of sodium. Decrease in intravascular volume causes a decrease in GFR and filtration of salt and water. Tubular reabsorption of sodium is affected by renal sympathetic tone, which results in diminished Na\(^+\) excretion and by the renin–angiotensin–aldosterone system, which plays a vital role in increasing sodium reabsorption and renovascular tone. Subsequent release of aldosterone from the adrenal cortex acts on the collecting ducts causing a further increase in sodium reabsorption.\(^3\)

**Water balance**

Normal balance is maintained with intake and losses of 2.5–3 litres per day. Intake from ingested fluid (1300 ml), solid food (800 ml) and metabolic waste (400 ml) is balanced by insensible fluid losses of 0.5 ml kg\(^{-1}\) h\(^{-1}\) (850 ml) from skin and lungs; plus sensible losses from urine (1500 ml) and faeces (100 ml). These values are those in health, at normothermia and at rest.

**Electrolyte balance and clinical implications**

**Sodium**

Sodium balance is related to ECF volume and water balance; daily ingestion has a wide range (50–300 mmol). It is regulated by the kidneys in which the volume and constitution of filtrate reaching the collecting ducts is dependent on GFR, sympathetic tone and angiotensin II acting via the effects of ADH and aldosterone to conserve water and sodium (see above). Normothermic extra-renal losses are minimal (~10 mmol day\(^{-1}\)).

Hypernatraemia leads to pyrexia, nausea, vomiting, convulsions, coma and focal neurological signs. Correction is advisable over 48–72 h with 5% dextrose. In hyponatraemia, symptoms depend on the cause, magnitude and rapidity. Acute symptomatic hyponatraemia is a medical emergency. The aim of treatment is to raise plasma concentration to 125 mmol litre\(^{-1}\) gradually over a period of no less than 12 h while treating the underlying cause.\(^1\)

**Potassium**

The total amount of potassium in the ECF is less than the average daily intake (50–200 mmol), so a potassium load must be cleared rapidly from this compartment. The physiological mechanisms that contribute to this are the release of both insulin and glucagon to increase intracellular transport and aldosterone release which stimulates the active transport of potassium from peritubular fluid into the cells of the distal convoluted tubule. K\(^+\) regulation is also inversely related to the pH. Potassium regulation is less efficient than sodium regulation and extra-renal losses are minimal. Hypokalaemia leads to anorexia, nausea, muscle weakness, paralytic ileus and cardiac conduction abnormalities. Treatment is with potassium supplements and treatment of the underlying causes. Hyperkalaemia results in cardiac arrhythmias which may be life threatening. Immediate treatment is necessary if plasma potassium concentration exceeds 7 mmol litre\(^{-1}\) or if there are serious ECG abnormalities. Treatment options include calcium gluconate, glucose and insulin, sodium bicarbonate, calcium resonium, and peritoneal or haemodialysis.\(^1\)

**Chloride**

Chloride is the main anion in the ECF. It is important in maintaining a normal acid–base state, normal renal tubular function and in the formation of gastric acid. Chloride loss is mainly from the stomach, bile, pancreatic and intestinal secretions. Regulation of chloride is passively related to sodium and inversely related to plasma bicarbonate. In the renal proximal tubule, chloride is excreted with ammonium ions to eliminate hydrogen ions in exchange for sodium and can result in the production of acid urine. In the erythrocytes, carbon dioxide is converted by the action of carbonic anhydrase to bicarbonate. About 70% of the bicarbonate produced will diffuse into the plasma and chloride shifts into the cell to maintain electrochemical neutrality.
The reverse occurs when the blood reaches the lungs. In respiratory disturbances (acidosis or alkalosis), 30% of an acid load can be buffered by such shifts between the ICF and ECF.4

It is important to recognise the clinical implication of excessive use of chloride (normal saline) in fluid resuscitation. According to Stewart,5 the major determinant to H+ concentration is the strong ion difference (SID) in the body. A normal SID (42-46 mmol litre−1) is obtained by adding together the concentrations of the main cations in solution (Na+, K+, Ca2+, Mg2+) and subtracting the concentrations of the main cations (Cl−, lactate).

