REVIEW ARTICLE

'ROSE concept' of fluid management: Relevance in neuroanaesthesia and neurocritical care

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Abstract

Fluid therapy in neurosurgical patients aims to restore intravascular volume, optimise haemodynamic parameters and maintain tissue perfusion, integrity and function. The goal is to minimise the risk of inadequate cerebral perfusion pressure and to maintain good neurosurgical conditions. However, fluid management in brain-injured patients has several distinctive features compared with non-brain-injured critically ill patients. The ROSE concept advocates the restriction of fluids, which is consistent with the prevention of a 'tight brain' in neurosurgery. Whether this imbalance in fluid management studies between different types of brain injuries is a reflection of differences in clinical relevance of fluid management is not clear. Further randomised controlled trials in the future are essential in subarachnoid haemorrhage and traumatic brain injury patients who are critical and need long-term Intensive Care Unit stay to elucidate and define the role and relevance of the ROSE concept in neuroanaesthesia and neurocritical care.

Key words: De-resuscitation, fluid overload, resuscitation

INTRODUCTION

Fluid therapy in neurosurgical patients aims to restore intravascular volume, optimise haemodynamic parameters and maintain tissue perfusion, integrity and function. The goal is to minimise the risk of inadequate cerebral perfusion pressure (CPP) and to maintain good neurosurgical conditions. However, fluid management in brain-injured patients has several distinctive features compared with non-brain-injured critically ill patients. Both the type of fluids used and the volume is very important. The use of hypotonic fluids can lead to tissue oedema, which results in oxygen diffusion and cerebral blood flow (CBF) impairments.[1] Fluid management in other critically ill patients is commonly guided by haemodynamic monitoring, while sophisticated monitoring tools for CBF, and cerebral oxygenation are generally less well implemented in clinical practice. Recent data suggest that the fluid administration may have an impact on outcome.[2-4]

Positive fluid balance is associated with worse morbidity and mortality in multiple studies which show worse overall mortality in critically ill patients,[5] and increased mortality in patients with acute kidney injury,[4] as well as prolonged recovery in patients with acute lung injury/acute respiratory distress syndrome (FACTT trial). Increased mortality in septic shock patients (VAST trial) and worse morbidity in colorectal surgery patients has been demonstrated. It is also associated with intra-abdominal hypertension.[5]

The ROSE concept has been advocated by Malbrain *et al.*,[5] after reviewing the association between a positive

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fluid balance and fluid overload and the outcomes in critically ill adults. Positive fluid balance is a state of fluid overload resulting from fluid administration during resuscitation and subsequent therapies. Fluid overload is defined by 'a cut off value of 10% of fluid accumulation as this is associated with worse outcomes'.[5]

The percentage of fluid accumulation can be defined 'by dividing the cumulative fluid balance in litre by the patient's baseline body weight and multiplying by 100%'.[5]

These fluid management strategies can be used as guidelines for neurosurgical patients but need to be individualised and guided by an understanding of the underlying normal physiological and pathophysiological mechanisms.

ANATOMY AND PHYSIOLOGY

Water comprises 60% of the total body weight of an adult, and is divided functionally into the extracellular (extracellular fluid [ECF] = 20% of body weight) and the intracellular fluid spaces (ICF = 40% of body weight) [Figure 1]. It is separated by the cell membrane with its active sodium pump, which ensures that sodium remains mainly in the ECF. The cell contains large anions such as protein and glycogen, which cannot exit and therefore draw in K^+ ions to maintain electrical neutrality (Gibbs–Donnan equilibrium). These mechanisms ensure that Na^+ and its anions Cl^- and HCO^-_3, are the mainstay of the ECF osmolality, and K^+ has the corresponding function in the ICF.[6]

FLUID MOVEMENTS BETWEEN VASCULATURE AND TISSUES

Ernest Starling described the forces that determine the movement of water between vasculature and tissues,

