PERIOPERATIVE FLUID AND ELECTROLYTE MANAGEMENT IN PAEDIATRIC PATIENTS
Dr. Suresh G. Nair¹ Dr. Rakhi Balachandran²

Introduction

Fluid management of the paediatric surgical patient is a critical element in the care of infants and children who are sensitive to small degrees of dehydration. Complex surgical procedures are often associated with rapid changes in fluid requirements necessitating frequent assessment and modifications of fluid therapy. In the operating room, the fluid requirements may rapidly change during the conduct of anaesthesia and surgery, coincident with changes in temperature, metabolism and fluid volume shifts. The trauma, haemorrhage and tissue exposure associated with surgery shifts body fluids between compartments, necessitating fluid replacement with solutions that compensate for energy, water, protein and electrolyte losses. The anaesthesiologist must determine the nature and magnitude of these losses and be alert both to the obvious fluid losses of serum and urine and to hidden fluid losses, which can occur, with insensible loss and third space loss of fluid.

This short review will deal with the fluid and electrolyte management in the perioperative period of infants and children without going into any specific situations.

Physiological considerations in infants and children

Before one can scientifically approach the subject of fluid management in infancy and childhood, one must understand neonatal physiology and the changes that take place with time.

Total body fluid

The total body fluid (TBF) is divided into extra cellular fluid (ECF) and intra cellular fluid (ICF). Although body cells and the surrounding fluid remain in electrical equilibrium, the proportion of ECF and ICF changes with age. A 28 week foetus weighing 1 kg will be 80% water and only 1% total body fat. At term, the total body fluid (TBF) decreases to 70-75% and a gradual shift of the extra cellular fluid into the intra cellular compartment has occurred. The fat component has now increased to 17%. At 3 months, when most infants have doubled their weight to 6 kg the fat component is 30% of their weight. In addition, the TBF has decreased to 65%.¹ This is associated with a further increase in their intracellular fluid.

Extra cellular fluid

This fluid includes the intravascular plasma volume and the interstitial fluid volume. The plasma volume and the interstitial fluid volume together constitute the functional extra cellular fluid volume (FEFV). Extra cellular fluid also includes the physiologically non-functional third space or trans cellular fluid.

The interstitial space acts as a reservoir, which can accept fluid filtered from the vascular compartment when the circulating volume is high. In situations where the circulating volume is low (haemorrhage), fluid from the interstitial space move into the vascular compartment to build up the circulating volume. The interstitial space also acts as a reservoir where proteins generated from the cells are stored before they are actually transferred to the vascular compartment through lymphatic channels.

During adolescence the volume of the interstitial space is about 20%. Adding the plasma volume of 7-10% to the interstitial volume, gives a FEFV of 27-30% in this age group. In term infants the FEFV may be as high as 45%.

Trans cellular (third space) fluid is non-functional extra cellular fluid. It is an unavailable pool of water formed by transudation of fluid from the cells and the extra cellular space. This includes fluid within the gastrointestinal tract formed during intestinal obstruction, ascites, urine, pleural effusions etc. Fluid that enters the trans cellular space is essentially lost from the FEFV.

Intracellular fluid

TBF minus the ECF gives the intra cellular fluid volume. Cell volume remains constant during administration of isotonic solution due to free movement of water from within the cells. However, cell volume may rapidly increase during administration of hypotonic solutions due to inward movement of water. Much of the intracellular water is bound to proteins. Energy is required to transport potassium

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into the cell and for the sodium to be transported outside the cell.

Renal physiology in neonates
Most of the postnatal shifts in body fluids are mediated by sodium and water excretion by the immature kidneys. At birth the glomerular filtration rate (GFR) is just 25% (20 ml/min\(^{-1}\) 1.73 m\(^{-2}\)) of the adults. The GFR rapidly increases during the first two weeks of life and then at a slower rate till it reaches adult function by the age of 2 years. But despite the low GFR, all infants can handle up to twice the normal maintenance fluid load because the negative effects exerted by the low GFR is countered by the positive effects of low concentrating and high diluting capacity of the newborn kidney.

Concentrating capacity: The concentrating capacity of an infant’s kidney is well below that of the adult. In response to water deprivation, the infant kidney can increase osmolality to a maximum of 500-600 mOsmkg\(^{-1}\). In contrast, an adult kidney can generate urine with an osmolality of 1200 mOsmkg\(^{-1}\). This is because of decreased tonicity in the medullary interstitium.

Diluting capacity: Although dehydrated newborns cannot concentrate their urine as efficiently as adults, water loaded term infants have free water clearance well above adults. After a water load, infants can excrete markedly dilute urine of 30-50 mOsmkg\(^{-1}\) in contrast to adults who can concentrate only up to 70-100 mOsmkg\(^{-1}\).

Electrolyte physiology in infants and neonates
Sodium physiology: Serum sodium is variable in the neonate and hence cannot be used as an indicator of the hydration status of the infant. The daily sodium requirement of a term infant is 2-5 meqkg\(^{-1}\)day\(^{-1}\) (table 1). Term infants, like adults, can retain sodium in the face of a negative sodium balance but have a diminished capacity to excrete excess sodium when in positive balance.

