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The positives and negatives of preoperative electrolyte disturbances

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Introduction

The physiological principles of fluid and electrolyte management are well described. However, a gap exists between the theoretical knowledge and clinical practice, especially with respect to postponement of surgery. This article seeks to address the recommendations with respect to the most common electrolyte abnormalities that may result in postponement of surgery (namely sodium, potassium, calcium and magnesium). Patients are subject to large fluid shifts in theatre, and postoperatively. Surgical patients often present with acute or chronic renal injury, they are often sedated, fasted, and may have intravenous fluid infusions prescribed for extended periods. Preoperative bowel obstruction or bowel preparation can result in profound dehydration, and therefore electrolyte abnormalities occur commonly.[1-3] The electrolyte composition of body fluids is presented in Table I.

Table I: Electrolyte composition of body fluids[1]

Substance	Plasma (mmol/l)	Interstitial fluid (mmol/l)	Intracellular Fluid (mmol/l)
Sodium	145	142	10
Potassium	4	4	140
Calcium	2.7	2.4	< 1
Magnesium	1	1	40

Sodium

Sodium (Na^+) balance is related to extracellular fluid (ECF) volume and water balance. The common causes of sodium abnormalities are presented in Table II. Consideration of the volume status of patients with sodium abnormalities is essential. Causes can be defined as those relating to hypovolaemia, euvolaemia, and hypervolaemia.[1,2]

Hypernatraemia

Hypernatraemia is associated with the development of pyrexia, nausea, vomiting, convulsions, coma, and focal neurological signs. Hypernatraemia is either caused by excessive salt intake, or (much more frequently) inadequate water intake. As with hyponatraemia, consideration of the volume status of the patient is essential.[1-3]

In general, it is recommended that elective surgery by postponed in patients with significant hypernatraemia (> 150 mmol/l) until the cause is established and fluid deficits corrected or until symptoms have resolved.[4-7] Correction is advisable over 48–72 hours.[1-3] Firstly, any volume deficit should be corrected, with 0.9% saline until the hypovolaemia, as measured by orthostatic hypotension improves. The cause of fluid loss should also be investigated and treated.[1]

Table II: Common causes of sodium disturbances[1,2]

Disturbance	Cause			
	Hypovolaemic (renal)	**Hypovolaemic (extrarenal)**	**Euvolaemic**	**Hypervolaemic**
Hypernatraemia (Na^+ > 150 mmol/l)	• Loop or osmotic diuretics • Post obstruction • Intrinsic renal disease	• Vomiting • Diarhoea • Burns • Excessive sweating • Fistulae	• Diabetes Insipidus • Insensible losses	• Sodium ingestion • Conn's syndrome • Cushing's syndrome • Administration of hypertonic saline or $NaHCO_3$ • Dialysis against hypertonic dialysate
Hyponatraemia (Na^+ < 135 mmol/l)	• Diuretic excess • Mineralo-corticoid excess • Salt-wasting nephropathy • Proximal renal tubular acidosis • Ketonuria • Osmotic diuresis	• Vomiting • Diarrhoea • Burns • Pancreatitis • Trauma	• Glucocorticoid deficiency • Hypothyroidism • Syndrome of inappropriate ADH secretion (SIADH) • Drugs (acting via ADH pathway) • Psychogenic polydipsia • Hypotonic fluid replacement post-operatively	• Acute or chronic renal failure • CCF • Nephrotic syndrome • Cirrhosis

The total body water deficit can be calculated based on the serum sodium and the assumption that 60% of the body is water. This deficit should then be corrected with 5% dextrose, with half given in the first 12–24 hrs, and the rest over the next 24–36 hrs.[1-3] The equations used to calculate total body water deficit and the change in serum Na^+ are shown in Figure 1.

Free Water Deficit = [(serum Na^+ - 140)/140 X Total Body Water (TBW)]
TBW = 0.6 X Body Weight (kg)
Calculating the effect of 1L of an intravenous solution on serum sodium =
Change in serum Na^+ = (Na^+ infused – Na^+ serum)/ (TBW +1)

Figure 1: Equations for calculating Free Water Deficit, Total Body Water, and the change in serum Na^+ after administering an intravenous solution[1-3]

The following is recommended should emergency surgery be required:

- Frequent perioperative sodium measurements should be made.
- Invasive blood pressure (BP) monitoring as hypovolaemia will accentuate any vasodilation or cardiac depression from anaesthetic agents, predisposing to hypotension and hypoperfusion.
- Additional fluid administration, vasopressors, or inotropes may be required.
- Careful titration of intravenous agents. Consider reduction of doses of most intravenous agents due to decreases in the volume of distribution. However, as a result of a reduction in cardiac output, uptake of inhalational anaesthetics may be enhanced.[4-6]