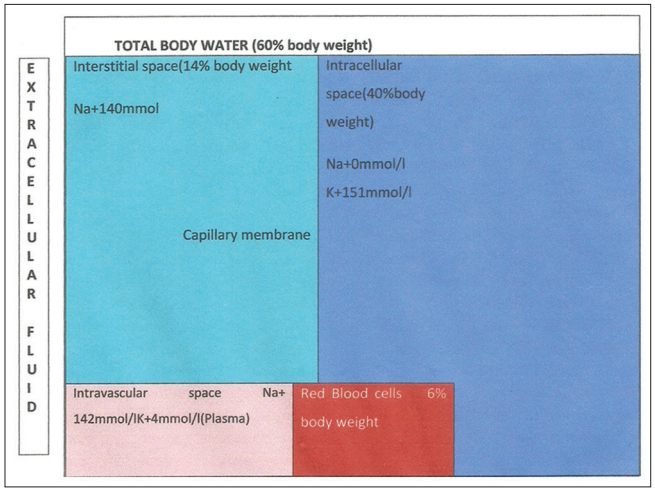

Figure 1: Distribution of total body water

which was formalised subsequently into Starling's equation, as follows:

$$Q_f = K_f S ([P_c - P_t] - \delta[\pi_c - \pi_t])$$

Where Q_f is the net amount of fluid that moves between the capillaries and the surrounding extracellular space, K_f is the filtration coefficient for the membrane, S is the surface area of the membrane, P_c is the hydrostatic pressure in the capillary membrane, P_t is the hydrostatic pressure of the surrounding tissue, δ is the coefficient of reflection which can vary from 1 = no movement to 0 = free diffusion of the solute across the membrane, π_c is the oncotic pressure of the plasma and π_t is the oncotic pressure of the fluid in the extracellular space.

The capillary pressure, the tissue pressure and the tissue oncotic pressure all act to draw fluid from the capillaries into the extracellular space of the tissue.

In peripheral tissues only the plasma oncotic pressure serves to maintain intravascular volume, this plasma oncotic pressure is produced pre-dominantly by albumin, immune globulins, fibrinogen, and other high molecular weight plasma proteins. Normally, the sum of the forces results in a Q_f value slightly >0, indicating an outward flux of fluids from the vessel into the tissue extracellular space. This fluid is cleared from the tissue by the lymphatic system, thereby preventing the development of oedema.

FLUID MOVEMENTS BETWEEN THE CAPILLARIES AND THE BRAIN

The brain and the spinal cord are isolated from the intravascular compartment by the blood-brain barrier, which composed of endothelial cells that form tight junctions severely limiting the diffusion of molecules between the intravascular space and the brain. Osmolarity is the primary determinant of water movement across the intact blood-brain barrier.[7] The administration of excess free water results in an increase in the intracranial pressure and an oedematous brain.[8] Conversely, the administration of hyperosmolar crystalloids (e.g., mannitol) results in decrease in brain water content and ICP.

Cerebral autoregulation and the blood brain barrier are two protective mechanisms that usually preserve normal cerebral function. Either or both may be impaired in a patient with neurologic injury.

CBF is determined by cerebral metabolic needs. Cerebral autoregulation ensures that CBF and cerebral oxygen delivery is constant in spite of moderate changes in systemic blood pressure, CPP, haematocrit, blood viscosity, partial pressures of arterial oxygen and carbon

dioxide. If cerebral autoregulation fails, the CBF and oxygen delivery is compromised and is then dependent on CPP and viscosity.

The blood-brain barrier is composed of brain endothelial cells with tight junctions creating an effective pore size of 0.7–0.9 nm.[7] These tight junctions do not permit the passage of even small molecules and ions. It is permeable only to water, hence small molecules and ions produce the osmotic gradient.[8] Thus, even small increases in the concentrations of plasma electrolytes can exert a large osmotic pressure gradient across the blood brain barrier and redistribute water to the intravascular space. In the normal brain changes in oncotic pressure do not significantly influence the osmotic pressures in the brain. However, if the blood brain barrier is damaged secondary to various types of trauma it becomes permeable to a variety of molecules, and then hydrostatic pressure becomes important in determining brain water.