Sodium administration stimulates growth. Acute changes in sodium balance can lead to gross variations in blood pressures and intracerebral haemorrhage. Positive pressure ventilation and use of positive end expiratory pressures (PEEP) irrespective of the presence or absence of respiratory distress, is associated with natriuresis and with increased water retention and vasopressin release.

Potassium physiology: The usual recommended dose is 2-4 meqkg\(^{-1}\)day\(^{-1}\) given after the first few days of life (table 1). There has been a reluctance to administer potassium in the first two postnatal days or immediately after surgery. This is due to the fear of immature kidneys and defective renal function leading to hyperkalaemia. In critically ill infants, many factors, including increased steroid and prostaglandin secretions, high urine output and the use of diuretics, lead to a negative potassium balance. To prevent this hypokalaemia, 1-2 meqkg \(^{-1}\) day \(^{-1}\) of potassium is recommended parenterally to postoperative infants when there is adequate urine output.

Because more than 98% of the total body potassium stores are in the intracellular compartment, serum potassium levels are a poor indicator of the total potassium stores.

The daily requirements of other major electrolytes in the body are given in table 1.

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Recommended Daily Requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>20-150 meq</td>
</tr>
<tr>
<td>Potassium</td>
<td>20-240 meq</td>
</tr>
<tr>
<td>Acetate</td>
<td>20-120 meq</td>
</tr>
<tr>
<td>Chloride</td>
<td>20-150 meq</td>
</tr>
<tr>
<td>Calcium</td>
<td>5-20 meq</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>4-24 mmol</td>
</tr>
<tr>
<td>Magnesium</td>
<td>4-24 meq</td>
</tr>
<tr>
<td>Sodium</td>
<td>2-5 meqkg(^{-1})</td>
</tr>
<tr>
<td>Potassium</td>
<td>2-4 meqkg(^{-1})</td>
</tr>
<tr>
<td>Calcium</td>
<td>0.5-3 meqkg(^{-1})</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>0.5-1.5 mmolkg(^{-1})</td>
</tr>
<tr>
<td>Magnesium</td>
<td>0.25-1 meqkg(^{-1})</td>
</tr>
</tbody>
</table>

Cardiovascular considerations in infants and neonates
The incomplete development of the myocardium and the immature sympathetic nervous system makes infants and neonates more sensitive to hypovolaemia than older children and adults. Myocardial contractility, ventricular compliance and vascular tone are lower and less variable, making tachycardia the primary compensatory mechanism during volume depletion. Cardiac output decreases when the limits of tachycardia are reached. Anaesthetic depression of cardiovascular function further accentuates the effects of hypovolaemia. Thus maintenance of effective vascular volume in paediatric patients is essential to sustain circulatory function and vital organ perfusion in the perioperative period.

Determining fluid requirements

Methods
Many systems have been devised to calculate the amount of fluids, calories and minerals required for
continuing growth, maintenance during anaesthesia, replacement of fluid losses and fluid shifts and recovery from surgical stress.

**Body Surface Area (BSA) method**

BSA method of calculating the fluid and energy requirements is based on the concept that caloric expenditure is proportional to body surface area. Based on this principle, the water and electrolyte requirements are 1500 ml m$^{-2}$ day$^{-1}$, the sodium requirements are 30-50 meq m$^{-2}$ day$^{-1}$ and potassium requirements are 20-40 meq m$^{-2}$ day$^{-1}$. However, fluid requirements based on BSA are not recommended at present, as they are prone to errors.5

**Calorie consumption and body weight**

Calorie expenditure has become the standard for determining the fluid and energy requirement in children.6 Caloric requirements equal the fluid requirements in infants.7 Metabolism of 1 calorie produces 0.2 ml of water and also consumes 1.2 ml of water. Thus in an awake child the fluid and caloric consumption are considered equal. In 1957 Holliday and Segar assessed the metabolic and active energy requirements in awake hospitalized children.8 The calculated energy requirements of hospitalized infants up to 10 kgs were 100 cal kg$^{-1}$day$^{-1}$. Of this 50% was utilized for basal metabolism while the remaining 50% was utilized for growth. In children weighing more than 10 kgs, growth slowed and the caloric requirements decreased to 50 cal kg$^{-1}$day$^{-1}$ for the weights above 10 kgs (i.e. 1000 cal+50 cal kg$^{-1}$day$^{-1}$). Metabolic requirements were further reduced in children weighing more than 20 kgs. For the weights above 20 kgs, the caloric requirements were reduced to 20 cal kg$^{-1}$day$^{-1}$ (i.e. 1500 cal+20 cal kg$^{-1}$day$^{-1}$). 

