Fluid and electrolyte management

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Learning objective

- To understand the distribution and composition of body fluids, and how these may change following surgery
- To understand types of intravenous fluid therapy and common electrolyte disorders

The management of a patient' s fluid status is vital to a successful outcome in surgery. This requires preoperative assessment, with resuscitation if required, and postoperative replacement of normal and abnormal losses until the patient can resume a normal diet.

Body fluid compartments (Figure 2.1)

In the ' average ' person, water contributes 60% to the total body weight: 42 L for a 70 kg man. 40% of the body weight is intracellular fluid, while the remaining 20% is extracellular. This extracellular fluid can be subdivided into intravascular (5%) and extravascular, or interstitial (15%). Fluid may cross from compartment to compartment by osmosis, which depends on a solute gradient, and filtration, which is the result of a hydrostatic pressure gradient.

The electrolyte composition of each compartment differs. Intracellular fluid has a low sodium and a high potassium concentration. In contrast, extracellular fluid (intravascular and interstitial) has a high sodium and low potassium concentration. Only 2% of the total body potassium is in the extracellular fluid. There is also a difference in protein concentration within the extracellular compartment, with the interstitial fluid having a very low concentration compared with the high protein concentration of the intravascular compartment.

Knowledge of fluid compartments and their composition becomes very important when considering fluid replacement. In order to fill the intravascular compartment rapidly, a plasma substitute or blood is the fluid of choice. Such fluids, with high colloid osmotic potential, remain within the intravascular space, in contrast to a saline solution, which rapidly distributes over the entire extravascular compartment, which is four times as large as the intravascular compartment. Thus, of the original 1 L of saline, only 250 mL would remain in the intravascular compartment. 5% dextrose, which is water with a small amount of dextrose added to render it isotonic, will redistribute across both intracellular and extracellular spaces.

Osmolality refers to the number of osmoles of solute particles per kilogram of water. Total osmolality is comprised of both effective and ineffective components. Effective osmoles cannot freely permeate cell membranes and are restricted to either the intracellular or extracellular fluid compartments. The asymmetric accumulation of effective osmoles in either extracellular fluid (e.g., Na+, glucose, mannitol, and glycine) or intracellular fluid (e.g., K+, amino acids, and organic acids) causes transcompartmental movement of water.

Because the cell membrane is freely permeable to water, the osmolalities of the extracellular and intracellular compartments are equal. The effective osmolality of a solution is equivalent to its tonicity. Ineffective osmoles, in contrast, freely cross cell membranes and therefore are unable to affect the movement of water between compartments.

Plasma osmolality= 2[Na+] + [Glucose]/18 + [BUN]/2.8

The normal range is 280-290 mOsm/L

Fluid and electrolyte losses

In order to calculate daily fluid and electrolyte requirements, the daily losses should be measured or estimated. Fluid is lost from four routes:

the kidney, the gastrointestinal tract, the skin and the respiratory tract. Losses from the last two routes are termed insensible losses.

Table 2.1 Normal daily fluid losses

Fluid loss	Volume (mL)	Na^+ (mmol)	K^+ (mmol)
Urine	2000	80–130	60
Faeces	300		
Insensible	400		
Total	2700		