FLUID MANAGEMENT

Fluid maintenance

Maintenance solutions are given to provide the daily requirements of water and electrolytes. In healthy adults, sufficient water is required to balance gastrointestinal losses of 100–200 ml/day, insensible losses (respiratory and cutaneous) of 500–1000 ml/day. Urinary output exceeding 1000 ml/day may represent an appropriate physiologic response to extracellular volume expansion or an inability to conserve salt or water. The daily adult requirement for sodium is 75 mEq (1.5 mEq/kg/day). Patients with a normal cardiac and renal reserve are capable of markedly increased excretion or extreme conservation (<10 mEq excreted/day). A daily requirement for potassium is 1 mEq/kg/day. Physiologic diuresis typically induces an obligate potassium loss of at least 10 mEq/L of urine. To estimate maintenance water requirements using body weight, provide 4 ml/kg/h for the first 10 kg, an additional 2 ml/kg/h for 11–20 kg, and 1 ml/kg/h for each additional kilogram after 20 kg. Therefore, the daily maintenance requirements for water, sodium and potassium for a healthy 70 kg adult consist of 2500 ml/day of a solution containing sodium 30 mEq/L and potassium 25–30 mEq/L.[10]

Fluid resuscitation

Neurosurgical procedures and isolated head injury are associated with minimal loss of ECF, although associated injuries may necessitate aggressive fluid replacement. Surgical and traumatic losses in patients at risk for intracranial hypertension can be replaced with 0.9% saline. The findings of '0.9% saline versus 4% albumin fluid evaluation' study suggest that colloids are associated with adverse outcomes as compared to crystalloids in traumatic brain injury (TBI) patients.[11] Hypoosmolar solutions such as albumin, lactated ringer and some gelatin solutions increase brain volume and intracranial pressure.

More recent data suggest improved outcomes with balanced salt solutions, as compared to 0.9% saline.[12,13] Substantial or chronic loss of gastrointestinal fluids requires replacement of other electrolytes (i.e., potassium, magnesium, phosphate). Replacement of fluid losses also must compensate for sequestration of interstitial fluid that accompanies trauma, haemorrhage and tissue manipulation.

The simplest formula provides in addition to maintenance fluids and replacement of estimated blood loss, 4 ml/kg/h for procedures involving minimal trauma, 6 ml/kg/h for those involving moderate trauma and 8–15 ml/kg/h for those involving severe trauma.

ROLE OF HYPERTONIC SALINE

The use of hypertonic saline, an effective plasma volume expander with decreased oedemagenesis, in resuscitation in patients in hypovolemic shock has been growing over the past decades. Intravascular administration of hypertonic saline creates an osmotic gradient resulting in the transfer of fluid from the intracellular space to the intravascular space. In head trauma, it has been shown to be effective for volume resuscitation, with a beneficial effect on intracranial pressure and decreasing brain oedema in uninjured areas. It does not cause the hypotension seen when mannitol is used and increases cardiac output, decreases peripheral resistance and decreases intracranial pressure. Its use is economical and free from infectious risk. Its use has been associated with increases in plasma osmolarity and sodium and chloride levels and hypokalaemia. Cerebral dehydration and central pontine myelinolysis due to rapid changes in serum sodium levels are possible.[14] Hypertonic-hyperoncotic fluids have the potential advantage in acute resuscitation in that small volumes are more effective than isotonic crystalloids in maintaining blood pressure and cardiac output. Recommendations for the use of hypertonic saline are close monitoring of serum sodium levels (<60 mEq/L) and serum osmolarity (<350 mosm/L).

BLOOD TRANSFUSION

Many neurosurgical as well as non-neurosurgical procedures and neurotrauma can be associated with significant bleeding. Perioperative transfusion helps improve cerebral oxygen delivery. Transfusion thresholds are undefined, and clinical studies have shown that subarachnoid haemorrhage (SAH) and TBI patients have a worse outcome with anaemia. Fresh

frozen plasma should be given only to correct any coagulopathy and not as a volume expander.

The beneficial effects of haemodilution are based on the correlation of haematocrit and whole blood viscosity. With decrease in haematocrit and viscosity, the cerebrovascular resistance decreases and CBF increases. However, it is important to keep in mind that the oxygen-carrying capacity of the blood will also decrease. It is suggested that a haematocrit of 28%–33% provides an optimal balance between viscosity and oxygen carrying capacity.

