

Perioperative fluid therapy

Sahir S Rassam MB ChB MSc FFARCSI

David J Counsell MB ChB FRCA

Basic fluid and electrolyte physiology and the effect of their disturbances have been discussed in a previous article.¹ In this article, an outline of practical perioperative fluid management, fluid solutions and the use of fluids in special clinical circumstances are discussed.

Assessment of hydration

Clinical assessment of intravascular volume and laboratory investigations are essential to guide perioperative fluid therapy. Thirst, skin turgor, hydration of mucous membranes, core-peripheral temperature gradient, pulse rate and volume, changes in blood pressure in the upright position and urine output are invaluable clues to the state of hydration. However, haemodynamic variables are affected by factors other than volume status such as drugs and the physiological effects of surgical stress. Therefore, it is useful to look at trends rather than a single reading.²

Thirst occurs in response to hypovolaemia (via baroreceptors) and to changes of as little as 1% in osmolarity. In normal life, thirst plays a crucial role as a sensitive indicator of fluid deficit; it must be distinguished from dry mouth, which can result from oxygen therapy or drugs. Fluid balance is rarely a major problem in those patients who are able to drink.

Loss of skin turgor indicates an intravascular deficit of about 10%. Orthostatic and supine hypotension implies deficits of 20% and 30%, respectively. Healthy individuals may sustain 20% loss of circulating volume and only exhibit postural tachycardia. With autonomic dysfunction, postural hypotension may occur in normovolaemia. Signs of hypervolaemia are suspected in the presence of pitting oedema and increased urinary flow in patients with normal cardiac, hepatic and renal function. Pulmonary oedema is a late sign of hypervolaemia.

Laboratory signs of dehydration include a rising haematocrit, progressive metabolic acidosis, hypernatraemia and urinary sodium >20 mmol litre⁻¹. Changes in plasma urea and creatinine should be interpreted in the

light of other factors such as age and lean body mass.² Central venous pressure readings must be interpreted in light of the clinical setting. Low values (<5 mm Hg) may be normal unless associated with other signs of hypovolaemia. The principle behind a fluid challenge is to assess the compliance of the circulation and to re-evaluate intravascular volume status by observing the haemodynamic and clinical response.

Rational approach to clinical fluid balance

In practice, previously healthy patients with an uncomplicated perioperative course tolerate even the most bizarre fluid regimens. Problems are much more common in those with pre-existing renal or cardiopulmonary disease and those who develop complications.

(i) First consider normality: basic adult water requirements are 35 ml kg⁻¹ day⁻¹ (2.5–3 litres) with sodium 2 mmol kg⁻¹ and potassium 1 mmol kg⁻¹.³ In practice, this amounts to 1 litre of 0.9% saline the rest of the volume made up with 5% dextrose. Addition of potassium to each bag (20–27 mmol litre⁻¹ as available) covers the potassium requirement.

Next consider circumstances requiring alterations from this basic regimen.

(ii) Consider previous fluid status: the need to 'catch up' with fluids in the perioperative period is often underestimated. Contributing factors include vomiting, diarrhoea, ileus, pyrexia, burns, haemorrhage, ascites, effusions, bowel preparation, confusion, prolonged 'nil-by-mouth' regimens and diuretics. Is the patient thirsty?

(iii) Consider previous electrolyte status: electrolyte imbalance is commonly iatrogenic. Give the daily potassium requirement even if serum potassium is within normal range. Excess loss of body fluids leads to excess losses of electrolytes. Losses caused by problems such as vomiting and diarrhoea will have a similar, though not identical, electrolyte profile to

Key points

Thirst plays a crucial role as a sensitive indicator of fluid deficit and fluid balance is rarely a major problem in those patients who are able to drink.

Iatrogenic problems are common; a structured approach to fluid management is required.

'Third-space' fluid losses must be taken into account in the perioperative period.

Alteration of fluid prescribing is required according to the clinical circumstances.