Fever increases the caloric requirements by 10-12% for every centigrade rise in temperature above normal. Reduction in metabolic requirements reduces energy requirements in a similar manner.8

**Fluid management**

This is divided into 3 phases

- a. Deficit therapy
- b. Maintenance therapy
- c. Replacement therapy

**Deficit therapy**

This refers to the management of fluid and electrolyte losses that occur prior to presentation for surgery and itself has 3 components: a) estimation of dehydration severity, b) determination of fluid deficit type and c) deficit repair.

Dehydration severity is usually estimated from the history and clinical evaluation (table 2). Four pertinent investigations that will confirm the type of dehydration include:9

- a. Serum osmolarity and serum sodium
- b. Acid-base status, serum pH and base deficits
- c. Serum potassium compared with the pH
- d. Urine output (rule out acute tubular necrosis)

<table>
<thead>
<tr>
<th>Signs &amp; Symptoms</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight loss</td>
<td>3-5%</td>
<td>6-9%</td>
<td>&gt; 10%</td>
</tr>
<tr>
<td>General condition</td>
<td>Alert, restless</td>
<td>Thirsty, lethargic</td>
<td>Cold, sweaty, limp</td>
</tr>
<tr>
<td>Pulse</td>
<td>Normal rate, volume</td>
<td>Rapid, weak</td>
<td>Rapid, feeble</td>
</tr>
<tr>
<td>Respiration</td>
<td>Normal</td>
<td>Deep, rapid</td>
<td>Deep, rapid</td>
</tr>
<tr>
<td>Ant. Fontanelle</td>
<td>Normal</td>
<td>Sunken</td>
<td>Very sunken</td>
</tr>
<tr>
<td>Systolic pressure</td>
<td>Normal</td>
<td>Normal or low</td>
<td>Low, unrecordable</td>
</tr>
<tr>
<td>Skin turgor</td>
<td>Normal</td>
<td>Decreased</td>
<td>Markedly decreased</td>
</tr>
<tr>
<td>Eyes</td>
<td>Normal</td>
<td>Sunken, dry</td>
<td>Grossly sunken</td>
</tr>
<tr>
<td>Mucus membrane</td>
<td>Moist</td>
<td>Dry</td>
<td>Very dry</td>
</tr>
<tr>
<td>Urine output</td>
<td>Adequate</td>
<td>Less, dark</td>
<td>Oliguria, anuria</td>
</tr>
<tr>
<td>Capillary refill</td>
<td>Normal</td>
<td>&lt; 2 sec</td>
<td>&gt; 3 sec</td>
</tr>
<tr>
<td>Estimated deficit</td>
<td>30-50 ml kg$^{-1}$</td>
<td>60-90 ml kg$^{-1}$</td>
<td>100 ml kg$^{-1}$</td>
</tr>
</tbody>
</table>

The above clinical and biochemical investigations will reveal whether the type of dehydration is hyponatraemic (serum osmolarity <270 mOsmL$^{-1}$, serum Na <130 meqL$^{-1}$), isonatraemic (serum osmolarity 270-300 mOsmL, serum Na 130-150 meqL$^{-1}$) or hypernatremic (serum osmolarity >310 mOsmL$^{-1}$, serum Na >150 meqL$^{-1}$). However, treatment of the fluid deficit should be initiated before all the investigations are available. Initial fluid resuscitation can be initiated with a bolus of normal saline given over 10-20 minutes to improve circulation and restore renal perfusion. For patients with known contraction alkalosis, 5% Dextrose with 0.9% saline would be a reasonable fluid of choice. In patients with known metabolic acidosis, removing 250 ml of 0.9% saline from a 1 L container and replacing it with 28 ml of 7.5% sodium bicarbonate solution and 232 ml of 5% dextrose in 0.9% saline can formulate a more appropriate solution. The resulting solution contains approximately 1.2% Dextrose, 140 meq of sodium, 115 meq chloride and 25 meq of sodium bicarbonate.7 Giving lactate or acetate containing solution to children with severe metabolic acidosis can aggravate their acidosis especially if these precursors of bicarbonate cannot be metabolized to.
bicarbonate by the liver because of the poor circulatory status.

Even a 1% reduction of blood volume is associated with a rectal temperature rise of 0.3°C. The mechanism for a febrile response to volume contraction may be related to a decrease in skin blood flow, which prevents dissipation of heat. In addition, hyperosmolarity elevates the threshold for sweating. This in turn will increase their caloric and fluid requirements.

**Fluid deficit due to overnight fasting**

To avoid the complication associated with pulmonary aspiration during induction of anaesthesia, prolonged fasting has generally been advocated. Recent studies have shown that the residual gastric volume was lower and the pH higher, in children allowed clear fluids up to 2 hours before surgery. Sips of clear fluids stimulate peristalsis but do not stimulate gastric secretion if no protein is present. H₂ blockers effectively elevate the gastric pH and further reduces gastric volume. Present recommendations include administration of clear fluids up to two hours before surgery or milk feeds up to 4 hours before surgery.

In general, deficits caused by preoperative restriction of fluids are calculated by multiplying the hourly maintenance requirements times the number of hours of fluid restriction. Of the total amount, 50% is replaced in the first hour and 25% each in the next 2 hours. The fluid requirements for covering the deficits caused by preoperative fasting in neonates and older children are given in table 3. This is similar to maintenance fluid requirements during the course of the surgery.