Intravenous fluids are given during resuscitation to increase venous return and stroke volume, but <50% of haemodynamically unstable patients are fluid responders following a fluid challenge. Any further fluid administration not only serves no purpose in those patients that do not respond to the initial fluid bolus, but it may also be harmful them. The fluid redistributes within 60 min, and the stroke volume returns to baseline.[9] Excessive fluid at this stage can result in tissue oedema, which impairs oxygen, distorts tissue architecture, and impedes capillary blood flow and lymphatic drainage. This compromises blood flow in encapsulated organs such as kidney and liver, as well increase intra-abdominal pressure (IAP).

Studies show that a positive fluid balance is independently associated with impaired organ function and an increased risk of death, while the negative fluid balance is associated with improved organ function and survival.[15]

PATHOPHYSIOLOGY OF FLUID OVERLOAD

In response, pro-inflammatory cytokines and stress hormones after injury the Ebb Phase is initiated which represents a distributive shock, characterised by arterial vasodilatation and transcapillary albumin leakage. Secondary interstitial oedema leads to systemic hypoperfusion and impaired tissue oxygenation. In this stage of shock, adequate fluid therapy is required to prevent the development of multiple organ dysfunction syndrome. Compensatory neuroendocrine reflexes and potential renal dysfunction result in sodium and water retention and positive fluid balances. Patients with higher severity of illness need more fluids, and therefore fluid balance may be considered a biomarker of critical illness, as proposed by Bagshaw and Bellomo.[16] Subsequent haemodynamic stabilisation and restoration of plasma oncotic pressure sets off the Flow Phase with the resumption of diuresis and mobilisation of extravascular fluid resulting in negative fluid balances. However, some non-responders stay in the Ebb Phase and progress to global increased permeability syndrome (GIPS), characterised by high capillary leak index ([CLI] expressed as the ratio of C-reactive protein over albumin), excess interstitial fluid and persistent high extravascular lung water index (EVLWI), no late conservative fluid management (LCFM) achievement, resulting in positive fluid balances and progression to organ failure and death. These patients require restrictive fluid strategies and even fluid removal guided by extended haemodynamic monitoring including lung water measurements (late goal-directed fluid removal [LGFR]). Restrictive fluid strategies may necessitate a greater use of vasopressor therapy, resuscitation with hyperoncotic solutions (e.g., albumin 20%) and early initiation of diuretics and renal replacement therapy.

There is literature to show that a negative fluid balance increases survival in patients with septic shock.[15] Patients managed with a conservative fluid strategy also seem to have improved lung function, shorter duration of mechanical ventilation and intensive care stay without increasing non-pulmonary organ failure. The adverse effects of fluid overload and interstitial oedema have an impact on all end organ functions, particularly in the lungs. Therefore, monitoring of EVLWI provides information on lung water and tissue oedema, and can guide fluid management in the critically ill. A high EVLWI indicates a state of capillary leakage, associated with higher severity of illness and mortality.[17]

MALBRAIN *ET AL.*'S 'ROSE' CONCEPT OF PHASES OF CRITICAL ILLNESS

It has four phases' resuscitation (R), optimisation (O), stabilisation (S) and evacuation (E), which have been described below.[5]

Resuscitation phase (R)

Occurs within minutes of severe body injury such as sepsis, burns, pancreatitis or trauma, and the patient enters the Ebb Phase of shock. It is characterised by low mean arterial pressure (MAP), low CO, and microcirculatory impairment leading to decreased tissue perfusion and oxygenation. Fluids should be given at a rate of 1 mL/kg/h in combination with replacement fluids when indicated. Correct monitoring is essential before fluids administration. The use of non-invasive or minimally invasive cardiac output monitors is recommended to assess fluid responsiveness. This phase corresponds of the 'R' or resuscitation within the ROSE concept.

- Salvage or rescue treatment with fluids administered quickly as a bolus (4 mL/kg over 10–15 min)
- The goal is early adequate goal-directed fluid management, fluid balance must be positive and the suggested resuscitation targets are: MAP > 65 mmHg, cardiac index (CI) >2.5 L/min/m^2, pulse pressure variation (PPV) <12%, left end-diastolic area index (LVEDAI) >8 cm/m^2.