Hyponatraemia

Acute symptomatic hyponatraemia is considered a medical emergency. The normal range of serum sodium is usually 135–145 mmol/l, however, levels between 125 mmol/l and 150 mmol/l are often asymptomatic. Outside this range there is an increasing frequency of nausea, lethargy, weakness, and confusion. Levels above 160 mmol/l and below 110 mmol/l are strongly associated with seizures, coma, and death.[1,2,7]

As serum sodium and osmolarity fall, water tends to enter the cells resulting in oedema. Clinically this is most important in the brain. Several factors put patients at increased risk of complications of hyponatraemia or its treatment:

- Postoperative patients, premenopausal women, elderly women taking thiazides, children, and patients who are hypoxaemic are all at increased risk of acute hyponatraemic cerebral oedema.
- Malnourished patients, alcoholics, those with burns or hypokalaemia are all at increased risk of osmotic myelinolysis due to overly rapid correction of hyponatraemia.[1,2,7]

Cancelling of elective surgery is in general considered if patients are symptomatic or if plasma Na^+ is found to be less than 120 mmol/l. Generally it is considered that a plasma sodium greater than 130 mmol/l is safe for general anaesthesia.[1,2,7]

Management depends on the speed of development of the abnormality and therefore it is important to distinguish between hyponatraemia that has developed acutely (usually taken to mean over < 48 hrs) and chronic hyponatraemia.[1,2,7]

The aim of treatment is to raise plasma concentration to 125 mmol/l gradually over a period of no less than 12 hours while addressing the underlying cause.[1]

The treatment of chronic hyponatraemia is determined by the presence or absence of symptoms. In the presence of symptoms, a rapid correction of up to 10 mmol/l may be permissible. Following this, however, the rate of reversal should be limited to 1.5 mmol/l/hr, and to 8.0 mmol/l over 24 hrs. Some sources suggest that a rate of 12 mmol/L in 24 hrs may be safe.[1,2,7]

Fluid restriction is the mainstay of treatment in these patients, who need to have regular neurological assessment and rechecking of serum electrolytes at least every 12 hrs. In the long term, treatment is aimed at identifying and dealing with the underlying cause.[1,2,7]

Hyponatraemia that has developed acutely (for instance, in the immediate postoperative period) can be safely treated with rapid correction. Rapid correction should only be undertaken in patients who are symptomatic, and the aim of treatment is to correct the level until the symptoms resolve. Some authors have suggested that correction by up to 2.0 mmol/l/hr is safe in the initial treatment of acute hyponatraemic states. Correction to a serum Na^+ of > 135 mmol/l may be safe in this situation, but it is not necessary to correct rapidly once the symptoms have resolved. Methods of rapid correction might include the administration of furosemide and/or hypertonic saline, but in this case management should be by a specialist in an appropriate setting, with monitoring of serum Na^+ levels hourly.[1,2,7]

In all cases, hypovolaemia if present, must be corrected first with 0.9% saline. This removes the ADH response that is accentuating the sodium/water imbalance. In patients who are hypervolaemic, the treatment is aimed at fluid restriction, salt restriction, and loop diuretics. While evidence is lacking that chronic hyponatraemia is associated with worse surgical outcomes, anything more than mild, asymptomatic hyponatraemia should be regarded as a relative contraindication to elective surgery.[1,2,7]

Consideration of the osmotic state and volume status of the patient is essential in the evaluation and management of hyponatraemia. The causes of altered volume states are outlined in Table III.

Potassium

Hypokalaemia

The effects of hypokalaemia depend upon the serum level. A normal value of 3.5–4.5mmol/L is generally considered acceptable. Levels of 3.0–3.5 mmol/L are usually asymptomatic,

Table III: Causes of altered volume states in patients with hyponatraemia[1,2,7]

Hypovolaemic hyponatraemia	Euvolaemic hyponatraemia	Hypervolaemic hyponatraemia
• Loss of both sodium and water, but proportionately more sodium. • Caused by solute and water losses from either a renal or gastrointestinal source. • Usually these patients are consuming water or hypotonic fluid, although not in quantities sufficient to restore normovolaemia. • An estimation of the urinary sodium level can be helpful: a level below 30 mmol/L suggests an extrarenal cause, while a level above 30 mmol/L suggests a primary renal problem.	• The most common form seen in hospitalised patients. • May have a slight increase or decrease in volume, but it is not clinically evident, and they do not have oedema. • The most common cause is the inappropriate administration of hypotonic fluid. • The syndrome of inappropriate ADH secretion (SIADH) also causes euvolaemic hyponatraemia; in order to make this diagnosis one must first exclude renal, pituitary, adrenal or thyroid dysfunction, and the patient must not be taking diuretics.	• Characterised by both sodium and water retention, with proportionately more water. • Causes are all characterised by disordered water excretion, and are usually easy to diagnose.