Deficit replacement revolves around the restoration of cardiovascular function, CNS function and renal perfusion.

As mentioned earlier, deficit should be replaced with a balanced salt solution based on the type and severity of the dehydration. This should take into account the type of fluid loss from the body (table 4) and a suitable type of balanced salt solution (table 5). Total fluid replacement may take considerable time and potassium losses in particular cannot be replaced immediately. Potassium should be replaced only after adequate renal perfusion is established, acidosis corrected and child starts putting out urine.

<p>| Table - 3 : Maintenance fluid requirements in neonates and infants: Daily and hourly. |</p>
<table>
<thead>
<tr>
<th>Age (days) / Weight (kgs)</th>
<th>Requirements: mlkg⁻¹day⁻¹</th>
<th>Hourly: mlkg⁻¹hr⁻¹</th>
<th>Type of fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20-40</td>
<td>2-3</td>
<td>10% dextrose</td>
</tr>
<tr>
<td>2</td>
<td>40-60</td>
<td>3-4</td>
<td>10% dextrose in 0.22% saline</td>
</tr>
<tr>
<td>3</td>
<td>60-80</td>
<td>4-6</td>
<td>10% dextrose in 0.22% saline</td>
</tr>
<tr>
<td>4</td>
<td>80-100</td>
<td>6-8</td>
<td>5-10% dextrose in 0.22% saline</td>
</tr>
<tr>
<td>0-10 kgs</td>
<td>100</td>
<td>4 mlkg⁻¹hr⁻¹</td>
<td>5% dextrose in 0.45% saline</td>
</tr>
<tr>
<td>10-20 kgs</td>
<td>1000 + 50 mlkg⁻¹</td>
<td>40 ml + 2 mlkg⁻¹hr⁻¹</td>
<td>5% dextrose in 0.45% saline</td>
</tr>
<tr>
<td>&gt; 20 kgs</td>
<td>1500 + 20 mlkg⁻¹</td>
<td>60 ml + 1 mlkg⁻¹hr⁻¹</td>
<td>5% dextrose in 0.45% saline</td>
</tr>
</tbody>
</table>

<p>| Table - 4 : Electrolyte composition of body fluids. |</p>
<table>
<thead>
<tr>
<th>Electrolytes</th>
<th>Gastric</th>
<th>Pancreatic</th>
<th>Bile</th>
<th>Ileostomy</th>
<th>Diarrhoea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺ (meqL⁻¹)</td>
<td>70</td>
<td>140</td>
<td>120</td>
<td>130</td>
<td>50</td>
</tr>
<tr>
<td>K⁺ (meqL⁻¹)</td>
<td>5-15</td>
<td>5-5</td>
<td>15-20</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>Cl⁻ (meqL⁻¹)</td>
<td>120</td>
<td>100-100</td>
<td>120</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>HCO₃⁻ (meqL⁻¹)</td>
<td>0</td>
<td>35</td>
<td>40</td>
<td>25-30</td>
<td>50</td>
</tr>
</tbody>
</table>

<p>| Table - 5 : Composition of commonly used intravenous fluids in children. |</p>
<table>
<thead>
<tr>
<th>Electrolytes (meqL⁻¹)</th>
<th>Normal saline</th>
<th>Ringers lactate</th>
<th>Intracef A</th>
<th>Plasmalyte A</th>
<th>Dextrose 9%</th>
<th>Albumin 5%</th>
<th>Hetastarch 6%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td>154</td>
<td>130</td>
<td>26</td>
<td>140</td>
<td>-</td>
<td>145±15</td>
<td>154</td>
</tr>
<tr>
<td>K⁺</td>
<td>-</td>
<td>4</td>
<td>21</td>
<td>5</td>
<td>-</td>
<td>&lt; 2.5</td>
<td>-</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>154</td>
<td>109</td>
<td>21</td>
<td>98</td>
<td>-</td>
<td>100</td>
<td>154</td>
</tr>
<tr>
<td>Ca²⁺</td>
<td>-</td>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mg²⁺</td>
<td>-</td>
<td>3</td>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Acetate</td>
<td>-</td>
<td>-</td>
<td>24</td>
<td>27</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Lactate</td>
<td>-</td>
<td>28</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Glucose (gm%)</td>
<td>-</td>
<td>-</td>
<td>5</td>
<td>-</td>
<td>5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Phosphate (mg%)</td>
<td>-</td>
<td>-</td>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Osmolarity (mOsmL⁻¹)</td>
<td>308</td>
<td>274</td>
<td>-</td>
<td>206</td>
<td>252</td>
<td>330</td>
<td>310</td>
</tr>
</tbody>
</table>

**Maintenance fluid therapy**

The maintenance fluid meets the ongoing fluid and electrolyte requirements during the course of surgery. It does not take into account blood loss or third space loss of fluid into interstitial space or gut. Based on the Holliday-Segar calculations, the hourly maintenance fluid requirements should be replaced (table 3).
The fluids for maintenance therapy replace losses from two sources:
1. Insensible losses (evaporative loss)
2. Urinary loss.