Optimisation phase (O)

Occurs within hours and is the phase of ischaemia and reperfusion. Positive fluid balance seen during this phase which a biomarker of the severity of illness. Thermodilution methods may be used to monitor preload and EVLWI. The goal is to ensure adequate tissue perfusion with titration of fluids to maintain a neutral fluid balance:

- Targets: MAP >65 mmHg, CI >2.5 L/min/m^2, PPV <14%, LVEDAI 8–12/cm/m^2, IAP (<15 mmHg) is monitored and abdominal perfusion pressure (APP) (>55 mmHg) is calculated. Preload optimised with global end-diastolic volume index (GEDVI) 640–800 mL/m^2.

Stabilisation phase (S)

This phase evolves over days and fluid is needed for maintenance and replacement of normal losses:

- Monitor daily body weight, fluid balance and organ function
- Targets: Neutral or negative fluid balance; EVLWI <10–12 mL/kg PBW, pulmonary vascular permeability index <2.5, IAP <15 mmHg, APP >55 mmHg, colloid oncotic pressure (COP) >16–18 mmHg, and CLI <60.

Evacuation phase (E)

There may be some patients who do not transition from the 'ebb' phase of shock to the 'flow' phase after the '2nd hit' develop GIPS. They require LGFR ('de-resuscitation') to achieve negative fluid balance:

- Need to avoid over-enthusiastic fluid removal resulting in hypovolaemia
- Diuretics or renal replacement therapy (in combination with albumin) can be used to mobilise fluids in haemodynamically stable patient.

De-resuscitation is characterised by the discontinuation of invasive therapies leading to a negative fluid balance. Furthermore, refers to 'LGFR', which involves 'aggressive and active fluid removal by means of diuretics and renal replacement therapy with net ultrafiltration'.

RECOMMENDATIONS

'A goal of a 0 to negative fluid balance by day 3 and to keep the cumulative fluid balance on day 7 as low as possible (Grade 2B)'.[5]

'Diuretics or renal replacement therapy (in combination with albumin) can be used to mobilise fluids in haemodynamically stable patients with intra-abdominal hypertension and a positive cumulative fluid balance after the acute resuscitation has been completed, and the inciting issues/source control have been addressed (Grade 2D)'.[5]

Cordemans *et al*.[17] suggested the 'PAL' approach as a method of de-resuscitation:

- High PEEP for 30 min (at least equal to IAP) – to drive fluid from the alveoli into the interstitium
- Albumin administration (e.g., 2 × 100 mL 20% albumin over 60 min on day 1, then titrated to albumin >30 g/L) – to pull fluid from the interstitium into the circulation
- Frusemide ('Lasix') infusion started 60 min after albumin at 60 mg/h for 4 h, then titrated between 5 and 20 mg/h to maintain > 100 mL/h urine output.

During this phase, an overzealous evacuation of fluids can lead to hypovolemia, hypotension and hypoperfusion of tissues and should be monitored and avoided. Recent studies show that patients treated with conservative initial and late fluid management had the best outcome, followed by those who received initial adequate and LCFM.[18]

MONITORING

Monitoring though is essential in the operating room and the Intensive Care Unit (ICU), will only be of value as long as it is accurate and reproducible, and no measurement has ever improved survival, only a good protocol can do this.

The traditional markers of the adequacy of fluid resuscitation, including haemodynamic parameters like heart rate, blood pressure, filling pressures and urine output, remain the essential indicators of successful resuscitation. American Stroke Association, American Heart Association and Neurocritical Care Society have given weak recommendations for monitoring in certain situations such as aneurysmal SAH to maintain CBF and oxygenation.

Level 1 recommendation includes the use of the initial base deficit, lactate level, or gastric intramucosal pH to stratify patients with regard to the need for ongoing fluid resuscitation and the risk of multiple organ dysfunctions and death.[19]

Monitoring may include invasive methods (e.g., transpulmonary thermodilution-guided) or less invasive methods (e.g., oesophageal Doppler). Further, fluid management based on fluid responsiveness,[20] other dynamic haemodynamic measures (e.g., PPV) or volumetric measures of preload (e.g., GEDVI) may be favoured over filling-pressure measures such as pulmonary artery occlusion pressure.[21]

The modern approach to fluid management is based on the concept of goal-directed therapy, in which it is believed that interventions should be performed specifically to affect a meaningful clinical variable.

