10 Fluid and Electrolytes Management in Children Undergoing Neurosurgery

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Key Points

- The fluid management plan for children undergoing neurosurgical procedures should consider preoperative assessment, correction of existing deficit, calculation of intraoperative requirements and management thereof, postoperative management of the imbalances, and fluid therapy monitoring.
- Among children, a fall in blood pressure is usually a late sign of hypovolemia, and it should be rapidly corrected to maintain cardiac output and cerebral perfusion. Slower correction of dehydration, without the signs of hypovolemia, is acceptable.
- Avoid, if possible, colloids and glucose infusion. In the case of hypernatremia as a consequence of pituitary surgery (such as diabetes insipidus), infusion of 5% glucose solution may be considered.
- Electrolytes dysregulation, such as hyponatremia or hypernatremia, and K^+ alterations are associated with worsening of the outcome.
- Attention should be paid to postoperative complications, particularly after surgery for sellar lesions (diabetes insipidus, syndrome of inappropriate antidiuretic hormone secretion, cerebral salt wasting syndrome).

10.1 Introduction

Advances in neurosurgical techniques have changed the face of pediatric neurosurgical management, leading to generally improved outcomes. However, with the increasing demand, complexity, and improvement of care for children undergoing neurosurgical procedures, there is an increased need to develop guidelines to improve the level of care and assure uniform patient management. In particular, fluid and electrolytes management in the pediatric neurosurgical population requires careful attention to the intravenous (IV) fluid administration and close monitoring of fluid balance and assessment of the clinical status of the patient to prevent and correct perioperative complications. Neurosurgical patients are usually complex and often need a large number of fluids and hemo-components as well as postoperative monitoring in an intensive care setting. Moreover, several pathophysiological processes occur in this group of patients that make them vulnerable to peculiar electrolyte and fluid disturbances, including syndrome of inappropriate antidiuretic hormone (SIADH) secretion, cerebral salt wasting syndrome (CSWS), and cranial diabetes insipidus (DI). This chapter

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will review the main principles for fluid and electrolytes management in the pediatric population undergoing neurosurgical procedures.

10.2 General Considerations

Management of infants and children posted for neurosurgical procedures requires careful considerations of the unique challenges they present with. There are several important differences between the procedures performed in pediatric neurosurgical patients and those performed in the adult population, which are usually due to the anatomo-physiologic peculiarities of children. The types and characteristics of the central nervous system (CNS) tumors seen in the pediatric population are different from those in adults. Also, the management of some conditions (such as hydrocephalus) is much more challenging from both clinical and surgical perspectives, particularly when its onset is in infancy or childhood [1].

Because of the particular anatomical characteristics of the pediatric population, including a more compliant (viscoelastic) and not fully ossified skull, injuries to the brain mass (including tumors, hemorrhages, etc.) tend to progress indolently when compared with adult lesions. Additionally, considerations like the development of the spinal cord and associations with congenital disorders in pediatric patients also impact the decision-making process perioperatively compared to the adults.

Several anatomical, physiological, and anesthetic implications have to be taken into account (Table 10.1). A higher percentage of total body water in the pediatric population than adults makes them vulnerable to develop serious morbidity caused by fluid and electrolytic imbalance. They need a large volume of distribution for water-soluble medications, and low fat and muscle content provides a small reservoir for drugs that depend on redistribution into these tissues for their metabolism and the execution of their effect. Therefore, water-soluble drugs require larger doses for clinical effect (such as antibiotics and muscle relaxants), while drugs like thiopental (redistribution into fat) and fentanyl (redistribution into the muscle) have a longer clinical effect.

Table 10.1 Peculiar characteristics of neonatal physiology regarding fluid and electrolytes

Neonatal physiologic variables	Characteristics regarding fluid and electrolytes
Body water percentage and distribution	• Term neonates: 75% (40% ECF, 35% ICF) • Preterm neonates (23 weeks): 90% water (60% ECF, 30% ICF) • Adults: 60% water (20% ECF, 40% ICF)
Fluid losses	• Skin (70%) and respiratory tract (30%) are the major sources of insensible water losses (IWL) in neonates • Preterm infants have a generally higher IWL
Cardiovascular physiology	• Reduced cardiac contractility (low contractile mass/gram of cardiac tissue) • Reduced ability to ↑ stroke volume • Dependence on heart rate (↑ HR) to ↑ cardiac output (Treppe effect) • High sensitivity to hypovolemia and anesthetic agents
Renal function	• 25% functionality at birth with maturation by 2 years of age • $T_{0.5}$ of drugs excreted by glomerular filtration is prolonged • Normal urine osmolality: 50–600 mEq/L in preterm and (50–?) 800 mEq/L in term infants

ECF extracellular fluid, *ICF* intracellular fluid

Moreover, renal and cardiovascular physiology and fluid losses present some differences in the pediatric population compared with adults (Table 10.1), making them more vulnerable to fluid dysregulation. In children, the fall in blood pressure is usually a late sign of hypovolemia, and hence, it is to be corrected rapidly to maintain effective cardiac output and cerebral perfusion.