A decrease in SID is associated with a metabolic acidosis and this can be precipitated by large volume of saline because renal excretion of Na+ occurs in preference to Cl− and H+. The cause of metabolic acidosis may be erroneously attributed to tissue hypoperfusion and cellular hypoxaemia and treating acidaemia with liberal volume infusions may worsen the acidosis rather than correcting it.5 6

Bicarbonate

Bicarbonate forms the main buffer and facilitates the carriage of carbon dioxide in the blood (80% as bicarbonate). Most of the filtered bicarbonate is reabsorbed in the proximal tubule as a result of H+ secretion from tubular cells into the lumen, the remainder is reabsorbed in the distal tubule and collecting ducts. Bicarbonate regulation is related to renal acid secretion; this in turn is altered by changes in PaCO2, K+ concentration, carbonic anhydrase level, and adrenal cortical hormone concentration. When PaCO2 is high (respiratory acidosis) more intracellular bicarbonate is available to buffer the hydroxyl ions and acid secretion is enhanced, whereas the reverse is true when PaCO2 falls. Acid secretion is also increased by aldosterone owing to increased reabsorption of Na+ and when there is K+ depletion because this causes intracellular acidosis even though the plasma pH may be elevated. Acid secretion is inhibited when carbonic anhydrase is inhibited because the formation of bicarbonate is decreased.

When the plasma bicarbonate concentration is high, it appears in urine, which becomes alkaline. Conversely, when the plasma bicarbonate falls, more H+ becomes available to combine with other buffer anions and the urine becomes more acidic.

The main implications of bicarbonate in perioperative fluid therapy are in the correction of metabolic acidosis and the emergency treatment of hyperkalaemia. However, over-treatment can lead to deleterious effects including an increase in 

Anaesthesia, surgery and fluid balance

Fluid shifts during the perioperative period and the physiological responses to surgical stress have significant implications for perioperative fluid prescribing. Many patients are dehydrated before theatre owing to prolonged fasting, the use of purgatives or diuretic therapy. Intraoperative losses are frequently underestimated and excess losses, both surgical and third-space losses, persist into the early postoperative period. Therefore, a general tendency towards hypovolaemia is usually present leading to thirst and vasopressin secretion.

There are two main components to the stress response to surgery: the neuroendocrine response and the cytokine response.7 The neuroendocrine response is stimulated initially by painfulafferent neural stimuli reaching the CNS. It may be diminished by dense neural blockade from regional anaesthesia. The cytokine response is stimulated by local tissue damage at the site of surgery itself (the more extensive the surgery the higher the response) and is independent of neural blockade.

The most important response to anaesthesia and surgery in the perioperative period is sodium and water retention. In general, the tendency to retain water is directly related to the magnitude of surgery. A number of factors may contribute to this including: the effects of anaesthetic agents on renal blood flow and GFR; effects of intraoperative hypotension or hypovolaemia on renal function; increased sympathetic tone and circulating catecholamines causing renal vasoconstriction; the salt and water retaining effects of increased plasma cortisol and aldosterone levels in response to the stress of surgery; and increased ADH activity. One of the most important of these is the increase in ADH activity. This is almost invariable; during surgery the ADH concentration may increase 50-100-fold. This concentration falls at the end of surgery but does not return to normal for 3-5 days (similar to the period of postoperative oliguria). This response is partly related to drugs, pain and other factors attributable to the stress of surgery; however, mostly it is a physiological response to the loss of intravascular fluid into cells or by its sequestration and immobilisation in damaged tissues (i.e. ‘third-space’). The difference is important because it determines the choice between fluid loading and fluid restriction as the most physiological approach to fluid therapy in the perioperative period.3

Changes in capillary membrane porosity occur during surgery largely as a result of the cytokine-mediated responses to tissue injury and bacteraemia. Despite a considerable increase in lymphatic drainage, fluid accumulates in previously ‘dry’ tissues. The situation often exists where circulatory hypovolaemia is significant enough to threaten organ perfusion whilst these ‘third-space’ tissues are waterlogged. More fluid is required in

<table>
<thead>
<tr>
<th>Substance (mmol litre−1)</th>
<th>Plasma</th>
<th>Interstitial fluid</th>
<th>Intracellular fluid</th>
<th>Gastric fluid</th>
<th>Intestinal fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>145</td>
<td>142</td>
<td>10</td>
<td>60</td>
<td>140</td>
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<tr>
<td>Potassium</td>
<td>4</td>
<td>4</td>
<td>140</td>
<td>&lt;1</td>
<td>5</td>
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<tr>
<td>Calcium</td>
<td>2.7</td>
<td>2.4</td>
<td>&lt;1</td>
<td>10</td>
<td>5</td>
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<tr>
<td>Magnesium</td>
<td>1</td>
<td>1</td>
<td>40</td>
<td>130</td>
<td>104</td>
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<tr>
<td>Chloride</td>
<td>112</td>
<td>117</td>
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<tr>
<td>Bicarbonate</td>
<td>25</td>
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<td>10</td>
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<td>30</td>
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<tr>
<td>Protein</td>
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<td>&lt;1</td>
<td>45</td>
<td></td>
<td></td>
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<tr>
<td>Phosphate</td>
<td>8</td>
<td>8.4</td>
<td>154</td>
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</tr>
</tbody>
</table>