but below 3.0 mmol/L general symptoms of weakness, lassitude, and constipation are common. Below 2.5 mmol/L muscle necrosis has been described, and below 2.0 mmol/L an ascending paralysis may be seen, eventually leading to respiratory compromise.[1,2,8]

The therapeutic goals with respect to hypokalaemia are as follows:

- To prevent life-threatening complications (arrhythmias, respiratory failure).
- To correct potassium (K^+) deficit.
- To minimise on-going losses through the treatment of an underlying cause.
- To identify and correct hypomagnesaemia to allow effective K^+ repletion.[1,2,4-6,8]

The methods used to calculate the K^+ deficit are presented in Figure 2.

Old formula:

K^+ deficit = (desired – actual)/ 0.27 X 100
= (3.5 – 2.8)/ 0.27 X 100
= 260 meqs

New paradigm:

< 2.0 = 600 meqs deficit
2.0-3.0 = 400 meqs deficit
3.0-4.0 = 200 meqs deficit

Figure 2: The methods used to calculate potassium deficit[1,2,8]

The clinical risk and decision to treat perioperative hypokalaemia is controversial and should take into account the chronicity and severity of the deficit, especially as the administration of K^+ may result in harm as a result of administration errors or due to diminished potassium regulation in patients with diabetes or renal failure. Evaluation of the risk benefit ratio is essential. Generally, it is considered that there is no increased risk of morbidity or mortality in patients undergoing surgery with a K^+ > 2.6 mmol/l. However, should patients have other clinical risk factors, e.g. cardiac failure or digoxin therapy, correction prior to surgery may be critical. In these cases plasma K+ > 4.0 mmol/l are recommended as hypokalaemia may predispose patient to digoxin toxicity, heart blocks, supraventricular tachycardia, or other arrhythmias.[1,2,8]

Therefore, the decision to delay elective surgery should be based on the:

- Rate at which hypokalaemia developed (chronicity is suggested by an elevated bicarbonate).
- Presence of secondary organ dysfunction.
- Level of hypokalaemia.
- Urgency of surgery.[1,2,4-6]

Intraoperative management of these patients should include:

- Vigilant ECG monitoring.
- Intravenous replacement of K^+ should arrhythmias develop.
- Administration or application of insulin, glucose, bicarbonate, β-agonists, diuretics and/or hyperventilation induced respiratory alkalosis to avoid worsening hypokalaemia.
- Reduction in the dosages of non-depolarising muscle relaxants (NDMBs), and the use of neuromuscular monitoring as an increased sensitivity to NDMBs may occur.[1,2,4-6]

Hyperkalaemia

Hyperkalaemia results in cardiac arrhythmias which may be life threatening. Immediate treatment is necessary if plasma potassium concentration exceeds 7.0 mmol/l, or if there are serious ECG abnormalities. The level at which hyperkalaemia should be treated is controversial, but generally recommendations tend to be conservative. Although some authors recommend the treatment of hyperkalaemia when K^+ is greater than 6.0 mmol/l, it is recommended that serum potassium levels should be below 5.5 mmol/l for elective surgery.[1,2] Clinical signs and symptoms sometimes correlate poorly with plasma concentrations, therefore there is little evidence on which to suggest therapeutic thresholds.[1,2]

Intraoperative management should include:

- Meticulous ECG monitoring.
- Avoidance of succinylcholine administration.
- Avoidance of metabolic or respiratory acidosis (may increase the shift of potassium extracellularly).
- Avoidance of the infusion of potassium-containing solutions.

- Neuromuscular monitoring (hyperkalaemia may accentuate the effect of NDMBs).[1,2,4-6,8]

Hyperkalaemia generally requires treatment with insulin, dextrose, and sodium bicarbonate prior to arrival at theatre. Dextrose 1 g/kg IV over 15 minutes with 0.2 unit insulin/kg, and calcium chloride 4–5 mg/kg IV over 5–10 minutes is administered to stabilise the cardiac membrane. The aggressiveness of therapy for hyperkalaemia is directly related to the rapidity with which the condition has developed, the absolute level of serum potassium, and the evidence of toxicity. The rate of rise in the potassium level, the higher the level, and the greater the evidence of cardiotoxicity, the more aggressive therapy should be.[1,2,8]

Calcium

Calcium (Ca^{2+}) is the key component in a multitude of physiological functions including:

- Muscular contraction (smooth, skeletal, and cardiac).
- Intracellular secondary messenger systems controlling exocrine, endocrine and neuroendocrine secretion, cellular growth, and fluid and electrolyte transport and secretion.
- Coagulation.[1,2,9]

Normal plasma and ionised calcium levels are 2.12–2.62 mmol/l and 1.15–1.30 mmol/l respectively. Total Ca^{2+} is usually reported, but only free calcium is active. Therefore, the patients' plasma albumin levels and acid-base status should be taken into account. Acid-base changes can also cause a change in the albumin binding of Ca^{2+}. An increased pH can lead to increased protein ionisation resulting in less free Ca^{2+}.[1,2]

Methods used to calculate corrected Ca^{2+} are shown in Figure 3.