In addition to all of the above considerations, in-depth knowledge of anticipated major possible adverse events is also essential in managing some

of the unique surgical challenges. Fluid and electrolyte imbalance can also increase morbidity and mortality, and its prevention and prompt treatment should be a priority concern in pediatric neurosurgery.

10.3 Choice of Fluid During Pediatric Neurosurgery

Planning for fluid management in neurosurgical cases is extremely important, and communication between the surgeon and the anesthesiologist in this regard must be clear. In general, the target is to ensure euvolemia, avoiding both hypovolemia and hypervolemia and consequently hypoperfusion of the brain and other organs, and attenuate edema. Crystalloids should be the first choice, and in particular, 0.9% normal saline is commonly used because of its slight hyperosmolality (308 mOsm/L), which can help in attenuating the occurrence of brain edema (Table 10.2) [2]. Although there is no absolute contraindication, caution must be exercised with the use of colloids (hydroxyethyl starch). A recently published meta-analysis concluded that the established adverse effect profile of colloids such as renal injury and coagulopathy were not observed with their perioperative administration in noncardiac surgeries [3].

In patients with intracranial hypertension, osmotherapy using mannitol or hypertonic saline (3% or higher concentration) should be considered in order to reduce intracranial pressure (ICP). However, if possible, it is important to avoid using hypertonic solutions if a central line is not available so to avoid the risk of phlebitis. Attention should also be paid to the administration of diuretics (such as furosemide), which can be used to induce systemic diuresis, improve overall cerebral water transport, and decrease cerebrospinal fluid (CSF), but they can also determine hypovolemia and dehydration and reduced cerebral perfusion pressure (CPP).

Hypotonic saline solutions (including 0.45% NaCl, 0.45% NaCl +5% dextrose, 0.18% NaCl +10% dextrose, 0.18% NaCl +4% dextrose, and 10% dextrose solutions) should be used with extreme caution in neurosurgical patients, as they can worsen cerebral edema, and should only be used to treat acute hypernatremia (Na >150 mEq/L) [4]. In particular, small children and neonates are more susceptible to hypoglycemia than adults, which may occur after preoperative fasting. Thus, when initiating fluid management in neonates, the use of 0.9% NaCl and dextrose should be considered. Complications of hyperglycemia in neonates and preterm infants include dehydration due to diuresis and electrolyte disturbances and an increased risk of hypoxic-ischemic central nervous system (CNS) damage and have to be strictly avoided [5]. The current anesthetic practice involves dextrose solutions at a lower concentration (1–2%) instead of the traditional use of 5% solutions [6]. In children older than 5 years, in those who have a low risk of fasting-induced hypoglycemia, or in patients with documented hyperglycemia, 0.9% NaCl without dextrose is usually the standard of care [7]. Although a hyperglycemic response during surgery and anesthesia is anticipated (increased sympathetic system activity and gluconeogenesis), such a response may not occur in all patients and makes children especially prone to hypoglycemia. The incidence of intraoperative hypoglycemia has been estimated at 0–2.5% and

Table 10.2 Commonly used intravenous fluids

Types	Na^+	K^+	Cl^-	Ca^{2+}	Mg^{2+}	Acetate lactate	Glucose	Phosphate	Osmolality
NS	154		154						308
RL	1	4	109	3		9			274
Isolyte P	26	21	21		3	24	5	3	
Plasmalyte	140	5	98		3	27			295
Albumin 5%	150	<2.5	100						330
Hetastarch	154		154						310

NS normal saline, *RL* Ringer's lactate

is generally seen with longer fasting periods (8–19 h). Hence, it becomes essential to monitor blood glucose levels intermittently when fasting is prolonged and glucose is not being supplemented intraoperatively [8].

Fluid management for neurosurgical procedures can be divided into several phases [9]:

- Fluid considerations before surgery.
- Preoperative assessment for fluid deficits and methods for correction.
- Estimating the requirements for maintenance fluids.
- Management of other losses (blood and third space loss) during surgery.
- Postoperative fluid management and modalities to monitor fluid therapy.