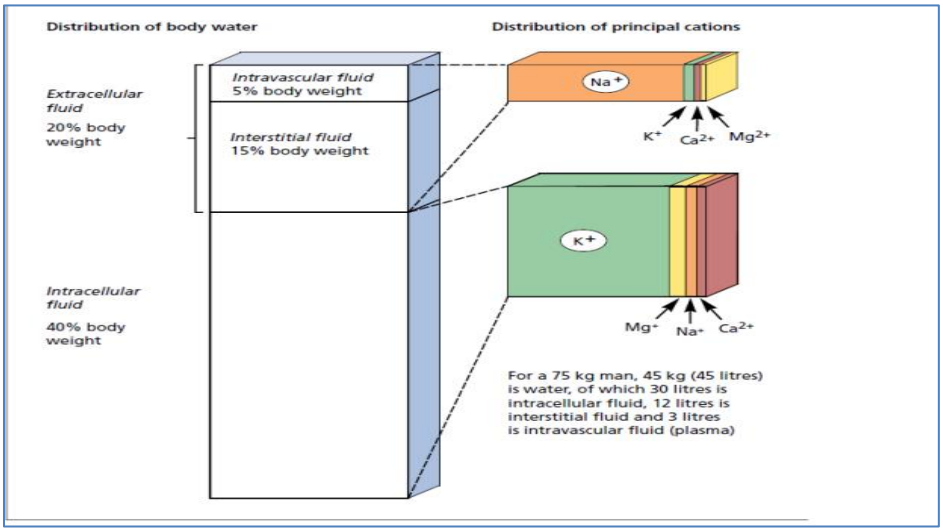

Figure 1: Distribution of fluid and electrolyte in body

Effects of surgery

ADH is released in response to surgery, conserving water. Hypovolaemia will cause aldosterone secretion and salt retention by the kidney.

Potassium is released by damaged tissues, and the potassium level may be further increased by blood transfusion, each unit containing in excess of 20 mmol/L. If renal perfusion is poor, and urine output sparse, this potassium will not be excreted and instead accumulates, the resultant hyperkalaemia causing life - threatening arrhythmias.

This is the basis of the recommendation that supplementary potassium may not be necessary in the first 48 hours following surgery or trauma.

Prescribing fluids for the surgical patient

The majority of patients require fluid replacement for only a brief period postoperatively until they resume a normal diet. Some require resuscitation preoperatively, and others require replacement of specific losses such as those from a fistula.

In severely ill patients, and those with impaired gastrointestinal function, long - term nutritional support is necessary.

Types of intravenous fluids

The fluids used in clinical practice are usefully classified into colloids and crystalloids.

1-Colloids: solutions that contain large molecules that can't pass through gap junctions of blood vessels.

When infused, they remain in the intravascular compartment and expand it and they draw fluid from extravascular spaces via their higher oncotic pressure.

Indications:

- Rapid replacement of intravascular fluid (so called plasma expander)
- Correct albumin and protein level

Examples: albumin, plasma, blood and its products, dextran.

2- Crystalloids: solutions contain small molecules that flow easily across cell membranes, allowing for transfer from the bloodstream to the cells and tissues. This will increase fluid volume in both the interstitial and intravascular spaces.
It is divided according to osmolarity into:

- Isotonic :ex. 0.9% NaCl, lactate ringer , 5% dextrose
- Hypotonic: 0.45%NaCl
- Hypertonic: D5 in 0.9% normal saline.

INDICATIONS OF FLUID THERAPY

I. Fluid resuscitation

Patients who are in hypovolemic shock require rapid fluid infusions in the form of fluid challenges to maintain intravascular volume. Rapid infusion of a 1000 mL bolus of normal (isotonic) saline (NS) or lactated Ringer's solution (RL) within 15 minutes.

II. Replacement of free water deficit

Indicated to treat dehydration and/or hypernatremia
Free water deficit (in liters) = 0.6 × weight (kg) × (Current [Na+]/140 – 1)

- Intravenous fluids that can be used to replace free water deficit 5% dextrose Hypotonic saline (e.g., ½NS, ¼NS)

III. Replacement of ongoing fluid loss

Fluids are also indicated in the post-resuscitation phase when the patient is no longer hypovolemic but still has ongoing abnormal fluid loss that cannot be compensated for by oral intake alone. Some common conditions associated with an ongoing fluid loss are:

- Burns
- Polyuria (high output renal failure, diabetes insipidus)
- Surgical drainage
- Significant ongoing gastrointestinal loss (vomiting, diarrhea)

Table 2.2 Electrolyte content of intravenous fluids

Intravenous infusion	Na^+ (mmol/L)	Cl^- (mmol/L)	K^+ (mmol/L)	HCO_3^- (mmol/L)	Ca^{2+} (mmol/L)
Normal saline (0.9% saline)	150	150	–	–	–
4% dextrose/ 0.18% saline	30	30	–	–	–
Hartmann's (compound sodium lactate)	131	111	5	29	2
Normal plasma values	134–144	95–105	3.4–5.0	22–30	2.2–2.6

