Chapter 4
Management of Fluids, Electrolytes, and Blood Products in Neurosurgical Patients

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Overview

- The major fluid compartments of the body are the intracellular compartment and the extracellular compartment, which is subdivided into intravascular and interstitial spaces.
- The volume of the individual compartments may change in a disease state or as the body adapts to environmental stress.
- In peripheral tissues, the primary determinant of fluid movement across capillaries (i.e., between the intravascular and interstitial spaces) is the oncotic gradient produced by large plasma proteins such as albumin.
- Unlike the peripheral tissues, the brain and spinal cord are isolated from the intravascular compartment by the blood–brain barrier.
- The primary determinant of water movement across the intact blood–brain barrier is the osmotic pressure gradient produced by osmotically active particles including plasma sodium and other electrolytes.
- Intravenous infusion of solutions hyperosmolar to plasma (e.g., 3% sodium chloride, mannitol) will lead to a decrease in brain water content and intracranial pressure (ICP). Administration of excess free water (e.g., hypoosmolar or dextrose-containing electrolyte-free solutions) will lead to increased brain water content and ICP.
- Osmotically active particles as well as plasma proteins may "leak" into the cerebral tissue where the blood–brain barrier has been disrupted and thus contribute to worsening cerebral edema in such regions.

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- Intravenous administration of hyperosmolar solutions results in a decrease in water content in brain where blood brain barrier is intact to make room for the injured brain.

Implications for the Neurosurgical Patient

Perioperative fluid management in neurosurgical patients poses special challenges.

- The presence and treatment of elevated ICP, surgical bleeding, and a variety of pathophysiological derangements associated with neurologic injury may lead to significant hypovolemia, electrolyte abnormalities, anemia, and coagulopathy.

Care must be taken to:

- Maintain hemodynamic stability, optimal cerebral perfusion pressure, and oxygen delivery to the CNS tissue and
- Minimize the impact of fluid resuscitation on the development or exacerbation of cerebral edema.

The goals of fluid resuscitation are (see Tables 4.1–4.5):

- Restore intravascular volume and cerebral perfusion pressure and
- Achieve a slightly hyperosmolar state.

The clinician can choose from a variety of intravenous fluids including crystalloid, hypertonic saline, colloid, and blood products as dictated by the clinical scenario. The typical initial fluid choice for an elective craniotomy is a combination of Lactated Ringer's and 0.9% saline (Table 4.6).

Table 4.1 Commonly used IV solutions

Commonly used intravenous solutions	Osmolarity (mOsm/L)
Plasma osmolarity	270–295
Crystalloid	
Lactated Ringer's	273
0.9% Normal saline	308
D5 Lactated Ringer's	525
20% Mannitol	1,098
3% Hypertonic saline (HS)	1,026
Colloids	
6% Hetastarch	310
Pentastarch	326
6% Dextran (70)	300
5% Albumin	300
25% Albumin	1,500
HS colloid mixture	
7.5% HS 6% Dextran[a]	2,568

[a]Available in Europe

Table 4.2 Common causes of hyponatremia

Dilution
Excess water intake
Administration of hypoosmolar fluids
Use of diuretics
Mannitol, thiazides
Adrenal insufficiency
Hypothyroidism
Hyperglycemia
Cerebral salt wasting syndrome
Common in subarachnoid hemorrhage
Associated with hypovolemia
SIADH
CNS disorders
Chronic infections
Medications, e.g., carbamazepine
Organ failure
Cirrhosis, congestive heart failure, nephrotic syndrome
Associated with hypervolemia

SIADH syndrome of inappropriate antidiuretic hormone secretion

Table 4.3 Common causes of hypernatremia

Dehydration
Diabetes insipidus
Use of hypertonic saline

Table 4.4 Common causes of hypokalemia

Combined use of osmotic and loop diuretics
Hypomagnesemia
Intracellular potassium shift secondary to
Hyperventilation
Insulin infusion

Table 4.5 Considerations for assessing intravascular volume

History
Preoperative fasting and insensible losses
Presence of hemorrhage
Use of diuretics
Use of hyperosmotic intravenous contrast
Physical exam
Vital signs: presence of fever, tachycardia, hypotension
Orthostatic tachycardia and hypotension
Status of neck veins, skin turgor, mucous membranes
Oliguria
Pulmonary edema
Monitors
Trend in CVP or PAOP
Marked reduction in arterial pulse pressure or stroke volume with positive pressure ventilation signifying intravascular depletion