10.3.1 Fluid Considerations Before Surgery

In the preoperative phase, fluids are best administered through the oral/enteral route. The literature is not clear about which is the safest preoperative fasting practice in infants. Some authors consider it safe to anesthetize a child 3 h after the last breastfeeding; some suggest this concept is valid only in infants under 6 months of age, and others would allow surgery 4 h after the last breastfeeding [10]. It is generally accepted that children aged 6 months or older should safely be allowed to have clear fluids 2 h before surgery and that withholding of solid food should be done 8 h before surgery. This is essential to prevent dehydration in the child and, at the same time, reduce the risk of hypoglycemia and aspiration. Under 6 months of age, breast milk is allowed up to 4 h before surgery [11].

10.3.2 Assessment and Correction of Fluid Deficit

The preoperative assessment of the pediatric population undergoing neurosurgical procedures should take into account several factors. An accurate collection of information about the patient's past medical history is necessary, focusing on congenital or acquired diseases that can increase the risk for dehydration or electrolyte imbalance (heart or kidney diseases, burns, etc.). Physical examination should include the weighting of the child (one of the most important criteria to assess the fluid balance in children) and the characteristics of the skin and mucosa (including dry mucosa, edema, and altered skin turgor). Clinical signs due to dehydration are tardive, in particular the hypotension. Therefore, trying to calculate the water deficit precisely using clinical signs may result in inaccuracies. In mild dehydration, the only clinical signs/symptoms may generally be just an increased thirst with dry mucosal membranes; in moderate dehydration, tachypnea, cool and pale peripheries with prolongation of capillary refill times, sunken eyes, reduced skin turgor, and low urine output can be useful additional clinical signs/symptoms. Finally, in severe dehydration, in addition to the signs of moderate dehydration, the child may be irritable and/or lethargic and have severe hypotension with deep acidotic breathing, which is late premorbid signs (Table 10.3) [12–14].

Laboratory tests should be obtained alongside the accurate clinical assessment, including serum electrolytes and plasma osmolality, blood urea, serum creatinine, urine electrolytes, and specific gravity, and, if necessary, arterial blood gases (ABGs).

Patients undergoing minor and elective surgery usually have only a minor fluid deficit, which is usually not necessary to correct. For major surgery, an initial bolus of 10 ml/kg of isotonic crystalloid (0.9% normal saline) or Ringer lactate or Hartmann's solution should be given in the first hour to correct the fluid deficit. The fluid normally used to replace this deficit should be isotonic, in particular, 0.9% sodium chloride. Should hypovolemia be present, a bolus of 10–20 ml/kg of an isotonic fluid must be administered and repeated as per Advanced Pediatric Life Support (APLS) guidelines [15].

Table 10.3 Signs and symptoms of dehydration

Grade of dehydration	Mild (<3% water loss)	Moderate (3–10% water loss)	Severe (>10% water loss)
General	Alert	Thirsty, lethargic	Cold, sweaty, limp
Pulse	Normal rate and volume	Rapid and weak	Rapid, feeble
Systolic pressure	Normal	Normal	Low
Respiratory rate	Normal	Increased, deep	Deep
Dry mouth	No	Yes	Yes
Eyes	Normal	Sunken	Sunken
Anterior Fontanelle	Normal	Sunken	Very sunken
Skin turgor	Normal (recoils instantly)	Reduced (1–2 s)	Severely reduced (>2 s)
Capillary refill	Not prolonged	Slightly prolonged	Prolonged
Neurological deterioration	No	Drowsiness	Severe
Urine output	Normal	Reduced	Reduced
Deficit	30–50 ml/kg	60–100 ml/kg	>100 ml/kg

Table 10.4 Holliday and Segar recommendations for fluid management (4-2-1 rule)

Body weight (kg)	Daily fluid requirement
0–10	4 ml/kg/h
10–20	40 ml/h + **2** ml/kg/h above 10 kg
>20	60 ml/h + **1** ml/kg/h above 20 kg

Table 10.5 Fluid requirements for neonates

Newborn term	Daily fluid requirement (ml/kg)
Day 1	50–60
Day 2	80
>Day 7	100–150

10.3.3 Maintenance Fluid Requirements

Fluid requirements due to fasting should be replaced according to the recommendations of Holliday and Segar for children and infants over 4 weeks of age, using the child's body weight (Table 10.4) [16]. According to Furman et al., the total amount of hourly maintenance requirements multiplied by hours of fluid restriction should be administered by giving 50% in the first hour and 25% during each of the next 2 h [17]. This management strategy was further modified by Berry et al., as they suggested the administration of a salty solution bolus during the first hour of the surgery (25 ml/kg for children 3 years old and younger, 15 ml/kg for older than 4 years) [18].