Table 2.3 Daily volume and composition of gastrointestinal fluids

Fluid	Volume (mL)	Na^+ (mmol/L)	K^+ (mmol/L)	Cl^- (mmol/L)	H^+/HCO_3^- (mmol/L)
Gastric	2500	30–80	5–20	100–150	H^+ 40–60
Bile	500	130	10	100	HCO_3^- 30–50
Pancreatic	1000	130	10	75	HCO_3^- 70–110
Small bowel	5000	130	10	90–130	HCO_3^- 20–40

IV. Maintenance fluid therapy

Maintenance fluids are indicated in patients who cannot or are not allowed to take fluids orally. The most commonly used formula is Holliday-segar method which is 4-2-1 or 100-50-20.

- 100 ml/kg/24-hours = 4 ml/kg/hr for the 1st 10 kg
- 50 ml/kg/24-hours = 2 ml/kg/hr for the 2nd 10 kg

- 20 ml/kg/24-hours = 1 ml/kg/hr for the remainde

COMMON ELECTROLYTE DISORDERS

A. ***Sodium***

Physiology: The normal individual consumes 3 to 5 g of NaCl (130 to 217 mmol Na+) daily. Sodium balance is maintained primarily by the kidneys. Normal Na+ concentration is ***135 to 145 mmol/L*** (310 to 333 mg/dL). Potential sources of significant Na+ loss include sweat, urine, and gastrointestinal secretions.

1- Hyponatremia

I. ***Causes and diagnosis.*** The diagnostic approach to hyponatremia is illustrated in Figure 4-2.

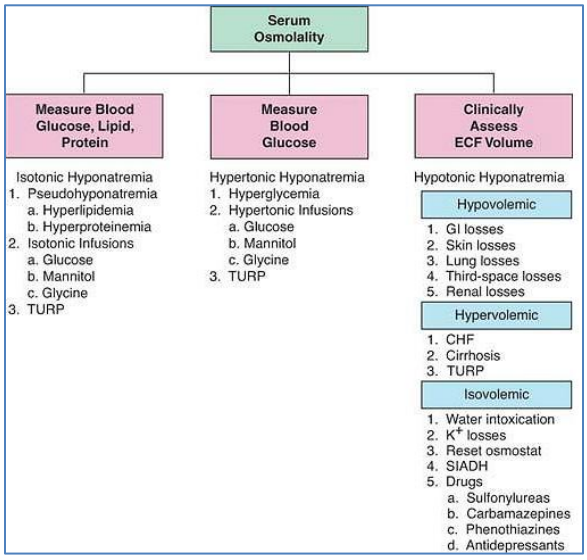

Figure 4-2

II. ***Clinical manifestations***. Symptoms associated with hyponatremia are predominantly neurologic and result from hypoosmolality. A decrease in Posm causes intracellular water influx, increased intracellular volume, and cerebral edema. Symptoms include lethargy, confusion, nausea, vomiting, seizures, and coma.

III.***Treatment***

1-Isotonic and hypertonic hyponatremia correct with resolution of the underlying disorder.

2-Hypovolemic hyponatremia can be managed with administration of 0.9% NaCl to correct volume deficits and replace ongoing losses.

3-Water intoxication responds to fluid restriction (1,000 mL/day).

4-For Syndrome of inappropriate antidiuretic hormone secretion (SIADH), water restriction (1,000 mL/day) should be attempted initially. The addition of a loop diuretic (furosemide) or an osmotic diuretic (mannitol) may be necessary in refractory cases.

5-Hypervolemic hyponatremia may respond to water restriction (1,000 mL/day) to return Na+ to greater than 130 mmol/L.

6-In the presence of symptoms or extreme hyponatremia [Na^+ <110 mmol/L], hypertonic saline (3% NaCl) is indicated.

2-Hypernatremia

i. ***Diagnosis***: Hypernatremia is uniformly hypertonic and typically the result of water loss in excess of solute. Patients are categorized on the basis of their extracellular fluid volume status. The diagnostic approach to hypernatremia is illustrated in Figure 4-3.