Sahir S Rassam MB ChB MSc FFARCSI

Consultant Anaesthetist
Department of Anaesthetics
University Hospital of Wales
Heath Park
Cardiff
CF14 4xW

David J Counsell MB ChB FRCA

Consultant Anaesthetist
Department of Anaesthetics
Wrexham Maelor Hospital
Croesnewydd Road
Wrexham
LL13 7TD
Tel: 01978 725955
Fax: 01978 725932
E-mail: dave.counsell@btinternet.com
(for correspondence)

plasma. Therefore, applying the principle of replacing like with like, with Hartmann's solution is sensible.

(iv) Anticipate ongoing excess losses: protracted losses (e.g. fistula, nasogastric tube) will have fluid and electrolyte implications and should be replaced with Hartmann's. Insensible losses, 'third-space' losses and the need to volume expand in anticipation of anaesthetic affects increase perioperative fluid requirements considerably.

(v) Anticipate excess fluid intake: this will need to be subtracted from the maintenance fluid regimen, for example multiple drug infusions, parenteral or enteric nutrition or antibiotics requiring large volume infusions.

The choice of fluids

Debate exists as to the most appropriate fluid type to use in the perioperative period. Systematic reviews show no difference in pulmonary oedema, mortality or length of stay between crystalloid and colloid resuscitation.^{4,5} The composition and uses of commonly used i.v. fluids are summarized in Table 1.

Distribution of fluid throughout the body is influenced by Starling forces, i.e. the hydrostatic and colloid pressure differences across capillary walls that cause movement of water and dissolves solutes into the interstitial spaces. The lungs are moderately permeable relative to other organs and, during pathophysiological processes such as surgical trauma, an increase in capillary permeability and leak may result. In these situations, colloid (and crystalloid) molecule leakage into the interstitial space causes further swelling of tissues.

Crystalloids provide water and electrolytes and expand intravascular fluid. Extracellular fluid (ECF) depletion is better replenished by crystalloids. The replacement requirement is 3- or 4-fold the volume of blood lost because it is distributed in a ratio 1:4 intravascular:extravascular.⁶ Allergic reactions are avoided and solutions are much cheaper than colloids. Excessive resuscitation with crystalloids dilutes the plasma proteins, reducing plasma oncotic pressure and resulting in fluid filtration from the intravascular to the interstitial compartment and the development of interstitial pulmonary oedema.

Colloids are larger molecules that cross the capillary membrane less easily returning quickly to the circulation via lymphatic drainage and exerting colloid oncotic pressure (COP). Therefore, less volume is required and there is less risk of peripheral or pulmonary oedema. Colloids have little effect on osmotic pressure and electrolytes are added to achieve iso-osmolality with blood. Increase amounts of colloid may increase the interstitial oncotic pressure, thus exacerbating oedema if vascular permeability is increased.

Crystalloids

Normal saline 0.9% is isotonic and iso-osmotic but contains more chloride than ECF. Administration of large volumes results in mild hyperchloraemia.¹ Dextrose 5% is iso-osmotic and functions as free water because the dextrose is metabolized. When a crystalloid is transfused, all Na⁺ remains extracellular because of the sodium pump. If 0.9% saline (isotonic solution) is infused, the osmolality of ECF remains the same (no water exchange

Table 1 Composition of commonly used i.v. fluids. Units are in mmol l⁻¹ unless otherwise stated. Osmolality in mosmol kg⁻¹

Fluid	Na ⁺	K ⁺	Ca ²⁺	Cl ⁻	Others	Osmolality	pH	Uses
Saline 0.9%	154			154		308	5	Brain injury, hypochloraemic metabolic alkalosis or hyponatraemia
Hartmann's	131	5	2	111	Lactate 29	281	6.5	Extra cellular fluid replacement
Glucose 5%					Glucose 50 g	278	4	Hypernatraemia, preventing hypoglycaemia in diabetics
Saline 0.45%	77			77		154	4-5	Water and Na replacement, hypernatraemia
Glucose 4%-saline 0.18%	31			31	Glucose 40 g	284	4-5	Maintenance of normal fluid volume
Bicarbonate								Severe metabolic acidosis
8.4%	1000				HCO ₃ ⁻ 1000		8	
1.26%	150				HCO ₃ ⁻ 150		7	
Gelofusine MW 30 000	154	0.4	0.4	125	Gelatin 40 g, Mg 0.4	274	7.4	Plasma volume expander
Haemaccel MW 30 000	145	5.1	6.25	145	Gelatin 35 g	301	7.3	Plasma volume expander
Dextran 70					Dextran 60 g, Glucose 50 g	287		Reduction of plasma viscosity, plasma volume expander
In 5% glucose					or as saline		5-6	
In saline 0.9%					0.9%		4-5	
MW 70 000								
HAS 4.5%	100-160	<2		100-160	Albumin 45 g, citrate <15	270-300	6.4-7.4	Abnormal loss of protein from the vascular spaces in peritonitis and burns
HAS 20%	50-120	<10		<40	Albumin 200 g	135-138	6.4-7.4	Abnormal loss of protein from the vascular space as in peritonitis and burns. Nephrotic syndrome
Hespan 6%	154			154	Starch 60 g	310	5.5	Plasma volume expander
MW 200 000								
Voluven	154			154	Starch 60 g	307	4-5.5	Plasma volume expander
MW 130 000								

