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# Fluid management on hospital medical wards

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Daily fluid administration is an essential task on most medical wards. It requires considerable clinical acumen and when done well is the hallmark of an outstanding physician. However, both under- and overhydration are common and associated with significant morbidity and mortality. The extent of the problem is difficult to quantify as it is often multifactorial and under-reported. Nevertheless, postoperatively, overhydration occurs in 17–54% of patients and is reported to prolong hospital stay, increase morbidity (eg pulmonary oedema) and is a potential cause in about 9,000 deaths annually in the USA.[1–3]

Three key issues contribute to poor fluid management:

1 Failure to understand the principles of fluid balance, with several studies demonstrating inappropriate fluid prescription in relation to available fluid balance information (eg serum electrolyte data, input/output charts, daily weights).[2,4] The studies also showed that few junior medical staff received formal education or were given guidelines on fluid and electrolyte prescribing.[4]
2 Poor documentation of fluid balance contributes to morbidity and mortality.[5] Fewer than half of fluid balance sheets are adequately completed and intravenous (iv) fluids are often administered at incorrect rates.[4,6] (Indeed, the accuracy of fluid rates was considered unimportant!)
3 Fluid prescription is often delegated to the least experienced members of the medical team. The National Confidential Enquiry into Peri-Operative Deaths ascribed many of the errors in fluid and electrolyte management to inadequate knowledge and training of junior medical staff.[5] This is reflected in audit evidence suggesting that fewer than 50% of junior doctors know the sodium content of normal saline.[7]

Fluid is best delivered by the oral or nasogastric route but iv fluid may be required in patients unable to drink (or absorb), have excessive fluid losses (eg diarrhoea, burns) or require resuscitation. The aims are to:

- replace normal fluid and electrolyte losses
- provide additional resuscitation fluids to correct for the effects of underlying pathology
- maintain cardiac output, blood pressure (BP), tissue metabolism and waste removal
- ensure a stable intra- and extracellular milieu to preserve membrane potential and transport mechanisms
- avoid oedema and associated complications.

## Factors that influence fluid requirements

### *Fluid and electrolyte compartments*

Water comprises 60% of total body weight, equivalent to about 42 litres in a 70 kg man, of which 25 litres is intracellular fluid (ICF) and 17 litres extracellular fluid (ECF). The ECF is divided into interstitial fluid (ISF) which surrounds the cells (11–14 litres) and intravascular plasma (3–4 litres). They are separated by the capillary endothelium which is permeable to low molecular weight (MW) solutes (eg sodium) but increasingly impermeable to high MW solutes (eg albumin).[1,8]

*Osmotic pressure.* Compartmental distribution of water is primarily dependent on the 'osmotic pressure' exerted by small diffusible ions. Osmotic pressure reflects the ion concentration gradients between compartments created by cellular ion-pumps, such that sodium ($Na^{2+}$) and chloride ($Cl^{-}$) ions are mainly extracellular, and potassium ($K^{+}$) and (phosphate) $PO_4$ ions intracellular. Thus, after a saline infusion the rise in extracellular $Na^{2+}$ and Cl- increases the osmotic pressure, drawing water out of the ICF and into the ECF compartment.

### *Intravascular volume*

The intravascular volume is determined by the 'oncotic pressure' of large MW, non-diffusible vascular plasma proteins, the permeability ('leakiness') of the vessels and circulatory hydrostatic pressure.

*Oncotic (colloid) pressure* describes the ability of 'vascular' plasma proteins to 'bind' and retain water in the circulation. Normal plasma oncotic pressure is about 3.4 kPa (26 mmHg), of which albumin

## Key points

**Fluid administration requires clinical acumen, and is the hallmark of a good clinician**

**Over- and underhydration are associated with significant morbidity and mortality**

**Fluid prescription should replace normal losses and deficits, and ensure adequate resuscitation**

**A normal adult loses 1.5–2.5 litres of water, 70–150 mmol of sodium and 40–70 mmol of potassium daily**

**Crystalloids are as effective as colloids for resuscitation but larger volumes may be required**

**Physiologically balanced crystalloid solutions are preferred to 'normal' saline**

**KEY WORDS: colloids, crystalloids, fluid management, overhydration, underhydration**

accounts for over 70%, haemoglobin (Hb) about 20% and globulins less than 5%. Total body albumin is about 275 g (125 g intravascular, 150 g ISF). One gram of albumin binds 18 ml of water; thus, intravascular albumin holds 2.25 litre (18 ml × 125 g) of water in the vascular compartment. Normal albumin leakage from blood to the ISF and subsequent return to the vascular compartment via lymphatic drainage is around 125 g/day.

