

Central Neurogenic Diabetes Insipidus, Syndrome of Inappropriate Secretion of Antidiuretic Hormone, and Cerebral Salt-Wasting Syndrome in Traumatic Brain Injury

Cynthia (Cindi) A. John, RN, MSN, CNRN
Michael W. Day, RN, MSN, CCRN

Central neurogenic diabetes insipidus, syndrome of inappropriate secretion of antidiuretic hormone, and cerebral salt-wasting syndrome are secondary events that affect patients with traumatic brain injury. All 3 syndromes affect both sodium and water balance; however, they have differences in pathophysiology, diagnosis, and treatment. Differentiating between hyponatremia (central neurogenic diabetes insipidus) and the 2 hyponatremia syndromes (syndrome of inappropriate secretion of antidiuretic hormone, and cerebral salt-wasting syndrome) is critical for preventing worsening neurological outcomes in patients with head injuries. (*Critical Care Nurse*. 2012;32[2]:e1-e8)

CE Continuing Education

This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. List potential causes of central neurogenic diabetes insipidus (CNDI), syndrome of inappropriate secretion of antidiuretic hormone (SIADH), and cerebral salt-wasting syndrome (CSWS)
2. Compare the signs, symptoms, and laboratory values for CNDI, SIADH, and CSWS
3. Discuss the treatment and nursing management for CNDI, SIADH, and CSWS

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Traumatic brain injury (TBI) in adults continues to be a major cause of death and disability in the United States. An estimated 1.7 million persons in the United States will sustain TBI; of these, approximately 52 000 will die of the injury, 275 000 will be hospitalized, and 1.4 million will be treated and released from an emergency department.¹ Although young children (0-4 years old) and adolescents (15-19 years old) have the highest

risk of TBI, older adults (≥ 75 years) have the highest rates of TBI-related hospitalization and death.¹ Patients who survive the initial injury are likely to have secondary complications that can result in permanent disability. Approximately 80 000 to 90 000 patients experience long-term disability each year because of TBI.¹

The most common causes of TBI are falls (35.2%), motor vehicle accidents (17.3%), being struck by or against objects (16.5%), assaults (10%), and sports-related injuries and penetrating trauma (21%).¹ Although as many as 10% of TBIs result in death due to the primary injury, in most patients, marked morbidity and mortality are due to the effects of secondary injury. Primary injury is the damage caused by the initial trauma. Secondary injury, which occurs seconds, minutes, hours, or even days after the

initial trauma, is the result of biochemical processes that occur at the cellular level when neurons are damaged.² Hypotension, hypoxia, cerebral edema, and electrolyte imbalance further worsen neurological outcomes and markedly affect morbidity and mortality.³ In this article, we discuss how electrolyte imbalances caused by injury of the pituitary gland complicate the recovery of patients with TBI.

Pathophysiology of TBI

Primary TBI is caused by 2 main mechanisms: direct impact (ie, an object strikes the head or the brain) and acceleration-deceleration injury (ie, the force of the impact causes the brain to ricochet inside skull, resulting in shearing of cerebral axons). Direct impact caused by blunt trauma, falls, or penetrating injuries can result in cerebral edema and intracranial hemorrhage, which can lead to severe deterioration in a patient's clinical condition and even death.²

Acceleration-deceleration injuries often cause diffuse axonal injury. The rotational shearing of gray and white brain matter results in microscopic damage of the axons of the brain. Initially, diffuse axonal injury usually is not visible on imaging studies; however, because of axonal degeneration, abnormal findings such as edema, atrophy, and

petechial hemorrhages eventually are visible on magnetic resonance images.² Acceleration-deceleration forces can also cause injuries of the cranial nerves, the hypothalamus, and the pituitary stalk.² The damage that occurs with the primary injury is soon overtaken by the secondary injury from the cerebral edema, hemorrhage, or the hypoxia caused by a chain of ischemic events at the cellular level.