MW = molecular weight.

occurs across cell membrane). If glucose 5% (hypotonic solution) is infused, ECF osmolality decreases and a shift of water occur from ECF to ICF in a ratio of 1:2 (66% of total body water is ICF).⁶

Sydney Ringer developed a solution with electrolyte constituents that have been shown to be essential for tissue function. Alexis Hartmann modified this solution for clinical use. The aim was to find an isotonic solution that produces an alkalizing effect to treat acidosis in sick children. With the addition of lactate to Ringer's solution, Hartmann's solution consists of: Na⁺ 131 mmol litre⁻¹, K⁺ 5 mmol litre⁻¹, Ca²⁺ 2 mmol litre⁻¹, Cl⁻ 111 mmol litre⁻¹ and lactate 29 mmol litre⁻¹. The lactate is metabolized mainly by gluconeogenesis (70%) in the liver. Hydrogen ions are consumed and a relative excess of bicarbonate ions is produced reducing acidosis in a controlled fashion (2CH₃CHOHCOO⁻ + 2H⁺ → C₆H₁₂O₆). If a pathological condition affecting lactate metabolism or the renal excretion of bicarbonate is present, as in some critically ill patients who are obligate excretors of acidic urine, a state of alkalosis and hypokalaemia may develop.⁷

Colloids

Gelatines

Gelofusine is prepared as a 4% solution of succinylated gelatine in saline. COP is 35 mm Hg and $t_{1/2}$ is 2–4 h. *Haemaccel* is a 3.5% solution of polygeline in a mixed salt solution. It is cross linked with urea, which may be released after hydrolysis, a potential problem in patients with renal failure. COP is 28 mm Hg and $t_{1/2}$ is 6 h. *Dextrans* are glucose polymers and available as solutions with either sodium chloride 0.9% or dextrose 5% in different molecular weight preparations.

Starches

Starches are composed of amylopectin that is linked with hydroxyethyl groups in a glucose moiety making the resultant polymer similar to glycogen. Solutions have a large range of molecular weights. *Hespan* is 6% hetastarch in sodium chloride with a COP of 20 mm Hg; 60% of the solution remains in the body for 24 h because of the hydroxyethyl–glucose bond. *Voluven* is similar to Hespan but with a lower molecular weight. Tissue storage is 75% less than Hespan and allergic reactions are less likely than with Voluven compared with other colloids. *Human albumin solutions* are derived from human plasma by fractionation. They are heat sterilized rendering the risk of infective transmission very low.

RescueFlow is a *hypertonic salt solution* and is available as 6% dextran 70/7.5% sodium chloride. The hypertonic saline induces a rapid fluid shift from the intracellular to intravascular space; dextran then sustains volume expansion. This may be useful in patients predisposed to tissue oedema e.g. severe burns, traumatic brain injuries. Hypernatraemia can occur easily if used in large amounts (>500 ml); plasma electrolytes should be monitored closely.

Clinical challenges in perioperative fluid therapy

Preoperative

Starvation

Free administration of clear fluids up to 2 h before an anaesthetic leads to lower gastric content volumes of higher pH value.⁸ Starvation and fluid restriction should be considered as separate issues and policies need to be developed to implement this change of practice. The fluid 'catch up' required as a result of outdated regimes can be considerable.