The reality is that fluids can be harmful, and should only be given when they are expected to produce some

benefit. Management of fluids such that stroke volume is optimised is an extremely well validated approach that has been shown repeatedly to reduce morbidity.

INTRACRANIAL SURGERY

Normovolemia and normotension should be maintained during intracranial surgery. Hyperglycaemia, which worsens the consequences of cerebral ischaemia[22] and hypo-osmolality (target osmolality 290–320 osm/kg), which can increase cerebral oedema, should be avoided. Iso-osmolar fluids should be preferred for infusion intraoperatively such as 0.9% NaCl, newer 6% hydroxyethyl starch in a balanced solution (~310 osml/kg), with no deleterious effects on coagulation. The haematocrit should be maintained above 28%. Fluids should be warmed to ensure normothermia during as well as at emergence from anaesthesia.[10]

TRAUMATIC BRAIN INJURY

During fluid resuscitation in TBI, the goal is to prevent secondary injury to the brain by preventing hypotension and maintaining cerebral perfusion between 50 and 70 mmHg. Maintaining serum osmolality and preventing a fall in COP, and restoring circulating blood volume achieve this. However, although hypovolemia is harmful, fluid overload should be avoided. Serum sodium levels should be carefully monitored in patients with head injuries. Hyponatremia is associated with cerebral oedema and should be prevented.

SUBARACHNOID HAEMORRHAGE

The intravascular volume status has been found to be abnormally low in 36%–100% of patients with SAH and the level of hypovolemia correlates with the clinical grade. This hypovolemia is associated with hyponatremia in 30%–57% of cases[23] and is referred to as cerebral salt wasting syndrome.

Triple H therapy is an effective regime to prevent and treat ischemic neurologic deficits due to vasospasm. The rationale being in SAH, the ischemic areas of the brain have impaired cerebral autoregulation, and thus CBF depends on CPP, which in turn depends on intravascular volume and mean arterial blood pressure.[24] Hypervolemia is generally achieved with infusions of isotonic colloids as well as crystalloids. Hetastarch and dextran solutions should be used cautiously as excess can lead to coagulopathy through interference with platelets and factor VIII.[25]

SPINE SURGERY

Fluid management in spinal surgery involves a balance between maintaining intravascular volume to ensure adequate perfusion and oxygenation of the spinal cord as well as avoiding venous congestion that may occur with fluid overload. Surgery involving extensive exposure of the spine with denuding of bone, extensive instrumentation and repair may be associated with significant blood loss requiring large amounts of asanguineous fluid as well as blood transfusions, with prolonged stays in the neurointensive care.[26]

CONCLUSION

The ROSE concept advocates restriction of fluids, which is consistent with the prevention of a 'tight brain' in neurosurgery; however, it is conflicting with the aim of normovolemia and maintenance adequate cerebral perfusion and oxygenation. Methods used to minimise intracranial interstitial water, however, may induce hypovolemia and electrolyte abnormalities and adversely affect cerebral as well as spinal cord perfusion. ROSE may be relevant in neurotrauma patients for resuscitation especially following polytrauma. Recent data indicate that positive fluid overloaded is detrimental to neurosurgical and neurocritical patients as well as other critical patients, and therefore the evacuation and de-resuscitation concept may be considered.

It is important to note that the current literature on fluid management in brain-injured patients has had a main focus on SAH, which is probably related to the well-known risk of hypovolemia associated with cerebral salt wasting syndrome after SAH, whereas studies on fluid management in TBI, ICH and ischaemic stroke are much less numerous. Whether this imbalance in fluid management studies between different types of brain injuries is a reflection of differences in clinical relevance of fluid management is not clear.[1]

Further randomised controlled trials in the future are essential in SAH and TBI patients who are critical and need long term ICU stay to elucidate and define the role and relevance of the ROSE concept in neuroanaesthesia and neurocritical care.

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