CVP central venous pressure, *PAOP* pulmonary artery occlusion pressure

Table 4.6 Indications for commonly used intravenous fluids and blood products

Indication	Fluid or blood product	Amount
Fluid maintenance Insensible and interstitial losses	Lactated Ringer's 0.9% Saline (normal saline, NS)	1:1 Crystalloid/fluid loss ratio; (usual rate: NS at 1.5 mL/kg/h)
Brain relaxation for exposure during craniotomy	20% Mannitol 3% Sodium chloride (hypertonic saline, HS)	0.25–2 g/kg 5 mL/kg
Treatment of elevated ICP	20% Mannitol 3% Saline (HS)	0.25–2 g/kg 200 mL
Replacement of blood loss	Lactated Ringer's, NS	3:1 Crystalloid/blood loss ratio
	Colloid	1:1 Colloid/blood loss ratio
	Hetastarch 6%	If used, limit to 20 mL/kg/24 h
	Red cells – ideally washed, leukoreduced, <15 days old	1 Unit should raise Hgb by 1 g/dL or Hct by 3%
Disseminated intravascular coagulation (DIC)		
Elevated INR, PTT	Fresh Frozen Plasma	Start at 10–15 mL/kg
Fibrinogen < 100 mg/dL	Cryoprecipitate	1 Pool (6 bags) raises fibrinogen by 45 mg/dL
Thrombocytopenia < 100,000 in a bleeding patient	Platelets pheresed	1 Bag (4 pooled units) raise platelets by 30,000/μL

Concerns and Risks (Table 4.7)

Anemia

- Has been associated with worse neurologic outcome in cardiopulmonary bypass surgery and with perioperative visual loss in prone spine surgery.
- The ideal hematocrit for optimizing cerebral blood flow and oxygen delivery in focal ischemia model is currently believed to be 30–34%. Higher hematocrit results in increased blood viscosity; hematocrit ≤ 25% results in decreased oxygen-carrying capacity.
- Normovolemic hemoglobin levels of 7–9 g/dL appear to be safe for the general ICU patient population.
- There is insufficient evidence to allow recommendations regarding:
  - The "safe" level of anemia for patients with neurologic injury; or
  - Whether correction of anemia by transfusing red cells has beneficial or detrimental effects on neurologic outcome.

Table 4.7 Concerns and risks of fluid management in neurosurgical patients

Under-resuscitation	Hypotension, inadequate cerebral perfusion pressure, secondary brain injury
Over-resuscitation	Exacerbation of cerebral edema
Hyponatremia	<120–125 mEq/L – change in mental status, seizures
Hypernatremia	>160–170 mEq/L – change in mental status, seizures
Lactated Ringer's	Hypoosmolar state, hyponatremia
0.9% Saline	Hyperchloremic metabolic acidosis
Dextrose solutions	Hypoosmolar state, hyperglycemia exacerbating cerebral injury
20% Mannitol	Hyponatremia Loss of bicarbonate – metabolic acidosis Excessive diuresis – intravascular volume depletion, electrolyte losses Rebound cerebral edema Hyperkalemia with high doses (2 g/kg)
Hypertonic saline	Hypernatremia Hyperchloremic metabolic acidosis Rebound cerebral edema when plasma sodium falls Tearing of cerebral veins – intracerebral hemorrhage Excessive diuresis – intravascular volume depletion, renal failure Central pontine myelinolysis – with rapid rise of plasma sodium from hyponatremic levels; malnourished and alcoholic patients at increased risk Sclerosis of veins Interference with coagulation and platelet aggregation
Synthetic colloids	Interference with coagulation, factor VIII complex; potential increased risk for intracranial hemorrhage No clear benefit compared with crystalloid as a resuscitative fluid Renal impairment Allergic reactions Pruritus Interference with blood cross-matching with dextran
Albumin	Expensive No clear benefit compared with crystalloid as a resuscitative fluid; potential harm in patients with traumatic brain injury

Transfusion of Blood Products

Current concerns regarding blood product transfusion in the developed world focus more on the immunomodulating effects of transfusion rather than transmission of infectious agents (Table 4.8). Transfusion-related acute lung injury (TRALI) is thought to be the leading cause of transfusion-related mortality.

Table 4.8 Examples of risks associated with transfusion of blood products

Risk	Risk per unit transfused
Infectious risks	
HIV	1:1.5–4.7 million
Hepatitis C	1: 1.9–3.1 million
Hepatitis B	1: 31,000–205,000
Hemolytic reactions	
Acute	1:13,000
Delayed	1:1,600
Alloimmunization	1:1,600
Immunosuppression	1:1
TRALI	1:5,000

Adapted in part from reference by Marik and Corwin 2008

Table 4.9 Special circumstances

Traumatic brain injury	Subarachnoid hemorrhage
Risk of coagulopathy and DIC	Electrolyte abnormalities
Multitrauma	Hypocalcemia, hypomagnesemia,
massive hemorrhage	Hypokalemia
dilutional coagulopathy	Hyponatremia
Neurogenic pulmonary edema	Cerebral Salt Wasting Syndrome
Hyponatremia	SIADH
Cerebral salt wasting syndrome	Avoid hypovolemia
SIADH	Vasospasm – therapeutic goal: hypervolemia

Special Circumstances (Table 4.9)

Key Points

- Fluid management goal is a euvolemic, slightly hyperosmolar state.
- Avoid hypoosmolar fluids and dextrose-containing solutions unless needed to treat hypoglycemia.
- Consider using hypertonic saline, unless contraindicated, to treat elevated ICP in a hypovolemic, hemodynamically unstable patient.
- In anemic patients with neurologic injury, there is insufficient evidence regarding transfusion thresholds. Do not use an arbitrary hemoglobin number; weigh risks of transfusion (e.g.,TRALI, immunosuppression) with benefits of oxygen delivery to injured CNS tissue.

Suggested Reading

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