However, this formula should be just considered as a starting point only. Every individual child should always be monitored for response to fluid therapy, and adjustments should be made accordingly; in particular, clinical parameters to be assessed include heart rate, blood pressure, and capillary refill time.

In term neonates (>36-week gestational age), maintenance fluid requirements are reduced in the first few days after birth (Table 10.5). The normal infant will lose up to 10–15% of its body weight in water during this time. For the older children, the 4–2-1 rule (Holliday and Segar method) is followed. Certain conditions like burns, radiant heaters or phototherapy pyrexia, or excessive sweating result in hypermetabolic states resulting in increased requirements in maintenance fluids [19, 20].

During neurosurgical procedures, most children may be given fluids without dextrose. The maintenance fluid most commonly used during neurosurgery is an isotonic crystalloid, 0.9% sodium chloride. However, blood glucose and electrolytes should always be monitored, especially in long procedures, major surgery, or pituitary surgery. Perioperative dextrose (1–2.5% dextrose in Ringer's lactate solution) was shown to increase blood glucose in pediatric patients during surgery, which returned to normal levels about 1 h after surgery. The use of 2.5% solution was shown to yield a much greater increase in blood glucose, compared with 1% solution [21].

10.3.4 Management of Other Losses During Surgery

Generally, replacement of all losses during surgery should be done with 0.9% sodium chloride, or eventually Ringer lactate/Hartmann's solution, while colloids should be used only when deemed necessary. Intraoperative losses include third space loss and blood loss. During surgery, third space loss occurs due to extravasation of fluids from the intravascular compartments out to the tissues around the surgery site, and they should be replaced. It is generally accepted that superficial surgeries, including ophthalmic and neurosurgeries, result in the least amount of third space loss estimated at 1–2 ml/kg/h [22].

Even in this phase, it is important to assess clinical signs like heart rate, blood pressure, and capillary refill time to ensure adequate replacement. In case of bleeding, blood products transfusion should be considered. The determinants for blood transfusion are based on clinical signs, estimated blood volume (Table 10.6), preoperative hemoglobin and hematocrit values, and coexisting illnesses.

According to a study, a hemoglobin threshold of 7 gm/dL for red blood cell transfusion can decrease transfusion requirements without increasing adverse outcomes in critically ill children [23]. A fall in hematocrit of up to 25% from baseline values may be acceptable in children aged more than 3 months; however, in children with cyanotic congenital heart disease or severe respiratory diseases, a higher hematocrit target should be used to maintain appropriate tissue oxygenation. In infants younger than 3 months of age, it is not clear which is the hemoglobin and hematocrit threshold to consider for transfusion, but blood administration should be individually considered, according to the clinical conditions and the gestational age. However, mildly low hemoglobin and hematocrit values could be acceptable in small, older preterm infants [24, 25].

Table 10.6 The estimated blood volume in neonates and infants

Age	Estimated blood volume (ml/kg)
Premature neonates	95–100
Full-term neonates	85–90
Infants	80

10.3.5 Postoperative Fluid Management and Monitoring of Fluid Therapy

Regarding postoperative fluid management (Table 10.7), it should be considered that surgery, pain, nausea, and vomiting are all potential causes of dehydration and ADH release, as well as other peculiar complications related to neurosurgical procedures, including DI or other electrolyte disturbances. The literature is not clear about which is the correct maintenance fluid rate during the postoperative period. Holliday and Segar's formula may be followed; isotonic fluids may be used to replace ongoing losses from drains or nasogastric tubes, with or without added electrolytes. Serum electrolytes, as well as hemoglobin and hematocrit, should be measured pre- and postoperatively. Moreover, children should be weighed prior and after the prescription and administration of fluids [21]. During the postoperative period, serum electrolytes should be monitored every 24 h in all children on IV fluids (or more frequently if abnormal). Particular attention is paid to patients who underwent pituitary surgery and who are vulnerable to electrolytic complications. Fluid monitoring should include a fluid input/output chart, urine output monitored hourly and replaced every 2–4 h. Apart from routine clinical parameters, advanced parameters

Table 10.7 Goals of postoperative fluid therapy

Parameter	Target
Urine output	1–3 ml/kg/h
Allowable weight loss	1–2% per day in first week
Urine specific gravity	1005–1015
Euglycemia	75–100 mg/dl
Normonatremia	135–145 mEq/L
Normokalemia	4–5 mEq/L
Prevention of failure	Absence of edema/ dehydration/hepatomegaly

like dynamic indices of fluid responsiveness like stroke volume variation (SVV), pulse pressure variation (PPV), and plethysmograph variability index (PVI) may be employed whenever available. Although they have been validated in adults, the data available for the pediatric population is limited due to the anatomical and physiological variations in cardiorespiratory parameters that make their efficacy limited [26, 27].