Ischemic Cascade of Neuronal Cell Death

Secondary injury of the brain is the damage that occurs seconds, minutes, hours, or even days after the traumatic event and may even be superimposed on a mechanical injury.² Because of the primary injury, oxygen and nutrients are not delivered to brain cells. Hypoxia due to decreased cerebral blood flow results in biochemical processes involving a cascade of ischemic events. This hypoxia causes dysfunction in normal cellular metabolism, and neurons die.³ The sequence of events begins with a lack of oxygen and cerebral perfusion, which causes the cellular ion pumps to fail, leading to anaerobic metabolism and buildup of lactic acid. Active transport of cellular ions becomes impaired, and calcium ions flow into the neurons. Excitatory neurochemical transmitter substances such

as glutamate are released, allowing even more calcium into the cells. This excess calcium causes release of oxygen free radicals and excess enzymes. The cell membrane is damaged by these enzymes, the mitochondria break down, and cellular death occurs. When cells die, more glutamate is released, more cells in the area are injured, edema increases, and the cascade spreads to undamaged neurons.³

Fluid and Electrolyte Imbalances

In addition to events at the cellular level, injury of the hypothalamus and the pituitary gland from forces transmitted to the head on impact, along with cerebral edema, often results in fluid and electrolyte disturbances that profoundly affect morbidity and mortality in TBI patients.⁴ Three common electrolyte imbalances are associated with the hypothalamic-pituitary dysfunction experienced by patients with TBI: central neurogenic diabetes insipidus (CNDI), syndrome of inappropriate secretion of antidiuretic hormone (SIADH), and cerebral salt-wasting syndrome (CSWS). CNDI is associated with hypernatremia, whereas SIADH and CSWS are associated with hyponatremia.⁵ Early recognition of all 3 syndromes is important in patients with TBI to prevent further neurological deterioration.

The pituitary gland, the pituitary stalk, and the hypothalamus are vulnerable to injury from head trauma.⁶ The hypothalamic-neurohypophyseal system is a collection of nuclei and tracts located in the hypothalamus and the pituitary gland that regulate body water balance. The paraventricular and supraoptic nuclei located in the

Authors

Cynthia (Cindi) A. John is a neuroscience nurse educator and Michael W. Day is trauma care coordinator at Providence Sacred Heart Medical Center and Children's Hospital, Spokane, Washington.

Corresponding author: Michael W. Day, RN, MSN, CCRN, 12915 E Main Ave, Spokane Valley, WA 99216 (e-mail: cnsd_ccrn@yahoo.com)

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hypothalamus synthesize antidiuretic hormone (ADH). ADH, which affects water balance by promoting reabsorption of water in the distal convoluted tubules and collecting ducts in the kidney, combines with a carrier protein called neurophysin. Together, neurophysin and ADH travel down the pituitary stalk and infundibulum through terminal nerve fibers to the pituitary gland.³ The ADH produced in the hypothalamus is stored in secretory granules in the posterior part of the pituitary gland, where the hormone can be released into the blood. The function of ADH is to maintain normal circulating blood volume and serum osmolality.⁵

Osmoregulation. Secretion of ADH is controlled by 2 principal negative-feedback mechanisms: osmoregulation and baroregulation. Osmoregulation is the mechanism used by the body to maintain water balance. Normal serum osmolality is 280 to 295 mOsm/kg. Even slight changes in serum osmolality can markedly affect ADH release.³ When serum osmolality is less than 280 mOsm/kg, an excess of body water occurs (the blood is diluted) and ADH is not secreted. When osmolality is greater than 295 mOsm/kg, a loss of body water occurs (the blood is more concentrated). ADH is then secreted to stimulate the collecting tubules of the kidney to increase water reabsorption to maintain water balance.³

Baroregulation. Regulation of ADH release is also affected by changes in blood volume and pressure. Baroreceptors located in the chest, left atrium, aortic arch, and carotid sinuses are sensitive to changes in blood pressure and circulating blood volume.³ Impulses from the

baroreceptors are transmitted through the vagus and glossopharyngeal nerves to the paraventricular and supraoptic nuclei in the hypothalamus. Increases in blood volume and blood pressure result in decreased ADH secretion. In patients with hypotension and hypovolemia (common in patients with TBI), secretion of ADH is increased, resulting in conservation of body fluids. Even a 5% to 10% decrease in blood volume or a 5% decrease in mean arterial pressure can stimulate the release of ADH.³

In general, the body first regulates ADH secretion in response to osmoregulation (concentration of body fluids). However, in severe volume depletion (hypotension or blood loss) baroreceptor stimulation of ADH takes precedence over osmoregulation.