*Endothelial permeability* is increased in pathological conditions, for example, surgery and sepsis, and albumin leakage may rise by 100% and 300%, respectively. The resulting fall in plasma albumin reduces intravascular volume, while raised ISF albumin increases tissue oedema.

### *Normal losses and renal function*

A normal adult loses 1.5–2.5 litres of water, 70–150 mmol of $Na^{2+}$ and 40–70 mmol of $K^+$ daily.[9] A further 0.7 litres of 'insensible fluid loss' occurs as lung water vapour and sweat; this can exceed 3 litres/day during febrile illnesses or with high ambient temperatures. Loss of skin or mucous membrane barriers (eg burns, ulcerative colitis), fluid-losing enteropathies (eg diarrhoea) and salt-losing renal failure is also associated with further large fluid and electrolyte losses.

The daily urine output contains a total solute load of about 620–840 mosmol (ie ca 500 mosmol/litre: $Na^{2+}$ (ca 70–150 mmol) x 2) + ($K^+$ (ca 40–70 mmol) × 2) + urea (ca 400 mmol). In healthy kidneys, maximum urine solute concentration is 1,000 mosmol/litre, but during acute illness this may fall to 500 mosmol/litre or less. Thus, to excrete the normal daily solute load, normal kidneys require a minimum urine output of 35 ml/hr (ca 0.5 ml/kg/hr), but during acute illness the urine volume must be doubled (ie 70 ml/hr). Unfortunately, catabolic metabolism also increases during serious illness or injury, increasing daily urea waste production to over 1,000 mosmol/day. This increases the solute load further, diminishing the capacity to excrete the total solute load.

Despite wide variations in salt and water intake the kidneys maintain extracellular $Na^{2+}$ concentration and osmolality within a narrow range. During salt and/or water depletion antidiuretic hormone (ADH) secretion increases urine concentration and reduces water clearance, while the renin-angiotensin-aldosterone system (RAAS) reduces urinary sodium to below 5 mmol/litre. The response to sodium excess is less efficient and depends on passive suppression of the RAAS rather than on active elimination by natriuretic hormones. Even normal subjects are slow to excrete a sodium load.[10] Table 1 summarises the causes of sodium and water imbalance in acute illness.

During a difficult resuscitation it is not unusual for patients to be given 5 litres of normal saline (a total of 1,540 mosmol of solute) (Table 2). Normal excretion of waste solutes (eg urea) and electrolytes is about 700 mosmol/day; consequently, assuming a degree of renal impairment with loss of concentrating ability (see above), it may only be possible to excrete a small function of the 'additional' normal saline solute load each day (ie it will take several days to remove the solute load in 5 litres of normal saline). The retained Cl- may subsequently cause hyperchloraemic acidosis (HCA), renal vasoconstriction and reduced glomerular filtration.[7,10–12]

### *Response to stress*

After acute illness or injury the body retains salt and water. Total fluid retention may be more than 10 litres in some cases. It usually accumulates in the ISF, causing tissue or pulmonary oedema. Causative mechanisms include:

- ADH, RAAS and catecholamine mediated antidiuresis and liguria
- impaired renal function with reduced free water excretion and urine concentrating ability
- catabolic urea production which competes with sodium for excretion
- potassium depletion due to RAAS activity, reducing sodium excretion
- hyperchloraemia which reduces glomerular filtration and causes sodium retention[11,13]
- Na/K ATPase pump failure due to reduced cellular energy causing intracellular $Na^{2+}$ and water sequestration ('sick cell syndrome')
- reduced intravascular volume due to increased albumin leakage which activates RAAS with further salt and water retention and worsening interstitial oedema.