10.4 General Principles for Electrolytes Management

Neurosurgical patients receiving IV fluids should have their electrolytes checked daily by capillary sampling or venous blood gas analysis. Hyponatremia is the most common electrolytic disorder in this group of patients, and if SIADH or CSWS occurs, sodium may fall very rapidly, and the treatment becomes a clinical emergency. In general, any neurosurgical patient with a drop in sodium of more than 4 mEq/L since the last measurement or a Na^+ <131 mEq/L should have an urgent clinical assessment. Repeat electrolytes and ABG analysis are to be done to confirm hyponatremia, and appropriate treatment is to be instituted. Any patient with Na^+ <135 mEq/L on routine blood tests should be reviewed urgently, including the assessment of fluid balance, IV or enteral fluids, and replacement of Na^+ losses [28]. Finally, patients with extraventricular drainage (EVD) in situ should have their electrolytes monitored twice a week.

10.5 Sodium Disturbances

10.5.1 Hyponatremia

Hyponatremia may occur in up to 20% of patients in the postoperative period and reach up to 50% of neurosurgical patients. The early signs of hyponatremia are nonspecific, and often the first presenting symptoms are neurological deterioration, seizures, or respiratory arrest. Headache is considered an early and common sign of hyponatremia, but this sign might not be obvious in young children. Initial symptoms may also include nausea or vomiting; and progress to confusion, seizures, stupor, and coma as hyponatremia worsens. Finally, severe hyponatremia may result in brain swelling (cerebral edema) and symptoms related to increased ICP. The magnitude of the symptoms is related to both the severity and the rate at which serum sodium levels drop [29]. Hyponatremia can increase mortality, which is significantly higher and directly related to the severity of electrolyte disturbance. Therefore, aggressive treatment as a medical emergency and pediatric intensive care admission is mandatory precaution [30].

There are several conditions which can lead to hyponatremia in the pediatric population (Table 10.8); however, in neurosurgical patients, the main causes of hyponatremia are the SIADH, resulting from excessive water retention after ADH secretion dysfunction, or the salt-losing brain syndrome, characterized by hyponatremia, polyuria, and severe dehydration [31]. Pituitary tumors are at higher risk for developing both these conditions, whereas in tumors not involving the hypothalamo-pituitary axis, SIADH occurs commonly. As the management of both these conditions is vastly different, accurate diagnosis and clinical and laboratory criteria are most essential (Table 10.9). Physiologically, ADH is secreted in cases of high plasma osmolality and decreased effective blood volume, which is detected by the osmoreceptors and baroreceptors in the aortic arch, carotid sinus, left atrium, and hypothalamus. ADH acts on the distal convoluted tubule and collecting ducts in the kidney in order to reabsorb water without reabsorbing solute.

Inappropriate ADH secretion occurs when this process happens in response to non-osmotic stimuli (Table 10.10). The main risk factors are stress, cerebral injury, mechanical ventilation, pain, and several drugs, including anesthetic agents and antiepileptic drugs. Patients affected by SIADH present with euvolemia or hypovolemia with hyponatremia, and therefore the clinical management should be based on Na^+ replacement and fluid restriction.

In the salt wasting syndrome, there is an excessive release of the natriuretic peptide, which

Table 10.8 Causes of hyponatremia in the pediatric population

Reduced total body sodium	Clinical presentations
Extrarenal	Vomiting, diarrhea Fluid sequestration (sepsis, peritonitis, pancreatitis) Cutaneous losses (burns) Ventriculostomy drainage
Renal	Cerebral salt wasting syndrome Diuretics (osmotic/non-osmotic) Tubulointerstitial diseases Adrenal insufficiency Congenital adrenal hyperplasia, Addison's disease Renal diseases (obstructive uropathy, nephritis, pyelonephritis, renal tubular acidosis)
Increased total body sodium	Congestive heart failure Cirrhosis Nephrotic syndrome Renal failure
Normal total body sodium	SIADH Glucocorticoid deficiency Hypothyroidism Infantile water intoxication Abusive water intoxication