A. Corrected Ca^{2+}
- Ca^{2+} changes by 1.0 mmol/l for every 6g/l that albumin deviates from 40g/l
- If albumin is less than 40g/l add 0.1 mmol/l to the Ca^{2+} level.
- If albumin is more than 40g/l subtract 0.1 mmol/l from the Ca^{2+} level

B. Corrected Ca^{2+} (mmol/L) = Ca^{2+} measured (mmol/L) + 0.020(40 - albumin (g/L))].

C. Corrected calcium (mg/dL) = measured total Ca^{2+} (mg/dL) + 0.8 (4.0 - serum albumin [g/dL]).

mg/dl = 18 X mmol/l

Figure 3: Methods used to calculate corrected calcium[9,10]

Hypercalcaemia

Hypercalcaemia should be corrected prior to anaesthesia, and should include restoration of intravascular volume. Should emergency surgery be required, the following is recommended:

- Close haemodynamic monitoring.
- Monitoring of ionised calcium levels.
- Saline diuresis.
- Avoidance of hypovolaemia.
- Controlled ventilation to avoid respiratory acidosis (acidosis reduces calcium binding to albumin thus increasing the ionised calcium fraction).[4-6]

Hypocalcaemia

Life threatening complications frequently occur if ionised calcium levels drop below 0.5 mmol/l. Neuromuscular irritability causing laryngeal spasm and bronchospasm may develop. Hypocalcaemia can cause hypotension, cardiac failure, and bradycardia. Hypocalcaemia may result in digoxin insensitivity. Cardiac irritability resulting in arrhythmias (premature ventricular contractions and ventricular fibrillation) may occur. Only very low ionised calcium concentrations impair coagulation.

Symptomatic hypocalcaemia should be treated preoperatively and further decreases in ionised calcium intraoperatively should be avoided. Should emergency surgery be required the following is recommended:

- Increased awareness of the potential for exacerbation of myocardial depression and hypotension due to additive effects of anaesthetic agents.
- Neuromuscular monitoring due to prolonged action of NDMBs.
- Prevention of alkalosis and a further decrease in ionised calcium.[4-6]

Correction of significant hypocalcaemia should be titrated against measured plasma ionised calcium concentrations.[1,2]

Magnesium

Magnesium (Mg^{2+}) is an essential electrolyte that is involved in a large number of enzymatic reactions and contributes to homeostasis, membrane stability, cell division, and generation of action potentials. Mg^{2+}deficiency is often associated with hypocalcaemia. The normal serum magnesium concentration is considered to be 0.7–1.00 mmol/l.[11,12]

Hypomagnesaemia

Treatment depends on the severity and clinical status. Replacement is guided by serum magnesium levels, although serum magnesium levels are a poor reflection of adequacy of replacement as most magnesium is intracellular. Severe or symptomatic deficiency may be treated by a slow infusion of 2 g over 30 min. Patients with concomitant hypokalaemia or hypocalcaemia should also receive potassium and calcium replacement, because these disorders may take several days to correct when treated with Mg^{2+} alone. In the presence of hypocalcaemia, tetany can occur during the administration of magnesium sulphate if calcium is not supplemented, as ionised calcium levels can drop acutely from complexing of calcium with sulphate ions and increased urinary excretion.[4-6,11,12]

Summary

Surgical patients are frequently affected by electrolyte imbalances. Anaesthetic management with respect to fluid

and electrolyte derangements is complex, and requires careful consideration. In deciding when and how to treat a patient, a balance must be struck between the risks of the condition, and the risks of treatment. The anaesthetist must be cognisant of the general underlying condition of the patient, the cause of the electrolyte abnormality, the urgency of the surgery, the risks pertaining the electrolyte abnormality, the treatment and the speed at which the abnormality has been corrected, the risks of such treatment, the time needed for correction of the abnormality, and the risks related to delaying surgery. In general, the implications of increased or decreased serum electrolyte levels are dependent on the speed with which this change occurred.

Declaration of interests

No interests to declare

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