## Assessing fluid balance

Assessment of fluid balance and subsequent fluid prescription is a complex and difficult task and should not be delegated, as so often the case, to the most junior member of the medical team. The key to improvement is in better training of medical students and junior doctors to ensure that they have an understanding of the issues and the correct clinical assessment skills. In addition, easily accessible guidelines must be available.

**Table 1. Causes of sodium and water loss and retention in acute illness.**

| Sodium and water retention | Sodium and water loss |
|---|---|
| • Stress response (ADH release) (eg burns, surgery, trauma)<br>• Hyperosmolar states (ADH release) (eg uraemia, hyperglycaemia)<br>• Reduced ANP with aldosterone release (eg dehydration, low $Na^+$ input, hypovolaemia, excess $K^+$)<br>• Increased capillary permeability with oedema and hypovolaemia (eg trauma, burns, sepsis, pancreatitis) | • Acute blood loss (eg haemorrhage)<br>• Kidney (eg diuretics, renal disease, diabetes)<br>• Skin (eg sweating, burns, skin disease)<br>• GI tract (eg vomiting, fistula, diarrhoea)<br>• Fluid sequestration (eg bowel obstruction, ascites, pleural effusions) |

ADH = antidiuretic hormone; ANP = atrial natriuretic peptide; GI = gastrointestinal.

**Table 2. Composition of crystalloids and colloids.**

| Fluid | Na (mmol/l) | K (mmol/l) | Cl (mmol/l) | Osmolality (mosmol/l) | Additions (mmol/l) | |
|---|---|---|---|---|---|---|
| Plasma | 136–144 | 3.5–4 | 98–105 | 280–300 | | |
| Crystalloids: | | | | | | |
| • Dextrose 5% | 0 | 0 | 0 | 278 | Glucose: 280 | |
| • Saline 0.45% | 77 | 0 | 77 | 154 | Nil | |
| • Saline 0.9% | 154 | 3 | 154 | 308 | Nil | |
| • Hartmann's | 131 | 5 | 111 | 275 | $HCO_3$ 29, Ca 1.8 | |
| • Ringer's | 130 | 4 | 109 | 273 | Ca 22 | |
| | | | | | T½ (hrs) | PVE (%) |
| Colloids: | | | | | | |
| • Gelofusine | 154 | 0 | 125 | 279 | ~2 | 80 |
| • Haemaccel | 145 | 0 | ? | 300 | ~1 | 70 |
| • Albumin 5% | 130–160 | 0 | 150 | 300 | days | 100 |
| • HES 10% | 154 | 0 | 154 | 308 | 12–14? | 145 |

HES = hydroxyethyl starch; PVE = plasma volume expansion (% of volume given); T½ = approximate effective plasma half-life.

In most patients, clinical assessment is adequate, with review of fluid input (eg resuscitation fluids, feed) and output (eg urine output, gastrointestinal (GI) loss, drainage), non-invasive monitoring of vital signs, and measurements of serum (and occasionally urinary) electrolytes and urea. The need for accurate fluid balance charts must be emphasised to the nursing staff. This is frequently neglected on both medical and surgical wards and can result in carers missing gradually progressive over- or underhydration. Day-to-day trends in these parameters and daily weights usually guide fluid prescription.

In some unstable patients the cardiovascular (eg pulse, BP, central venous pressure) response to serial 'fluid challenges' (ca 250 ml over <20 min) is used to determine the need for volume replacement (Fig 1). This response is not always reliable as it is influenced by venous tone, does not have a linear relationship with cardiac output and lacks sensitivity and specificity. Other measures of inadequate tissue perfusion include an increase in serum lactate and mixed venous saturation ($SvO_2$) below 70%. To determine fluid needs in more complex cases may require non-invasive transoesophageal Doppler or invasive cardiac monitoring to assess cardiac output and filling pressures.