Table 10.9 Differential diagnosis between SIADH and CSWS

Parameters	SIADH	CSWS
Extracellular fluid volume	Normal to high	Low
Fluid balance	Positive or neutral	Negative
Urine volume	Decreased or normal	Increased or normal
Central venous pressure	Normal to high	Low
Urine Na^+	High (20–40 mEq/L)	High (>40 mEq/L)

SIADH syndrome of inappropriate antidiuretic hormone, *CSWS* cerebral salt wasting syndrome

Table 10.10 The causes of syndrome of inappropriate antidiuretic hormone (SIADH) secretion

Central nervous system	Meningitis, encephalitis Multiple sclerosis, neuropathy Brain trauma, tumor, and abscess Hypoxia Hydrocephalus Drugs (vincristine, salicylates) Cerebral thrombosis of hemorrhage Subarachnoid hemorrhage or subdural hemorrhage
Pulmonary system	Pneumoniae Asthma Pneumothorax Positive pressure
Drugs	ADH analogs Barbiturates Haloperidol, tricyclic, indomethacin, interferon, ecstasy
Miscellaneous	Tumors Postoperative and postprocedural patients

leads to primary natriuresis and volume depletion, and therefore patients present with hypovolemia and hyponatremia. Thus, given the severe dehydration risk, an aggressive volume replacement with an isotonic solution and increased sodium supply is necessary.

In general, asymptomatic hyponatremia should be treated with enteral fluids as tolerated or with intravenous 0.9% sodium chloride solution and, eventually, fluid intake restriction if the child presents normal or increased volume. Patients with hyponatremic encephalopathy (seizure, coma) should be aggressively treated with an infusion of hypertonic 3% sodium chloride solution. 1 ml/kg of 3% sodium chloride will normally raise the serum sodium by 1 mEq/L, and attention should be paid to the rate of Na^+ increase, which should not be higher than 1–2 mEq/L/h to avoid complications such as pontine myelinolysis. Hypertonic saline should be administered via a central vein, but it is important

not to delay sodium administration for the insertion of the central venous line.

The amount of Na^+ required can be calculated as:

$$\text{mmol of } Na^+ \text{ required} = \left(130 - \text{present serum } Na^+\right) \times 0.6 \times \text{Weight}\left(\text{kg}\right)$$

Sodium should be raised aggressively until serum Na^+ reaches 125–130 mEq/L, or after clinical improvement. A slower Na^+ correction should take place at this stage, and a 0.9% sodium chloride solution should be used. The addition of dextrose to this solution is still controversial, as generating hyperglycemia might worsen brain injury, and thus, should be considered on a case to case basis. During Na^+ replacement, patients should be monitored for signs of increased ICP, and electrolytes should be rechecked every 4 h until Na^+ is >130 mEq/L and, then, at least twice a day for the following 48 h. Finally, particular attention should be paid to patients with EVDs in situ, who might be more prone to hyponatremia due to sodium loss in CSF. The Na^+ levels in CSF are similar to those of plasma, and when the EVD drainage rate is >10 ml/h, Na^+ loss should be monitored and accurately replaced [32–34].

10.5.2 Hypernatremia

In hospitalized children, hypernatremia (serum Na^+ >150 mEq/L) commonly occurs due to excessive water loss, restricted intake, or an inability to respond to thirst. It is, therefore, generally related to a systemic dehydrated status (Table 10.11). The magnitude of the hypernatremia signs is more severe when it develops rapidly or when serum Na^+ >160 mEq/L, while chronic hypernatremia is often well tolerated. The severity of dehydration might be underestimated if clinical signs alone are used, compared to weight loss.

Improvement of hydration and establishing euvolemia should be targeted with 0.9% sodium chloride, given in boluses of 20 ml/kg. Once the initial fluid replacement is done, complete correction of hypernatremia should be done very slowly over at least 48 h to prevent cerebral edema, seizures, and brain injury. The correction rate should be no more than 12 mmol/kg/day and done with 0.45% or 0.9% sodium chloride with dextrose [35].