Bowel preparation

Bowel preparation is commonly achieved using osmotic purgatives that result in fluid losses in excess of the fluid administered. Weight loss, largely caused by fluid depletion, of up to 2 kg is not uncommon. Consider i.v. fluid administration particularly when late admission necessitates bowel preparation on the evening before surgery followed by fluid restriction.

Emergency patients

Audit data suggest that diarrhoea and/or vomiting occurs in ~60% of emergency general surgical admissions, often leading to significant dehydration. Rehydration could easily be achieved in most patients during the hours between admission and surgery but this preoperative 'window of opportunity' is rarely utilized effectively. The 'like with like' principle suggests the use of Hartmann's in this situation.

Intraoperative

Unmeasured losses amount to 10 ml kg⁻¹ h⁻¹ when the body cavity is open. Fluid responsible for external unmeasured losses is the sero-sanguinous fluid present in wounds and in the pleural and peritoneal cavities. This fluid has an electrolyte and colloid content similar to plasma and is lost along with the fluid on swabs, surgical gowns and drapes. 'Third-space' losses beginning in theatre are also responsible for further unobserved losses. Replacement of these fluids with a mixture of Hartmann's solution and a colloid is logical.

Postoperative

The cardiovascular effects of poor analgesia hide the cardiovascular manifestations of fluid deficit. The increased use of epidural analgesia has unmasked this deficit with significant hypotension occurring in ~25% of patients. Poor fluid management in the pre- and intraoperative periods contribute to this problem but other postoperative factors compound it.

Management of postoperative fluid replacement

Traditional teaching and prescribing

The need to restrict fluid and potassium in the first postoperative day because of SIADH is commonly taught. Considering the

perioperative loss and shifts of both fluid and electrolytes, a deficit of both is likely to be present early in the postoperative period. Administration of the daily potassium requirement, commencing straight from theatre is safe and may have benefits in terms of avoiding postoperative ileus. Dextrose/saline is often prescribed day after day with no potassium supplementation; this leads to hyponatraemia and hypokalaemia. The fear of fluid/ Na^+ overload in elderly patients with co morbidities can also lead to hyponatraemia and hypovolaemia.

Structured approach to fluid management

A more structured approach to fluid management is required to overcome these problems. Third-space losses are usually underestimated and postoperative fluid prescribing rarely takes them into account. It is common practice to include intraoperative fluids in the fluid balance on the day of surgery. Unfortunately, the operative losses and unmeasured losses are not included leading to confusion and the inappropriate use of diuretics instead of fluid challenges. The physiological figure quoted for insensible losses (850 ml day^{-1}) does not include faeces and unmeasured metabolic production. Sweat losses in warm hospitals has been suggested to be up to 1 litre, or more if pyrexial. A value of 1–1.5 litre is more appropriate in hospitalized post-operative patient.

Bowel obstruction

Patients with bowel obstruction are often elderly with limited physiological reserves and are dehydrated secondary to gastrointestinal fluid losses, decrease fluid intake for many hours and third-space losses. The degree of deficit can be masked by compensatory mechanisms as the volume depletion develops slowly. In general, perioperative fluid intake will be much greater than the measured output, which can sometimes be misinterpreted as fluid overload. In addition, nutrition is usually impaired preoperatively and protein losses into oedematous bowel and via damaged capillary membranes are increased leading to hypoalbuminaemia which further exacerbates fluid losses from the vascular space.

The goals of fluid management include restoration of the vascular and interstitial volumes, correction of electrolyte depletion and metabolic acidosis and optimisation of oxygen delivery. The rate at which the ECF volume can be replenished is limited in vasoconstricted patients. If fluids are infused more rapidly than the rate at which they can enter the ECF, increased filling pressures may result in pulmonary oedema. Frequent monitoring of arterial blood pressure, heart rate, urine output, central venous pressure, electrolytes, Hb and COP is essential. Fluid lost to the bowel and ECF is similar to plasma water in electrolyte composition and Hartmann's solution is a reasonable first choice. Albumin or colloids should be considered if COP is $<15 \text{ mm Hg}$. Maintenance fluid should be continued with saline, dextrose and potassium. Boluses of Hartmann's or colloids should be given when urine

output is low and when central venous pressure declines below baseline.⁶

Sustained upper gastrointestinal losses

This occurs because of prolonged vomiting or nasogastric losses and leads to depletion of sodium, potassium, chloride and hydrogen ions and hypovolaemia with metabolic alkalosis. The physiological priority is to restore volume by retaining sodium and water in the kidney at the expense of potassium and hydrogen ions which continue to be excreted thus worsening the alkalosis. Restoring volume with saline or Hartmann's is the key to successful management.⁶ Acidifying solutions ($\text{HCL}/\text{NH}_4\text{CL}$) are not usually required.