## Fluid choice

With the exception of 5% dextrose, almost all iv solutions contain $Na^{2+}$ and Cl- (Table 2), some in near physiological concentrations (eg Hartmann's) and others in supranormal amounts (eg 'normal' (0.9%) saline). The excess Cl- increases the risk of HCA.[7,10,11,13]

*Crystalloid solutions* contain low MW salts (eg NaCl) and sugars (eg glucose) which dissolve completely in water and pass freely between intravascular and interstitial compartments. Although inexpensive and usually isotonic, they redistribute rapidly (1–4 hr) following iv infusion from the intravascular to other fluid compartments. Consequently, large volumes are required to maintain intravascular volume, which may cause interstitial oedema. Low sodium fluids (eg 5% dextrose) disperse throughout all fluid compartments, whereas sodium containing fluids (eg 0.9% saline) have a smaller volume of distribution because cellular pumps remove sodium from ICF, limiting distribution to the ECF.

*Colloid solutions.* These contain complex, high MW molecules and are retained in the vascular compartment for longer periods than crystalloid solutions, exerting an oncotic pressure which expands the intravascular compartment. It is often quoted that about three to four times as much crystalloid is required for the same intravascular volume expansion as a colloid, but meta-analysis shows no advantage for colloids over crystalloids for initial resuscitation.[14] Both natural (eg albumin) and synthetic colloids (eg gelatin, hydroxyethyl starch (HES), dextran) are expensive and have specific properties and side effects. For example, albumin may aid lung water clearance in acute lung injury and is beneficial in hypoalbuminaemic (<15 g/dl) patients with severe sepsis. Low MW gelatins are

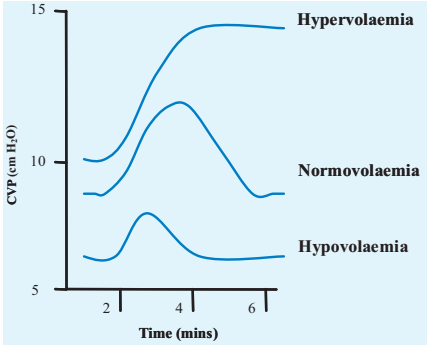


Fig 1. Central venous pressure (CVP) response to 500 ml fluid challenge.

**Table 3. Typical clinical scenarios requiring intravenous fluid replacement.**

| Complications | Explanation |
|---|---|
| Maintenance fluid | • Stable euvolaemic patients with normal renal function require 1–1.5 ml/kg/hr water, 70–150 mmol $Na^{2+}$ and 40–70 mmol $K^+$ daily. In an 80 kg man, this equates to 1 litre of normal saline and 2 litres of 5% dextrose with 20–40 mmol $K^+$ added to each litre of 5% dextrose |
| | • Some authorities recommend PBS (eg Hartmann's) as maintenance fluid as this reduces $Cl^-$ load when larger volumes are required |
| | • If there is significant hypernatraemia ( $\uparrow Na^+$, $\uparrow Cl^-$) or hyponatraemia ($\downarrow Na^+$, $\downarrow Cl^-$), 5% dextrose or saline 0.9%, respectively, should be used |
| Post-major surgery | • If hypovolaemia occurs despite normal maintenance fluid (1–1.5 ml/kg/hr), increase the infusion rate to 2–3 ml/kg/hr using PBS to reduce the $Cl^-$ load and give 0.5 litres gelofusine 'fluid challenges' to support the circulation |
| | • Maintain Hb >8 g/dl in stable patients (>10 g/dl in unstable cardiac patients) |
| | • Remember that large fluid requirements may indicate internal bleeding |
| Major haemorrhage | • Requires control of bleeding and restoration of circulating volume |
| | • Aggressive fluid resuscitation before surgery can increase blood loss due to dilutional coagulopathy, acidosis and hypothermia |
| | • Blood is the ideal resuscitation fluid, but requires time to prepare. Therefore, fluid replacement starts with 20–30 ml/kg PBS, followed by 1–1.5 litres gelofusine (±1–1.5 litres HES solution) |
| | • Ongoing resuscitation may require plasma and platelets |
| Sepsis and septic shock | • Intravascular hypovolaemia resulting from increased vessel permeability may be difficult to assess, requiring repeated 'fluid challenges' and close monitoring (eg urine output, lactate, CVP, $SvO_2$) |
| | • Fluid replacement follows the 'Surviving Sepsis Guidelines' and starts immediately in hypotensive patients or if lactate is raised.[18] Initially give 20–40 ml/kg PBS (eg Hartmann's) in aliquots of 0.5–1 litres based on clinical response |
| | • If hypotension or lactate elevation persist, insert a CVP line and continue resuscitation with gelofusine 1–1.5 litres and then HES solution 1–1.5 litres, aiming for a CVP of 8–12 mmHg, MAP >65 mmHg, urine output >0.5 ml/kg/hr, $SvO_2$ >70% and Hb 8–10 g/dl (but transfuse to only 10 g/dl if dysoxic) |
| Head injury | • Hyponatraemia, hypoxia, hypotension and hyperthermia worsen brain oedema |
| | • Normal saline is recommended for fluid resuscitation as a degree of hypernatraemia may be beneficial |
| | • Dextrose 5% should be avoided except with associated diabetes insipidus |
| Acute kidney injury | • Reduces the ability to excrete fluid and electrolytes; these patients are at significant risk of fluid and sodium overload and associated hyperkalaemia[4,12] |
| | • PBS containing potassium can be used cautiously in closely monitored patients, but if hyperkalaemia develops non-potassium containing crystalloid solutions should be used (eg 4%/0.18% dextrose/saline or 0.45% saline) |
| | • In established oliguric or anuric renal failure, fluid replacement is restricted to insensible loss (0.5–1 litre daily) to avoid fluid overload; electrolytes are monitored daily and sodium and potassium intake restricted |