10.5.3 Diabetes Insipidus (DI)

Neurosurgical patients, especially with suprasellar tumors, are at high risk for many postoperative complications, including DI (Table 10.12). The deficiency of ADH secretion

Table 10.11 Causes of hypernatremia in the pediatric population

Causes of hypernatremia	Clinical presentation
Low total body sodium	
Extrarenal losses Renal losses Inadequate intake	Vomit, diarrhea, profuse sweating Osmotic diuresis (mannitol, glucose, urea) Insufficient lactation
Increased total body sodium	
Increased Na intake	Excessive administration of Na^+, near-drowning (seawater)
Normal total body sodium	
Extrarenal losses Renal	Respiratory insensible losses, dermal insensible losses (fever, burns, radiant warmers, phototherapy) Diabetes insipidus

Table 10.12 Causes of diabetes insipidus

Central	Clinical presentations
Congenital Acquired	Inherited, idiopathic Cerebral trauma, sellar/suprasellar tumors Infections (meningitis, encephalitis) Post-neurosurgical procedures Vascular, aneurysms, thrombosis, etc.
Nephrogenic	
Congenital Acquired	Inherited, mutation Renal failure, tubular disease Hypercalcemia, K^+ depletion Drugs (alcohol, lithium, diuretics, amphotericin B, etc.) Dietary abnormalities (primary polydipsia, decreased sodium intake)

results in intravascular solvent loss, polyuria, and, dehydration, with consequent hypernatremia, which can occur within the first postoperative hours. In patients who underwent pituitary surgery, DI should always be suspected, and if serum Na^+ level increases over 150 mEq/L, with urinary sodium levels below 20 mEq/L, polyuria, and dehydration. The therapy for DI is desmopressin, a synthetic ADH analog, which should be administered early to prevent metabolic complications described above. This medication can be administered intravenously, orally, or intranasally. Hypotonic saline solutions (e.g., 0.45% NaCl, 0.45% NaCl +5% dextrose, 0.18% NaCl +10% dextrose, 0.18% NaCl +4% dextrose, and 10% dextrose solutions) should only be used to treat active hypernatremia (Na >150 mEq/L) [36].

10.5.4 Hyperkalemia

Hyperkalemia (serum K^+ >5.5 mEq/L in infants and >6 mEq/L in neonates) can cause general skeletal muscle weakness and substantial ECG changes, especially when serum K^+ >7 mEq/L. Hyperkalemia is most commonly an artifact due to either hemolysis or release of K^+ during clot formation in the specimen tube. Other causes of hypopotassemia are drugs, including β-blockers and digitalis, myonecrosis, and acidosis, as well as renal disorders or failure [37]. First-line treatment of hyperkalemia should include the administration of 100 μg/kg of 10% calcium gluconate. The second line of treatment consists of administration of sodium bicarbonate (1–2 mmol/kg), an infusion of 0.3–0.5 mg/kg/h of glucose with 1 unit of insulin for every 5 gm of glucose added, or an infusion of 2.5–5 mg of nebulized salbutamol (5 μg/kg in neonates, IV) to increase intracellular shift of potassium. Finally, the removal of potassium from the body is achieved by administering 125–250 mg/kg calcium resonium rectally or orally, by using either furosemide (1 mg/kg), dialysis, or hemofiltration.

10.5.5 Hypokalemia

Hypokalemia (serum K^+ <3.5 mEq/L) is usually caused by diuretic therapy. In intensive care and perioperative settings, additional causes include nasogastric suctioning, magnesium deficiency, and alkalosis or loss of K^+ as a consequence of vomiting/diarrhea. Children are usually asymptomatic until K^+ reaches 2.5 mEq/L, and then they might present with muscular symptoms such as cramps, cardiological complications (including arrhythmias, reduced cardiac contractility, ECG alterations like U waves, loss of T waves, and QT prolongation), and neurological deterioration. Hypokalemia is associated with poor outcomes in patients with aneurysmal subarachnoid hemorrhage and therefore should be aggressively treated [38]. This is also true for symptomatic hypokalemic children [39]. Management should focus on reversing the transcellular shifts (alkalosis) and on potassium replacement. Potassium can be administered orally (3–5 mmol/kg/day) or intravenously (recommended in severe hypokalemia, i.e., serum K^+ <3 mEq/L). Potassium correction should not be faster than 0.25 mmol/kg/h while using a maximal peripheral concentration of 40 mEq/L of KCl. When a rapid correction is being done, it is essential to carry out the administration via a central line while monitoring the patient in the intensive care unit [38].