Evaporative loss is composed of solute free loss of water through the skin and lungs. Under ordinary conditions this accounts for 30-35% of the total maintenance requirements. Insensible losses are affected by ambient humidity and temperature, gestational age of the infant, type of respiration and surface area exposed. Ventilation with humidified gases results in significantly lower insensible loss.

In a euvoalaemic state, urinary loss concentrations range from 280-300 mOsm/kg of water, with a specific gravity of 1.008-1.015. In some circumstances (eg. diabetes insipidus, premature infants), an obligatory production of dilute urine exists, and appropriate increases in maintenance fluid requirements should be made. On other occasions (excessive ADH secretion), a patient may be unable to decrease urine osmolality to 300 mOsm/kg of water, and the volume of maintenance fluids should be decreased. If the estimate of the maintenance fluid requirements is correct, the patient’s electrolyte levels should remain stable and the patient should remain clinically euvoalaemic.

Glucose requirements in maintenance fluids: In addition to the lower body stores of glycogen, infants have a higher metabolic rate and oxygen consumption than older children. Neonatal surgery, particularly pre-bypass surgery can induce significant life threatening hypoglycemia. However, intraoperative hypoglycemia is exceedingly rare in children. On the other hand, hyperglycemia is more commonly encountered during anesthesia and surgery. Intraoperative glucose uptake by the muscle is reduced. The response to anaesthesia, surgery, anxiety and pain further increase the blood sugar levels. Hyperglycemia may be further aggravated by the impaired effectiveness of insulin during anesthesia. One large study involving 238 children of varying age groups, undergoing preoperative starvation for variable periods, did not result in low blood sugar levels. A glucose administration rate of more than 10 mg/kg/min may overwhelm the renal threshold and result in glycosurea and osmotic diuresis.

With decreased tolerance to exogenous glucose and increased endogenous glucose production, a solution containing low concentrations of glucose in balanced salt solution may be required as maintenance fluid. The replacement fluid should either be free of dextrose or should not have more than 1% dextrose. Other studies have similarly confirmed the usefulness of low concentrations of glucose in the maintenance fluid. The present recommendations include the use of low dextrose containing solutions for maintenance fluid therapy. This would ensure adequate blood sugar levels without inducing hyperglycemia.

Replacement therapy

Replacement therapy is designed to replace ongoing abnormal fluid and electrolyte losses. Because the constituents of replacement fluid are usually different from that of the maintenance fluid, simply increasing the volume of maintenance fluid for the losses may be harmful.

Replacing fluid losses with balanced salt solution leads to less fluid retention and a natriuretic response is induced. In most patients, Ringer's lactate solution is a reasonable choice as the replacement fluid and is less expensive than other balanced electrolyte solutions. Normal saline with its higher sodium content may be preferable in children at risk of cerebral edema. Three factors are active in the restoration of fluid homeostasis when using a balanced salt solution: Fluid balance is sensed by volume receptors and osmoreceptors. Infusing a balanced salt solution that contains a relative excess of sodium replaces water while increasing total body sodium. This relative sodium excess helps to maintain circulation and to replete the FEFV. Increases in vascular capacitance stretch the cardiac atrium and increases renal blood flow. Natriuretic hormone further increases blood volume. By increasing renal blood flow and stimulating sodium excretion, body fluid homeostasis (via a diuretic and natriuretic response) re-equilibrates, which reduces peri-operative fluid retention. Excess water on the other hand produces tissue edema. This in turn hampers the transcellular flow of nutrients and outflow of waste from the cells, which ultimately leads to a vicious cycle leading to cell death.

Third space loss

Surgical trauma leads to fluid translocation from the FEFV to a non-functional compartment and these deficits must be replaced in order to maintain an adequate FEFV. Functionally this fluid is neither available to the vascular compartment nor the FEFV. The composition of this fluid is similar to that of the ECF in addition to a small amount of proteins. A balanced salt solution like Ringer’s lactate is again the preferred fluid for replacement of the third space loss. The replacement for third space losses depends on the severity of the surgical trauma. Table 6 shows the guidelines for replacing third space losses.
Table 6: Replacement for third space and evaporative losses.

<table>
<thead>
<tr>
<th>Surgical trauma</th>
<th>Type of surgery</th>
<th>Fluid replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimal</td>
<td>Inguinal hernia repair</td>
<td>1 – 2 mlkg⁻¹hr⁻¹</td>
</tr>
<tr>
<td>Moderate</td>
<td>Ureteral implantation</td>
<td>4 mlkg⁻¹hr⁻¹</td>
</tr>
<tr>
<td>Severe</td>
<td>Spinal and bowel obstruction</td>
<td>&gt; 6 mlkg⁻¹hr⁻¹</td>
</tr>
</tbody>
</table>

Replacement of blood loss

In children, all blood loss should be replaced. Replacement of blood loss can be either in the form of packed red cells, whole blood, colloids or crystalloids. The clinical evaluation of the “amount of blood loss” can be on simple clinical judgment, swab weighing or laboratory data. “Davenport’s law” is simple to apply, mainly for those who do not deal with children frequently; under 10% of blood loss no blood is required, over 20% losses must be replaced with either packed red cells or whole blood and between 10-20% we must consider case by case. Davenport’s law is questionable and not generally followed.