*Phosphate, sulphate, fluoride, bromide.
Table 2 Common causes of electrolyte disturbance

<table>
<thead>
<tr>
<th>Disturbance</th>
<th>Abnormal intake</th>
<th>Abnormal distribution</th>
<th>Abnormal losses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypernatraemia</td>
<td>Salt gain; salt ingestion, excessive i.v. administration</td>
<td>Water depletion: diabetes insipidus, chronic renal failure, osmotic diuresis, fever, hyperventilation, decrease water intake, vomiting, diarrhoea or sweating</td>
<td>Na⁺ retention owing to corticosteroid excess</td>
</tr>
<tr>
<td>Na⁺ &gt;150 mmol litre⁻¹</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyponatraemia</td>
<td>Decrease intake, insufficient sodium during i.v. fluid therapy</td>
<td>Water retention: cardiac, hepatic, nephrotic. SIADH (syndrome of inappropriate ADH release), drugs, postoperative stress</td>
<td>Diuretic therapy, hypoadrenalism, salt losing nephropathy, renal tubular acidosis. ExTRANAL: diarrhoea, vomiting, third-space losses</td>
</tr>
<tr>
<td>Na⁺ &lt;135 mmol litre⁻¹</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperkalaemia</td>
<td>Blood transfusion, excessive i.v. administration</td>
<td>Release from cells: burns, rhabdomyolysis, intravascular haemolysis, suxamethonium</td>
<td>Impaired excretion: renal failure, Addison’s disease, potassium sparing diuretics</td>
</tr>
<tr>
<td>K⁺ &gt;5 mmol litre⁻¹</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypokalaemia</td>
<td>Dietary deficiency, K⁺ deficient i.v. administration</td>
<td>Insulin therapy, alkalaea, β₂-agonists</td>
<td>Renal loss: diuretics, hyperaldosteronism, renal artery stenosis, diuretic phase of acute renal fluid gastrointestinal loss: vomiting, diarrhoea, nasogastric suction, fistulae</td>
</tr>
<tr>
<td>K⁺ &lt;3.5 mmol litre⁻¹</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperchloraeia</td>
<td>Excessive i.v. NaCl 0.9% administration</td>
<td>Decreased renal excretion: renal tubular acidosis, uretero-iliostomy, diabetes insipidus, respiratory alkalosis</td>
<td>Severe dehydration</td>
</tr>
<tr>
<td>Cl⁻ &gt;106 mmol litre⁻¹</td>
<td></td>
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<td></td>
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<tr>
<td>Hypochloraeia</td>
<td></td>
<td>Respiratory alkalosis</td>
<td>Pyloric stenosis</td>
</tr>
<tr>
<td>Cl⁻ &lt;95 mmol litre⁻¹</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bicarbonate excess</td>
<td>Iatrogenic</td>
<td>Metabolic alkalosis</td>
<td>Chronic diuretic use, chronic vomiting and potassium loss</td>
</tr>
<tr>
<td>Bicarbonate deficit</td>
<td>Exogenous acids: HCl, NH₄Cl</td>
<td>Metabolic acidosis</td>
<td>Gastrointestinal loss: fistulae, diarrhoea, renal: renal tubular acidosis, carbonic anhydrase inhibitors</td>
</tr>
</tbody>
</table>

this situation to maintain circulatory volume and adequate organ perfusion.

**Urine biochemistry and diagnosis of electrolytes abnormalities**

The aetiology of important electrolyte disturbances is summarized in Table 2. Measurement of urine and plasma osmolalities and urine volume helps in the diagnosis.

**Hypernatraemia**

If urine output is low and urine osmolality high, then both ADH secretion and the renal response to it are present. The cause here is most likely extrarenal water loss. If both urine output and urine osmolality are high, then osmotic diuresis is suspected. If urine osmolality is less than plasma osmolality, then the cause is attributable to reduced ADH secretion or abnormal renal response to ADH; in both cases, urine output is high.³

**Hyponatraemia**

If hypovolaemia is present, then the loss is either renal (urine sodium >20 mmol litre⁻¹) or extrarenal (urine sodium <15 mmol litre⁻¹); in these conditions, saline is required. If hypervolaemia (oedema) is present, the cause could be cardiac, hepatic or renal (nephrotic) and urine sodium is usually <20 mmol litre⁻¹. However, if hyponatraemia is associated with normovolaemia, the most common causes are SIADH (syndrome of inappropriate ADH release), postoperative stress, drugs and renal failure; plasma osmolality is usually decreased and fluid restriction is required.¹

**References**


See multiple choice questions 119–122