10.5.6 Hypocalcemia

Hypocalcemia (defined as corrected total Ca^{2+} <2 mEq/L, or <1.5 mEq/L in neonates) may produce various symptoms, including perioral, finger, and toe paresthesia, spasm, cardiac alterations including prolonged QT interval, and reduced cardiac contractility. Immediate treatment includes 10% calcium gluconate (0.5 ml/kg, up

to a maximum of 20 ml over 10 min) or a solution of 10% calcium chloride (0.2 ml/kg, up to a maximum of 10 ml over 10 min), possibly through a central venous route. Newborns are prone to hypocalcemia due to physiologically lower albumin concentration, a normal fall occurring after birth, which later rises after the second day, and maternal diabetes mellitus. Other causes include encephalopathy, renal failure, DiGeorge syndrome, and disordered maternal metabolism [38].

10.5.7 Hypercalcemia

Hypercalcemia presents with a low incidence (<1% of hospitalized patients), and most of the cases are caused by hyperparathyroidism or malignant tumors. More rare causes include thyrotoxicosis and drugs. Presentation is nonspecific, including cardiovascular (hypotension/hypovolemia and shortened QT interval on ECG), renal (polyuria, nephrocalcinosis), and neurological alterations. Patients require treatment if symptomatic, or if ionized Ca^{2+} >3.5 mEq/L (14 mg/dL). Hypercalcemia can also produce hypercalciuria, which results in osmotic diuresis and hypovolemia. These patients are volume depleted and, therefore, require saline infusions; furosemide can be used to enhance excretion, but it can exacerbate hypovolemia and hypotension.

10.5.8 Hypophosphatemia

Hypophosphatemia is seen when serum phosphate levels fall below 0.8 mEq/L (or <2.7 mg/dL) and occurs in 17–28% of postoperative patients. When glucose moves into cells, phosphate usually follows it, and thus, glucose overloading is the most common cause of hypophosphatemia in hospitalized patients [35]. Hypophosphatemia is often clinically asymptomatic, and even at levels of <1.0 mg/dL, it may not produce any obvious effects. However, common symptoms include muscle weakness and respiratory depression, and therefore, phosphate levels are to be monitored with attention in the postoperative period. Hypophosphatemia can lower cardiac output (CO), and patients with low CO and heart failure may respond to supplementation [38]. Phosphate replacement can be started intravenously if levels are very low, but when phosphate levels are higher than 2.0 mg/dL, a replacement can be given orally.

10.5.9 Hyperphosphatemia

It usually occurs after renal insufficiency (decreased secretion), widespread cell death related to tumor lysis, or rhabdomyolysis. Sucralfate calcium acetate tablets or antacids can be used to reduce serum levels.

10.5.10 Hypomagnesemia

Hypomagnesemia is found in 15% of ward patients and 60% of ICU patients. Main causes include other electrolyte abnormalities (hypokalemia, hypophosphatemia, hyponatremia, hypocalcemia), drugs (diuretics, furosemide, aminoglycosides, digitalis, amphotericin, cyclosporine), diarrhea, alcohol, and diabetes. The clinical findings include cardiac manifestations, ischemia, arrhythmia, and neurological deterioration. Hypomagnesemia has been shown to be studied in relation to vasospasm in patients with SAH, in both animal and human models. In a study including 283 SAH patients, 4 days of magnesium treatment compared with normal saline showed a favorable trend in the development of delayed cerebral ischemia (reduced by 34%) and resulted in a better outcome at 3 months [40]. Still, its role in a SAH is considered controversial.

10.5.11 Hypermagnesemia

Hypermagnesemia is found in up to 5% of hospitalized patients. The risk factors include massive hemolysis, renal adrenal insufficiency, hyperparathyroidism, and lithium toxicity. Magnesium is a calcium channel blocker, and thus, the promi-

nent effects of hypermagnesemia are cardiac (ECG alteration, including prolonged AV conduction, heart block, and cardiac arrest). To treat hypermagnesemia, IV calcium gluconate (1 mg over 2–3 min) should be given, and eventually, if not effective, dialysis should be started. With preserved renal function, aggressive fluid resuscitation and furosemide are appropriate as well.

10.6 Conclusions

Recognizing the diverse nature of pediatric neurosurgery complications is imperative for appropriate patient management during the perioperative periods. Disturbances in salts and water balances are relatively common in children, and present diagnostic and therapeutic challenges, especially after brain surgeries for suprasellar and pituitary tumors. Sodium disturbances, and in particular hyponatremia, are the most common and critical disturbances, even if DI is commonly encountered as well. A proper fluid balance assessment and a prompt diagnosis are mandatory to improve the outcome of the pediatric neurosurgical population.

Conflict of Interest None.

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