CVP = central venous pressure; Hb = haemoglobin; HES = hydroxyethyl starch; MAP = mean arterial pressure; PBS = physiologically balanced solution.

rapidly excreted through the kidneys producing short-term volume expansion. Other disadvantages of colloids include allergic reactions and clotting abnormalities.

*Blood* is given to maintain Hb concentration above 8 g/dl, but young patients and those with renal disease, haemoglobinopathies or chronic anaemia may tolerate lower levels. A Hb of about 10 g/dl improves outcome in cardiac patients.

*Sodium bicarbonate* (1.26%) is used in patients with metabolic acidosis (pH <7.2) due to renal failure or GI losses, and has been advocated in recent acute kidney injury (AKI) guidelines.[15]

## Fluid administration

Fluid selection is guided by the underlying condition, extracellular fluid status (eg oedema), fluid losses (eg diarrhoea), renal function and electrolyte concentrations. In the absence of normal homeostatic mechanisms, patients need:

- basic maintenance fluids to replace normal daily water and electrolyte losses, and
- additional resuscitation fluids to correct losses due to underlying pathology and to maintain an adequate circulation.[7,16,17]

In general, the fluid that is lost is replaced. Thus, blood is most appropriate for haemorrhagic loss. Replacement fluids should match normal daily losses in the euvolaemic, stable patient unable to take oral or enteral fluids. In acutely unwell patients (eg sepsis) and those with renal impairment or complex fluid losses (eg burns, fistulae) selection of replacement fluid (eg crystalloid, colloid) is more complex and specialty guidelines (eg AKI, sepsis, diabetic ketoacidosis) should be followed.

In many patients, particularly those with renal impairment, large volumes of

crystalloid (or colloid) engender large solute loads (eg $Na^{2+}/Cl^{-}$) which are difficult to excrete and precipitate HCA. In these circumstances, physiologically balanced solutions (PBS) with lower $Cl^{-}$ contents (eg Hartmann's, Ringer's lactate) are preferred and should replace 0.9% saline except in cases of hypochloraemia (eg vomiting). These PBS cause less HCA and reduce associated nausea, confusion and oliguria.

Solutions such as 5% dextrose and 4%/0.18% dextrose/saline are important sources of free water for maintenance, but should be used with caution if fluid requirements beyond maintenance are needed since excessive amounts may cause dangerous hyponatraemia, especially in the elderly. These solutions are not therefore appropriate for resuscitation or replacement therapy except in conditions of significant free water deficit (eg diabetes insipidus). Table 3 includes examples of some common clinical scenarios requiring different iv fluids.

## Conclusions

Optimum iv fluid prescription depends on a sound understanding of the basic principles of physiology as well as the pathophysiology of disease processes. When undertaken thoughtfully this – often neglected – clinical skill is likely to significantly enhance a patient's chances of making a good recovery and avoiding iatrogenic complications.

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