When a crystalloid is used for replacing blood loss, for each ml of blood lost, 3 ml of crystalloid should be replaced. Being a balanced salt solution it rapidly distributes to the extra cellular space and only 20-30% remains in the intravascular compartment. An adequate oxygen transport should be ensured. An acceptable hematocrit that ensures an adequate oxygen transport depends on various factors including age, duration of surgery, and expected further blood loss. However, a minimum haematocrit of 30% in older children and 40% for neonates is generally agreed upon.

Crystalloid or Colloid: The Controversy.

In spite of the controversy surrounding the use of a crystalloid or colloid, most agree that parenteral fluid administration should start with a balanced salt solution. The major disadvantage with the use of balanced salt solution is that within a period of two hours most of the administered solution distributes to all the active fluid compartments of the body. Therefore, haemorrhagic fluid losses require three to four times the volume as opposed to whole blood replacement.

Human albumin or synthetic colloids are recommended by some to maintain the intravascular compartment. In theory, administration of albumin should raise the oncotic and osmotic pressure, which would mobilize the intra cellular and interstitial fluid into the vascular compartment and by paralyzing the distal tubule would resolve the tissue edema and maintain the intravascular compartment.

However, this effect is rarely seen, particularly in patients with capillary leak syndrome, which prevents the albumin from remaining in the intravascular compartment. Albumin is most appropriately used during major surgery to maintain vascular volume.

Hetastarch is a group of compounds that has an intravascular life upwards of 3 hours and some particles remain in the vascular compartment for many days. The daily recommended dose should not exceed 20 mlkg⁻¹. A hybrid solution containing 6% Hetastarch in a balanced salt solution is also available. This combines the advantages of a colloid kick with the buffer in lactated Ringer’s solution.†

Table 4 gives an idea of the various crystalloids and colloids available for use in various situations.

Electrolyte imbalance in the perioperative period

Disturbances in sodium physiology

Sodium is the most abundant cation in extra cellular fluid and is critical in determining the extra cellular and intra cellular osmolality. Sodium requirements may vary with age. Sodium requirements for term infants is 2-3 meqkg⁻¹day⁻¹. Neonatal stool losses are 1 meqkg⁻¹day⁻¹ and growth requirements are about 0.5 meqkg⁻¹day⁻¹.

Hyponatraemia

Serum sodium concentration is less than 130 meqL⁻¹. This is a common perioperative electrolyte disturbance. It can be of different types based on the volume status of the patient.

1. Hypovolaemic hyponatraemia: There is a reduction in extra cellular fluid volume e.g. gastroenteritis, diuretic therapy or renal losses. Hypovolaemic hyponatraemia causes cerebral edema.
2. Hypervolaemic hyponatraemia. There is excessive extra cellular fluid volume eg. nephrotic syndrome.

Hyponatraemia is usually asymptomatic but obtundation and seizures can occur if the serum sodium level becomes very low (< 120 meqL⁻¹). Cardiac symptoms occur at levels below 100 meqL⁻¹.

Management

Appropriate therapy requires identification of the underlying cause. In hyponatraemia with decreased circulating volume, treatment is with administration of
supplemental volume. Patients with symptomatic hyponatraemia and clinical euvolaemia or hypervolaemia require infusion of hypertonic saline (3% or 5% saline). It has been shown that even small increases in serum sodium of the order of 5% can reduce cerebral edema or stop seizures. Correction should be of sufficient pace so as to reverse the manifestation of hyponatraemia but at the same time it should not pose a threat of demyelination. Rapid treatment of hyponatraemia can lead to central pontine myelinolysis. It is characterized by insidious onset of flaccid quadriplegia and cranial nerve abnormalities. Hypervolaemic hyponatraemia indicates excess body water and is often treated with a combination of Frusemide and hypertonic saline. The formula detailed below tells the change in serum sodium that can be expected after the infusion of 1 Litre of the solution.

\[
\text{Change in serum Na}^+ = \text{Infusate Na}^+ - \frac{\text{Serum Na}^+}{\text{TBW}+1}
\]

Estimates the effect of 1 litre of any infusate on serum Na\(^+\).

Total body water ((TBW) in litres)=Body weight x 0.6.

The infusate sodium for 5% saline is 855 meqL\(^{-1}\), 3% saline is 513 meqL\(^{-1}\) and normal saline is 154 meqL\(^{-1}\). Half the deficit can be corrected over 12-14 hours and the remaining over the next 1-3 days. Recommended indications for stopping the rapid correction of symptomatic hyponatraemia includes cessation of life threatening manifestations, moderation of symptoms or achievement of a serum sodium concentration of 125 to 130 meqL\(^{-1}\). It is generally agreed that the targeted rate of correction should not exceed not more than 8 meqL\(^{-1}\) on any day.