Central Neurogenic Diabetes Insipidus

CNDI is characterized by an abnormal increase in urine output, an increase in fluid intake, and thirst due to decreased secretion of ADH, resulting in elimination of extracellular fluid. In trauma patients, CNDI usually is due to damage of the posterior part of the pituitary gland where ADH is stored and secreted.³ In patients with neurological conditions, CNDI is often associated with neurosurgery, tumors, increased intracranial pressure, brain death, and central nervous system infections such as meningitis or encephalitis.³ CNDI occurs in up to 16% of all brain-injured patients and usually occurs 5 to 10 days after trauma.⁷

CNDI usually occurs in 3 phases. The first phase consists of polyuria due to the inhibition of ADH that

lasts a few hours or up to several days. The second phase (5-6 days) is characterized by near-normal urinary output because of the release of stored ADH. The third phase is transient or permanent excessive urinary output due to depletion of stored ADH or loss of functioning of the cells that produce ADH.⁶ If the lack of ADH is uncorrected in patients with TBI, CNDI results in severe dehydration and further worsening of electrolyte balance.

Signs and Symptoms. Signs and symptoms of CNDI include polyuria (large volumes of dilute urine, 250 mL/h), polydipsia (extreme thirst in patients who are awake and alert), hypovolemia, and hypernatremia.^{5,6} Urinary specific gravity is less than 1.005 (normal, 1.005-1.030), urine osmolality is less than 200 mOsm/kg, serum osmolality is elevated (>295 mOsm/kg), the serum level of sodium is elevated (>145 mEq/L), and the urinary level of sodium is markedly decreased. Marked urinary losses of other electrolytes (potassium and magnesium) may occur simultaneously. Patients may also have weight loss of approximately 3% to 5% of body weight. Hypovolemia associated with CNDI in patients with TBI must be corrected. Other assessment findings may include indications of dehydration: confusion, irritability, poor skin turgor, dry mucous membranes, hypotension, and/or tachycardia.³

Diagnosis. Diagnosis of CNDI in patients with TBI is based on clinical signs and symptoms and laboratory findings, specifically polyuria, low urinary specific gravity, low urine osmolality, hypernatremia, and elevated serum osmolality (see Table).

Table Comparison of central neurogenic diabetes insipidus, syndrome of inappropriate secretion of antidiuretic hormone, and cerebral salt-wasting syndrome

Feature	Central neurogenic diabetes insipidus	Syndrome of inappropriate secretion of antidiuretic hormone (ADH)	Cerebral salt-wasting syndrome
Definition	Fluid imbalance due to decreased secretion of ADH in the posterior lobe of the pituitary gland or to renal unresponsiveness to the release of ADH	Persistent production or overproduction of ADH resulting in water intoxication and a volume-expanded state	Renal loss of sodium leading to true hyponatremia and a volume-contracted state in which the kidneys do not reabsorb sodium
Cause	Hypotension, stress, pain, anxiety, and an upright position Trauma, surgery, or damage of the hypothalamus	Head trauma, brain tumor, abscess, subarachnoid hemorrhage, hydrocephalus, meningitis, encephalitis, Guillain-Barré syndrome Pneumonia Drugs associated with increased ADH secretion (oral hypoglycemic agents, nonsteroidal anti-inflammatories, opiates, anesthetics)	Cause unclear but often occurs in patients with intracranial abnormalities (head trauma, stroke, subarachnoid hemorrhage, brain tumors) Loss of both intravascular fluid and sodium
Serum level of sodium, mEq/L	Hypernatremia >145 (high)	Hyponatremia <135 (low)	Hyponatremia <135 (low)
Serum osmolality, mOsm/kg	>295 (high)	<275 (low)	<275 (low)
Urinary osmolality, mOsm/kg	Decreased (<200)	Elevated (>100)	Elevated (>100)
Urinary level of sodium, mEq/L	Within normal reference range or decreased	Within normal reference range or elevated (>25)	Elevated (>25)
Urine output	Increased (>250 mL/h)	Decreased (400-500 mL/24 h)	Decreased
Urinary specific gravity	<1.005 (very dilute)	>1.010 (concentrated, dark)	>1.010 (concentrated, dark)
Extracellular fluid volume	Decreased	Increased	Decreased
Serum urea nitrogen	Elevated	Normal or low (dilutional)	Elevated
Mental status	Normal to impaired	Confusion Lethargy	Decreased level of consciousness, agitation, coma
Body weight	Decreased	Normal or increased	Decreased
Heart rate	Tachycardia	Slow or normal	Resting or postural tachycardia
Blood pressure	Normal to mildly hypertensive progressing to hypotension	Hypertensive	Postural hypotension
Treatment	Fluid replacement (0.45% saline intravenously replaced milliliter for milliliter, or greater) ADH replacement with desmopressin acetate intranasally or orally, lypressin intranasally, or aqueous vasopressin intravenously	Fluid restriction (800-1000 mL/24 h) Slow sodium replacement with normal saline or hypertonic (3%-5%) saline intravenously	Replacement of fluid volume and sodium No restriction of fluids Slow sodium replacement with hypertonic (3%) saline intravenously

