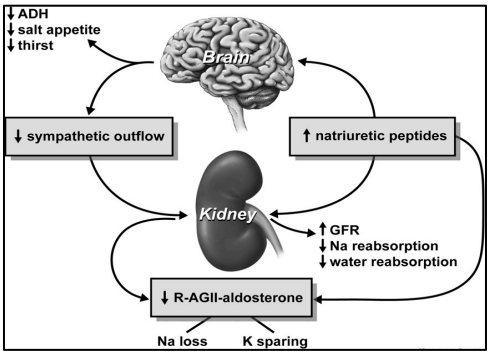
Cerebral Salt Wasting Syndrome

Anesthetic Pearls: Anesthetic Implications and Management of Cerebral Salt Wasting Syndrome

Hyponatremia can be a challenging problem for those who care for critically ill neurologic patients. The chief difficulty in this setting often lies in determining what is driving the fall in serum sodium concentration. Cerebral salt wasting (CSW) is a disorder of sodium and water handling that occurs as a result of cerebral disease in the setting of normal kidney function. It is characterized by hyponatremia in association with hypovolemia and, as the name implies, is caused by natriuresis. In routine clinical practice, distinguishing this condition from the more familiar syndrome of inappropriate secretion of antidiuretic hormone (SIADH) can be quite difficult. Nonetheless, this task is crucial because treatments for the two conditions are fundamentally different. Despite the clear association between the presence of CSW and severe neurologic disease, the mechanism underlying this association has not yet been clearly identified. Maintenance of body sodium and water homeostasis is a vital physiologic process and is largely governed by intricate interactions between the autonomic nervous system and humoral factors that influence the kidney's handling of sodium and water. Disruption of the normal interactions between these systems can generate sodium and water dysregulation at the level of the nephron and thereby lead to more global alterations in sodium and water homeostasis. It has been postulated that interference of sympathetic input to the kidney and the presence of abnormally high levels of circulating natriuretic factors after cerebral injury can lead to CSW.



Proposed Mechanism for Cerebral Salt Wasting Syndrome

RAAS Theory

The renin-angiotensin-aldosterone system (RAAS) is a hormonal pathway involving several enzymatic steps and humoral factors that serve a central role in maintaining whole-body sodium and water homeostasis. Renin is a circulating enzyme produced and stored within the kidney and released in response to low systemic and renal arterial perfusion. Once released, it initiates a series of intricate sequential enzymatic steps involving the well known angiotensin-converting enzyme, the ultimate product of which is the formation of angiotensin-II (AT-II). This potent vasopressor agent has immediate effects on blood pressure by influencing the constrictive properties of peripheral vasculature, increasing sympathetic tone, and stimulating the release of ADH.

Table 1
Differential diagnosis of CSW and SIADH

Variable	CSW	SIADH
Urine osmolality	↑ (>100 mOsm/kg)	↑ (>100 mOsm/kg)
Urine sodium concentration	↑ (>40 mmol/L)	↑ (>40 mmol/L)
Extracellular fluid volume	↓	↑
Body weight	↓	↔ or ↑
Fluid balance	Negative	Neutral to slightly +
Urine volume	↔ or ↑	↔ or ↓
Heart rate	↔ or ↑	↔
Hematocrit	↑	↔
Albumin	↑	↔
Serum bicarbonate	↑	↔ or ↓
Blood urea nitrogen	↑	↔ or ↓
Serum uric acid	↔ or ↓	↓
Sodium balance	Negative	Neutral or +
Central venous pressure	↓ (< 6 cm H_2O)	↔ or slightly + (6–10 cm H_2O)
Wedge pressure	↓	↔ or slightly ↑

Natriuretic Peptide Theory

Natriuretic peptides were initially discovered in the early 1980's after it was demonstrated that atrial myocardial extracts induced a potent natriuretic response when infused into rats. At about the same time, early studies investigating the pathogenesis of sodium and extracellular volume disturbances in patients with SAH led to the hypothesis that a natriuretic factor may be involved. Subsequently, a number of specific natriuretic substances were identified and their biologic effects have been intensely studied.

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Differentiating CSW from most other common causes of hyponatremia (diuretic use, adrenal insufficiency, extrarenal-induced volume-deplete states, hypothyroidism, congestive heart failure) is typically not difficult. The challenge lies in the differentiation of CSW from SIADH, because both disorders cause similar serum and urine laboratory abnormalities and occur in the same neurologic and neurosurgical diseases. SIADH is a euvolemic condition whereas CSW is a hypovolemic state. Accurately distinguishing between these two disorders is crucial, because misdiagnosis can lead to inappropriate therapy, often with serious consequences. Volume restriction instituted for a presumptive diagnosis of SIADH in patients with aneurysmal SAH and CSW, for example, has been shown to increase the risk of delayed ischemic deficits and mortality. Treatment based on an inaccurate diagnosis can also lead to progressive worsening of hyponatremia and its direct neurologic complications. Despite the availability and general ease in obtaining tests for the determination of electrolyte concentrations and osmolality in the serum and urine, only the careful determination of volume status in the hyponatremic patient accurately differentiates CSW from SIADH.

Treatment

The mainstay of therapy for CSW is replacement of the sodium and water that is lost as a result of pathologic natriuresis and diuresis. This is in direct contrast to the treatment of SIADH which is free water restriction. The key in diagnosis of CSW lies in distinguishing it from the more common SIADH, although the value of this often-imprecise process has recently been called into question. Volume status, but not serum and urine electrolytes and osmolality, is crucial for making this distinction. Volume and sodium repletion are the goals of treatment of patients with CSW, and this can be performed using some combination of isotonic saline, hypertonic saline, and mineralocorticoids.