Hyponatraemia

Hyponatraemia represents a deficit of water in relation to the body stores of sodium, which can result from a net water loss or hypertonic sodium gain. It can occur in the absence of a sodium deficit or in its presence. Hyponatraemia is defined as serum sodium levels in excess of 145 meqL\(^{-1}\). In children this may be associated with muscle weakness, hyperpnoea, restlessness, insomnia, lethargy and coma. Brain shrinkage can lead to cerebral haemorrhage.

Management : Of hyponatraemia is two pronged. The initial step is to identify and control the underlying cause (gastrointestinal losses or hyperglycemia induced diuresis). The amount of fluid that should be given to correct the hyponatraemia is given by the formula:

\[
\text{Change in serum Na}^+ = \text{Infusate Na}^+ - \frac{\text{Serum Na}^+}{\text{TBW}+1}
\]

Estimates the effect of 1 litre of any infusate on serum Na\(^+\).

Total body water ((TBW) in litres)=Body weight x 0.6.

Disturbances in potassium physiology

Potassium is the most abundant cation in the intracellular fluid. Potassium levels are influenced by insulin, pH of the blood and tissues, b-adrenergic agonists and aldosterone.

Hypokalaemia

This is a common postoperative finding and usually caused by gastrointestinal (diarrhoea, vomiting, villous adenoma) or renal losses (diuretics, chronic metabolic alkalosis, renal tubular acidosis) or abnormal electrolyte shifts induced by drugs (b-adrenergic agonists, alkalosis, insulin). Clinical symptoms include lethargy, muscle weakness, ECG changes and ventricular arrhythmias.

Management : Emergency management is indicated when the hypokalaemia is associated with cardiac arrhythmias. Rate of intravenous correction should not exceed 0.2 to 0.5 meqkg\(^{-1}\)hour\(^{-1}\). Deficits can be calculated from the formula:

\[
\text{Potassium deficit (meqL}^{-1}) = \text{Body weight} \times (\text{Expected serum K}^+ - \text{observed serum K}^+) \times 0.3
\]

Intravenous potassium should always be administered through a central line. The serum potassium levels should be monitored at very close intervals and the electrocardiogram should always be monitored.

Hyperkalaemia

Serum potassium levels in excess of 5.5 meqL\(^{-1}\) leads to hyperkalaemia. The condition requires immediate attention and may be fatal if not managed on an emergency basis. In the surgical setting the most common causes for hyperkalaemia include acute renal failure, metabolic acidosis, exogenous administration of potassium, effects of
cardioplegia during cardiac surgery, stored blood transfusions and extensive tissue necrosis. In a perioperative setting, succinyl choline has been implicated to cause potassium release from depolarized muscle tissue. This can lead to an increase in the serum potassium by 0.5 to 1 meqL⁻¹ in a normal patient; this may be disastrous in a situation where there is a pre-existing hyperkalaemia. Succinyl choline induced hyperkalaemic cardiac arrest was reported in apparently healthy children, almost half of whom had received a diagnosis of neuromuscular disorder. Acidosis is another common cause for hyperkalaemia. For every 0.1 unit decrease in serum pH, the serum potassium increases by 0.2 to 0.4 meqL⁻¹.

ECG changes associated with hyperkalaemia include:
- 5.5-6.5 meqL⁻¹: tall “T” waves
- 6.5-8 meqL⁻¹: small “p” and widening of QRS complex
- 8-9 meqL⁻¹: disappearance of P waves, QRS and T merge to form sine waves
- >9 meqL⁻¹: ventricular tachycardia, ventricular fibrillation, atrio-ventricular dissociation and cardiac standstill.

Management: Immediate therapy for hyperkalaemia is to prevent life threatening cardiac arrhythmias. If the patient is hemodynamically stable without ECG changes, then a recheck of the values is reasonable. If ECG changes are present, any exogenous source of potassium should be replaced with saline. This includes any potassium containing maintenance or replacement fluids. Intravenous calcium chloride 10-20 mgkg⁻¹ or calcium gluconate 60 mgkg⁻¹ will stabilize the myocardium and serve as a physiological antagonist to potassium. Correction of any metabolic acidosis with sodium bicarbonate will shift the potassium into the intracellular compartment. Intravenous glucose-insulin (0.1 Ukg⁻¹ of insulin with 0.5 gmkg⁻¹ of glucose) will have a similar effect.

Disturbances in calcium physiology
Calcium plays a key role in bone formation, in cell division, growth, coagulation and excitation contraction coupling of muscle tissue. Body contents of calcium in infants are 400 meqkg⁻¹ whereas it is 950 meqkg⁻¹ in adults. Body reserves of calcium are very low in preterm infants compared with those of term infants and hypocalcaemia occurs in about 90% of preterm infants.