Treatment. The goal in CNDI is to correct the ADH deficiency and restore fluid balance by promoting sodium and water reabsorption. In the acute phase of CNDI, exogenous ADH is provided, and fluid equivalent

to the amount of urine output is given either orally, if the patient can tolerate adequate oral intake, or intravenously.⁸ Patients with intact thirst centers who are able to take fluids orally are encouraged to drink

as much as possible when thirsty to keep up with fluid losses. However, in patients with TBI, complications from impaired level of consciousness, sensory and motor deficits, and dysphagia often preclude oral

intake, and intravenous solutions are required to meet the fluid demands. Intravenous hypotonic solutions most often used to replace lost body fluids include 0.45% saline titrated hourly to replace urine output.³

Exogenous ADH, either desmopressin (DDAVP), vasopressin, or lypressin, may be administered. Desmopressin can be administered nasally 5 to 2 µg/d in divided doses or parenterally 5 to 40 µg/d in daily divided doses.³ Vasopressin (aqueous Pitressin) can be administered intravenously 0.5 to 2 U every 3 hours for patients who have urine output of more than 300 mL/h for 2 consecutive hours.³ A vasopressin infusion may become necessary, which can be started at 0.2 U/min and titrated to a maximum dose of 0.9 U/min.⁵ Lypressin dosage is 5 to 20 U 3 to 7 times per day nasally.⁵

The following formula can be used to calculate the body water deficit (amount of fluid lost).

$(0.6 [\text{weight in kilograms}] \times (\text{serum sodium} - 140) \div 140 = \text{body water deficit (in liters)})$

For example, a patient with a serum sodium level of 150 mEq/L who weighs 70 kg would have a 3-L deficit:

$(0.6 [70] \times (150 - 140) \div 140 = 3 \text{ L})$
The resulting body water deficit can be used to calculate the volume of replacement fluids needed to restore hemodynamic stability in patients whose condition is unstable.⁹

Nursing Interventions. Caring for patients in the acute phase of CNDI requires monitoring of several parameters. Fluid intake and urinary output must be determined every 1 to 2 hours. Because the level of consciousness is usually impaired in patients with TBI, an indwelling

urinary catheter is necessary to monitor urine output accurately. Urinary output in the acute phase can be extraordinary: more than 250 to 800 mL/h (3-20 L/d). Urinary output greater than 200 mL/h for more for 2 consecutive hours should be reported to a physician.³ Urinary specific gravity should be determined every 1 to 2 hours; low specific gravity (<1.005) indicates that the kidneys are not concentrating urine. Serum osmolality and electrolyte levels, specifically sodium and potassium levels, should be measured at least daily, and frequently more often, depending on the findings and the patient's hemodynamic stability. Body weight should be determined daily, and patients should be assessed daily for signs and symptoms of worsening dehydration such as decreased skin turgor, dry mucous membranes, tachycardia, and hypotension. Trends are important. Is the patient getting worse? Or is the patient getting better? What do the laboratory values indicate?

Syndrome of Inappropriate Secretion of Antidiuretic Hormone

SIADH is characterized by abnormally high levels or continuous secretion of ADH, causing renal reabsorption and retention of water.¹⁰ Water is continually being reabsorbed by the kidneys, leading to concentrated urine, fluid retention, and hyponatremia. Hyponatremia is the most common electrolyte problem in patients with neurological problems and is particularly common after TBI; up to 33% of patients with TBI have hyponatremia.^{6,11}

Pathophysiologically, the negative feedback mechanisms that control the release of ADH do not function.