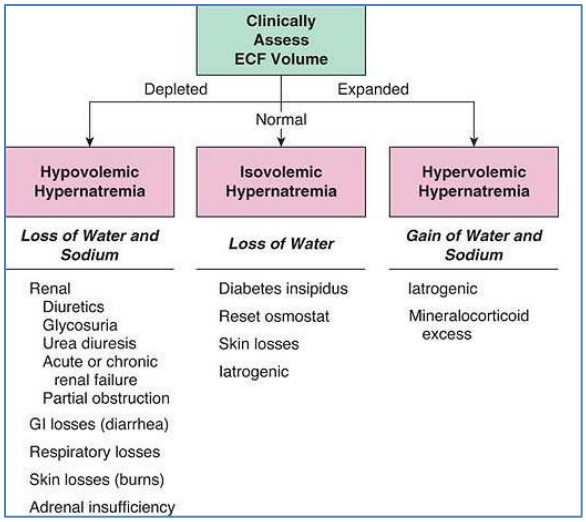

Figure 4-3

ii. ***Clinical manifestations***. Symptoms of hypernatremia that are related to the hyperosmolar state are primarily neurologic. These initially include lethargy, weakness, and irritability and may progress to fasciculations, seizures, coma, and irreversible neurologic damage.

iii. Treatment

1.Water deficit associated with hypernatremia can be estimated using the following equation where TBW = Total body weight:

TBW deficit(L)=0.6×(body weight kg)×([Na]/140]-1)

Rapid correction of hypernatremia can result in cerebral edema and permanent neurologic damage. Consequently, only one half of the water deficit should be corrected over the first 24 hours, with the remainder being corrected over the following 2 to 3 days. Serial Na+ measurements are necessary to ensure that the rate of correction is adequate but not excessive. Oral fluid intake is acceptable for replacing water deficits. If oral intake is not possible, D5 W or D5 0.45% NaCl can be substituted. In addition to the actual water deficit, insensible losses and urinary output must be replaced.

2.Diabetes insipidus

Central diabetes insipidus can be treated with desmopressin acetate administered intranasally [0.1 to 0.4 mL] or subcutaneously or intravenously [0.5 to 1 mL daily].

Nephrogenic diabetes insipidus treatment requires removal of any potentially offending drug and correction of electrolyte abnormalities. If these measures are ineffective, dietary sodium restriction in conjunction with a thiazide diuretic may be useful (hydrochlorothiazide, 50 to 100 mg/day orally).

B. Potassium

Physiology: K+ is the major intracellular cation, with only 2% of total body K+ located in the extracellular space. The normal serum concentration is 3.3 to 4.9 mmol/L (12.9 to 19.1 mg/dL). Approximately 50 to 100 mmol K+ is ingested and absorbed daily. Ninety percent of K+ is renally excreted, with the remainder eliminated in stools.

1.Hypokalemia

I. Causes. K+ depletion from inadequate intake alone is rare. Common causes of K+ depletion in the surgical patient include GI losses (e.g., diarrhea, persistent vomiting, and nasogastric suctioning), renal losses (e.g., diuretics, fluid mobilization, and amphotericin B), and cutaneous losses (e.g., burns). Other causes of hypokalemia include acute intracellular K+ uptake (associated with insulin excess, metabolic alkalosis, myocardial infarction, delirium tremens, hypothermia, and theophylline toxicity).

II.Clinical manifestations. Mild hypokalemia [K+ >3 mmol/L] is generally asymptomatic.

Symptoms occur with severe K+ deficiency [K+ <3 mmol/L)] and are primarily cardiovascular (arrhythmia). Early electrocardiogram (ECG) manifestations include T-wave flattening or inversion, ST-depression and less frequently prolong PR interval.

III.Treatment. identify and correct the cause. In mild hypokalemia, oral replacement 40 to 100 mmol (156 to 390 mg) KCl orally in single or divided doses is suitable.

2.Hyperkalemia

I.Causes and diagnosis. Hyperkalemia may occur with normal or elevated stores of total body K+. Pseudohyperkalemia is a laboratory abnormality that reflects K+ release from leukocytes and platelets during coagulation. Spurious elevation in K+ may result from hemolysis. Abnormal redistribution of K+ from the intracellular to the extracellular compartment may occur as a result of insulin deficiency, β-adrenergic receptor blockade, acute acidemia, rhabdomyolysis, cell lysis (after chemotherapy), digitalis intoxication, reperfusion of ischemic limbs, and Addison syndrome.