Liver failure

Patients with liver failure appear fluid overloaded and hypovolaemic at the same time. Fluid retention with renal impairment is usually present. Ascites and tissue oedema secondary to sodium and water retention occur mainly because of failure of the liver to catabolize aldosterone. This effect is potentiated by the abnormally high portal venous pressure, hypoalbuminaemia and increased intra-abdominal pressure leading to an increase in caval pressure. The latter decreases renal blood flow and glomerular filtration rate because the renal perfusion pressure gradient is decreased both by systemic hypotension and increased caval pressure. Therefore, renin is activated and aldosterone production is increased. These patients are functionally hypovolaemic despite normal or elevated total blood volume.

The goals of therapy are to avoid increasing interstitial fluid overload, and maintaining normal potassium concentration and intravascular volume. In the presence of heart failure, inotropes are needed with diuretics when filling pressures are increased. In acute hypovolaemia and in cases where the COP is low, albumin is a good choice and preferred to crystalloids, which tend to expand the already large ECF volume.⁶

Diabetes mellitus

A disturbance in fluid regulation is a serious and common problem in poorly controlled diabetics. Insulin deficiency causes: (i) decreased glucose uptake resulting in hyperglycaemia, glucosuria, osmotic diuresis and electrolyte disturbances; (ii) increased lipolysis resulting in ketogenesis and ketonuria; and (iii) increased protein catabolism resulting in an increase in plasma amino acids and nitrogen loss in urine. These three effects cause dehydration and acidosis.

The goals of therapy are hydration, correction of electrolytes disturbances and acid-base status and appropriate blood sugar control.³ Blood glucose control and management regimens are related to the severity of the condition and the type of surgery. In minor surgery and well controlled cases, there is no need to adjust therapy but early morning doses of oral hypoglycaemic

drugs and insulin should be avoided if blood glucose is low. In major surgery, good control of blood sugar is essential and can be achieved by glucose and insulin sliding scale regimens.

Normal saline is typically required to correct dehydration and sodium deficit that is usually present in moderate to severe conditions. When plasma glucose reaches $10 \text{ mmol litre}^{-1}$, an infusion of glucose–saline must be given to lessen the possibility of hypoglycaemia and to provide a continuous source of glucose for eventual normalization of intracellular metabolism. Hartmann's solution should be avoided in poorly controlled cases since the liver converts lactate to glucose. Potassium supplementation is essential; a state of intracellular hypokalaemia is usually present.³

Renal failure

The goals of therapy are to avoid excessive intravascular fluid administration and ECF volume expansion and to correct electrolytes and acid–base status. It is also important to avoid precipitating conditions that would require dialysis during the immediate postoperative period (e.g. hyperkalaemia, pulmonary oedema, and metabolic acidosis). Hypertension, diabetes and vascular disease are common in chronic renal failure and avoidance of hypotension is important to maintain coronary or cerebral perfusion. Hypotension also worsens ischaemic renal injury in acute renal failure. If dialysis is required it should be performed 12–24 h preoperatively as recent dialysis often induces acute electrolyte shifts and hypovolaemia. The use of colloids to treat

hypovolaemia is preferable. Third-space losses can be replaced with crystalloids but should be limited to $1\text{--}2 \text{ ml kg}^{-1} \text{ h}^{-1}$. An isotonic fluid without potassium with reduced amounts of chloride and increased amount of buffer is useful to correct sodium, potassium and acidosis. It is important to remember that 70% of normal fluid intake is used in excreting solute via the kidney, a route no longer available in these patients.

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See multiple choice questions 123–126