Hypocalcaemia
Hypocalcaemia is defined as a serum calcium less than 4.5 meqL⁻¹. Although there are a number of non surgical causes of hypocalcaemia (hypoparathyroidism, Vit. D deficiency, pancreatitis etc), the common causes for hypocalcaemia in the operating room is administration of blood in excess of 1.5 mlkg⁻¹min⁻¹ or due to acute hyperventilation. Low albumin levels can result in manifestation of symptoms of hypocalcaemia. Symptoms include neuromuscular irritability, weakness, paraesthesia, cardiac dysrrhythmias and prolonged QT interval in the ECG and carpo-pedal spasm.

Management: Parenteral calcium therapy for hypocalcaemia in preterm neonates can significantly improve their myocardial performance, particularly in patients with impaired myocardial function. Treatment includes correction of the underlying cause and intravenous infusion of calcium chloride (20 mgkg⁻¹) or an equivalent dose of 10% calcium gluconate (60 mgkg⁻¹). In these concentrations, the chloride and gluconate forms of the calcium salt are equally effective for increasing the ionized calcium concentrations in children.

Hypercalcaemia
Hypercalcaemia is commonly seen in patients with hyperparathyroidism, Vit. D intoxication, prolonged immobilization, use of thiazide diuretics, milk alkali syndrome, errors in total parenteral nutrition malignancies etc.

Management: The clinical picture dictates the management. Gastrointestinal symptoms of hypercalcaemia include anorexia, nausea, vomiting and constipation. Hypertension, augmentation in digoxin toxicity, renal dysfunction and central nervous system disturbances including coma are also seen during hypercalcaemia.

Management requires immediate hydration with normal saline. Loop diuretics, biphosphonates, plicamycin, calcitonin, steroids, phosphates and prostaglandin all have their place in chronic and acute therapies to reduce serum calcium.

Disturbances of Magnesium physiology
Magnesium is the fourth most abundant cation in the body. Because of its relative intra cellular abundance, it plays a major role in cellular enzyme regulation. Body magnesium content is 22 meqL⁻¹ in adults. Body magnesium content is 22 meqL⁻¹ in infants and 28 meqL⁻¹ in adults. Bone and muscle cells are major intra cellular pools of magnesium. Normal levels range from 1.5 to 1.8 meqL⁻¹.
Hypomagnesaemia

Magnesium deficiency is rare in newborns. In older infants it is associated with prolonged use of magnesium free hyper alimentation solutions. It is a common finding in critically ill children. Sixty five percent of patients in a critical care setting have magnesium deficiency, which may be aggravated by epinephrine infusions. Other causes include hyperaldosteronism, intestinal fistulas, starvation, pancreatitis and use of b-adrenergic agonists. Calcium deficiencies often accompany magnesium deficiencies. Manifestations include increased neuromuscular irritability, tetany, seizures, tremors and hyperreflexia. In older children, serum total magnesium levels less than 0.7-0.8 mmolL⁻¹, and for neonates, serum total magnesium levels less than 0.6 mmolL⁻¹ are recommended to diagnose hypomagnesaemia.

Magnesium has important cardiovascular and non-cardiovascular functions. Its important cardiovascular functions include energy metabolism in adenosine triphosphate dependant processes (eg. myocardial contraction), maintenance of cell membrane electrical potential and regulation of cardiovascular function. The primary risk to patients with hypomagnesaemia is ventricular dysrhythmias. Ventricular tachycardia, fibrillation and torsades de pointes can all occur with hypomagnesaemia.

Management: Treatment is with administration of intravenous magnesium sulphate 15-30 mgkg⁻¹ intravenously over 15–30 minutes.

Hypermagnesaemia

High magnesium concentrations are present in newborns of mothers getting magnesium salts for treatment of toxemia of pregnancy. These salts can cause sedation and may potentate muscle relaxants in parturients and infants. High magnesium concentrations can also result from renal failure, administration of magnesium containing laxatives and antacids.

Management: Any supplementary magnesium should be stopped. Elimination of magnesium involves fluid loading followed by or with concomitant diuresis. Definitive therapy involves dialysis. Temporary reversal of effects of magnesium can be managed with calcium therapy. As hypomagnesaemia potentiates the effects of depolarizing and non depolarizing muscle relaxants, these agents must be carefully titrated in conjunction with appropriate assessment of neuromuscular blockade.

Conclusion

Appropriate perioperative fluid management is essential for the paediatric patient. Management must be individualized, taking into account developmental and physiological considerations. The caloric method is appropriate for estimating maintenance fluid requirements, but the clinical response is the best indicator of adequate therapy. Preoperative fasting should be confined to the minimum time compatible with patient safety. Dextrose containing solutions are best confined to children who are at risk of hypoglycemia, particularly preterm infants and neonates. Restoration of the circulating volume and vital organ perfusion is the first priority in perioperative fluid management and is best accomplished with isotonic crystalloid. Therapy for perioperative fluid and electrolyte disorders maybe guided by serum sodium and potassium concentrations, volume status, urine output and urinary loss of electrolytes. Symptomatic hyponatraemia and hyperkalaemia are the electrolyte disturbances that warrant emergency management.

References


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