II.Clinical manifestations. Mild hyperkalemia [K+ = 5 to 6 mmol/L (19.5 to 23.4 mg/dL)] is generally asymptomatic. Signs of significant hyperkalemia [K+ >6.5 mmol/L] are, most notably, ECG abnormalities: symmetric peaking of T waves, reduced P-wave voltage, and widening of the QRS complex. If untreated, severe hyperkalemia ultimately may cause a sinusoidal ECG pattern.

III.Treatment

Mild hyperkalemia [K+ = 5 to 6 mmol/L] can be treated conservatively by the reduction in daily K+ intake and, if needed, the addition of a loop diuretic (e.g., furosemide) to promote renal elimination. Any medication that is capable of impairing K+ homeostasis (e.g., nonselective β-adrenergic antagonists, angiotensin-converting enzyme inhibitors, K+-sparing diuretics, and nonsteroidal anti-inflammatory drugs) should be discontinued, if possible.

Severe hyperkalemia [K+ >6.5 mmol/L]

Temporizing measures produce shifts of potassium from the extracellular to the intracellular space, these are:

NaHCO3 [1 mmol/kg or 1 to 2 ampules (50 mL each) of 8.4% NaHCO3] can be infused intravenously over a 3- to 5-minute period. This dose can be repeated after 10 to 15 minutes if ECG abnormalities persist.

Dextrose (0.5 g/kg body weight) infused with insulin (0.3 unit of regular insulin/g of dextrose) transiently lowers serum K+ (the usual dose is 25 g dextrose, with 6 to 10 units of regular insulin given simultaneously as an intravenous bolus).

Inhaled β-agonists [e.g., albuterol sulfate, 2 to 4 mL of 0.5% solution delivered via nebulizer] have been shown to lower plasma K+, with a duration of action of up to 2 hours.

Calcium gluconate 10% (5 to 10 mL intravenously over 2 minutes) should be administered to patients with profound ECG changes who are not receiving digitalis preparations. Calcium functions to stabilize the myocardium.

Therapeutic measures to definitively decrease total body potassium by increasing potassium excretion:

Sodium polystyrene sulfonate (Kayexalate), a Na+-K+ exchange resin, can be administered orally or rectally (as a retention enema) every 1 to 2 hours initially, followed by administration every 6 hours to promote K+ elimination. A decrease in serum K+ level typically occurs 2 to 4 hours after administration.

Hydration with 0.9% NaCl in combination with a loop diuretic (e.g., furosemide, 20 to 100 mg intravenously) should be administered to patients with adequate renal function to promote renal K+ excretion.

Dialysis is definitive therapy in severe, refractory, or life-threatening hyperkalemia.

C.Calcium

Physiology: Serum calcium (8.9 to 10.3 mg/dL or 2.23 to 2.57 mmol/L) exists in three forms: ionized (45%), protein bound (40%), and in a complex with freely diffusible compounds (15%). Only free ionized Ca^{2+} is physiologically active. Daily calcium intake ranges from 500 to 1,000 mg, with absorption varying considerably. Normal calcium metabolism is under the influence of parathyroid hormone (PTH) and vitamin D. PTH promotes calcium resorption from bone and reclamation of calcium from the glomerular filtrate. Vitamin D increases calcium absorption from the intestinal tract.

1.Hypocalcemia

I.Causes and diagnosis: Hypocalcemia most commonly occurs as a consequence of calcium sequestration or vitamin D deficiency. Calcium sequestration may occur in the setting of acute pancreatitis, rhabdomyolysis, or rapid administration of blood (citrate acting as a calcium chelator). Transient hypocalcemia may occur after total thyroidectomy, secondary to vascular compromise of the parathyroid glands, and after parathyroidectomy. In the latter case, serum Ca^{2+} reaches its lowest level within 48 to 72 hours after operation, returning to normal in 2 to 3 days. Hypocalcemia may occur in conjunction with Mg^{2+} depletion, which simultaneously impairs PTH secretion and function. Acute alkalemia (e.g., from rapid administration of parenteral bicarbonate or hyperventilation) may produce clinical hypocalcemia with a normal serum calcium concentration due to an abrupt decrease in the ionized fraction. Because 40% of serum calcium is bound to albumin, hypoalbuminemia may decrease total serum calcium significantly—a fall in serum albumin of 1 g/dL decreases serum calcium by approximately 0.8 mg/dL (0.2 mmol/L). Ionized Ca^{2+} is unaffected by albumin. As a consequence, the diagnosis of hypocalcemia should be based on ionized, not total serum, calcium.

II.Clinical manifestations. Tetany is the major clinical finding and may be demonstrated by Chvostek's sign (facial muscle spasm elicited by tapping over the branches of the facial nerve) and trousseau’s sign (carpal spasm by inflating blood pressure cuff and maintaining cuff pressure above systolic). The patient may also complain of perioral numbness and tingling. In addition, hypocalcemia can be associated with QT-interval prolongation and ventricular arrhythmias.

III.Treatment

Parenteral therapy. Asymptomatic patients, even those with moderate hypocalcemia (calcium 6 to 7 mg/dL or 1.5 to 1.75 mmol/L), do not require parenteral therapy. Symptoms such as overt tetany, laryngeal spasm, or seizures are indications for parenteral calcium. Approximately 200 mg of elemental calcium is needed to abort an attack of tetany. Initial therapy consists in the administration of a calcium bolus (10 to 20 mL of 10% calcium gluconate over 10 minutes) followed by a maintenance infusion of 1 to 2 mg/kg elemental calcium/hour. mEq). The serum calcium level typically normalizes in 6 to 12 hours with this regimen, at which time the maintenance rate can be decreased to 0.3 to 0.5 mg/kg/hour. Once the serum calcium level is normal, oral therapy can be initiated.

Oral therapy. Calcium salts are available for oral administration (calcium carbonate, calcium gluconate). Each 1,250-mg tablet of calcium carbonate provides 500 mg of elemental calcium (25.4 mEq), and a 1,000-mg tablet of calcium gluconate has 90 mg (4.6 mEq) of elemental calcium and supplemented with a vitamin D preparation such as 50,000 IU of calciferol.

2.Hypercalcemia

I.Causes and diagnosis: Causes of hypercalcemia include malignancy, hyperparathyroidism, hyperthyroidism, vitamin D intoxication, immobilization, long-term total parenteral nutrition, thiazide diuretics, and granulomatous disease. The finding of an elevated PTH level in the face of hypercalcemia supports the diagnosis of hyperparathyroidism. If the PTH level is normal or low, further evaluation is necessary to identify one of the previously cited diagnoses.

II.Clinical manifestations. Mild hypercalcemia (calcium <12 mg/dL or <3 mmol/L) is generally asymptomatic. The hypercalcemia of hyperparathyroidism is associated infrequently with classic parathyroid bone disease and nephrolithiasis. Manifestations of severe hypercalcemia include altered mental status, diffuse weakness, dehydration, adynamic ileus, nausea, vomiting, and severe constipation. The cardiac effects of hypercalcemia include QT-interval shortening and arrhythmias.

III.Treatment: of hypercalcemia depends on the severity of the symptoms. Mild hypercalcemia (calcium <12 mg/dL or <3 mmol/L) can be managed conservatively by restricting calcium intake and treating the underlying disorder. Volume depletion should be corrected if present, and vitamin D, calcium supplements, and thiazide diuretics should be discontinued. The treatment of more severe hypercalcemia may require the following measures:

NaCl 0.9% and loop diuretics may rapidly correct hypercalcemia.

Salmon calcitonin, in conjunction with adequate hydration, is useful for the treatment of hypercalcemia associated with malignancy and with primary hyperparathyroidism. Salmon calcitonin can be administered either subcutaneously or intramuscularly.

Pamidronate disodium, in conjunction with adequate hydration, is useful for the treatment of hypercalcemia associated with malignancy.

Plicamycin (25 μg/kg, diluted in 1 L of 0.9% NaCl or D5 W, infused over 4 to 6 hours each day for 3 to 4 days) is useful for treatment of hypercalcemia associated with malignancy. The onset of action is between 1 and 2 days, with a duration of action of up to 1 week.