In the general population of hospitalized patients, many precipitating factors can cause SIADH: bronchogenic carcinoma, lung disease, pneumonia, positive-pressure breathing during mechanical ventilation, and certain medications (eg, morphine, chlorpromazine, chlorpropamide, chlorothiazide, carbamazepine, and acetaminophen). Conditions that cause SIADH in patients with TBI include traumatic subarachnoid hemorrhage, increased intracranial pressure, and injury of the hypothalamic-neurohypophyseal system. The damage of the hypothalamus-pituitary region that causes ADH dysfunction results in increases in water reabsorption by the renal tubules, decreases in urine volume, fluid retention, and extracellular volume expansion.¹² Patients experience excessive water retention and dilutional hyponatremia with a 5% to 10% increase in body weight due to expansion of extracellular volume.⁵

Signs and Symptoms. The signs and symptoms of SIADH include decreased urinary output, to less than 400 to 500 mL/24 hours, and generalized weight gain due to excess fluid retention.⁵ Laboratory findings include low serum levels of sodium (<135 mEq/L), serum hypoosmolality (<275 mOsm/L), elevated urinary levels of sodium (>25 mEq/L), and elevated urine osmolality (greater than serum osmolality).⁵

Diagnosis. Diagnosis of SIADH is based on the clinical signs and symptoms and laboratory findings. Complications due to SIADH and hyponatremia include fluid retention, headache, nausea, vomiting, muscle twitching, fatigue, confusion, lethargy, and, possibly, seizures

(usually in patients with a serum level of sodium <120 mEq/L).³ In patients with TBI, the underlying pathological changes result in hyponatremia due to excessive ADH release, which causes renal reabsorption of water and expansion of extracellular volume.

Treatment. The treatment of SIADH is focused on fluid restriction (<1000 mL/24 h) and slow, careful replacement of sodium with an intravenous hypertonic solution of sodium chloride (3% saline) and/or diuretics such as furosemide. Medications to suppress ADH activity (demeclocycline hydrochloride) or inhibit renal response to ADH (lithium carbonate) are also options.^{3,6}

Administration of hypertonic intravenous solutions such as 3% sodium chloride requires careful titration because a too-rapid correction of hyponatremia can result in central pontine myelinolysis,¹³ an irreversible demyelination of the neurons in the pons of the brain stem. The exact mechanism that causes demyelination is unknown; the recommended rate of serum sodium correction is 10 to 20 mEq/d.¹³

Nursing Management. Nursing management for patients with SIADH and hyponatremia is comparable to the management of patients with CNDI. Intake of fluids, urinary output, and urinary specific gravity should be determined every 1 to 2 hours. Urinary and serum levels of electrolytes and osmolality should be monitored. Patients should be assessed for signs and symptoms of worsening outcome. Accurate body weights should be recorded daily for the purpose of monitoring fluid retention. Patients with SIADH require frequent oral care because

fluid restriction causes dry mouth. Indications of improvement include correction of low serum levels of sodium, lowered urine osmolality, lowered urinary levels of sodium, and increased serum osmolality.²

Cerebral Salt-Wasting Syndrome

Unlike SIADH, CSWS is characterized by a true hyponatremia, that is, hyponatremia that results in a loss of both sodium and extracellular fluid.¹¹ Even though ADH levels are elevated in patients with CSWS, the body loses extracellular fluid and plasma volume decreases, resulting in decreased body weight (volume-contracted state).¹⁰ The pathophysiology of CSWS is unclear but is thought to be due to multiple mechanisms that affect sodium and water balance.¹¹ The primary pathogenic mechanism is renal loss of sodium, which leads to hyponatremia and a decrease in extracellular volume.¹¹ Although the syndrome occurs most often in patients with stroke, intracerebral hemorrhage, subarachnoid hemorrhage, and intracranial surgery, it may develop in patients with TBI who have increased intracranial pressure.

Signs and Symptoms. Patients with CSWS may report headache and increased thirst. Clinical indications include orthostatic hypotension, tachycardia, dehydration, weight loss, dry mucous membranes, lethargy, decreased level of consciousness, seizures, and coma.³

Diagnosis. The diagnosis of CSWS is based on the clinical manifestations and the following laboratory findings: primary hyponatremia, serum hypoosmolality, elevated urine osmolality, elevated urinary levels of sodium, increased levels of serum

urea nitrogen, increased hematocrit, and increased urinary specific gravity. Even though the mechanism of CSWS is not well understood, the primary distinction between CSWS and SIADH is volume status.¹⁰ Indications of volume depletion (hypotension, weight loss, and decreased skin turgor) occur with CSWS, whereas indications of volume expansion occur with SIADH (decreased urine output and generalized weight gain due to fluid retention).

Treatment. Determining the cause (SIADH or CSWS) of hyponatremia in trauma patients is important. SIADH requires strict fluid restriction and/or slow, judicious administration of hypertonic saline, whereas CSWS requires replacement of fluid volume with physiological saline and intravenous replacement with hypertonic 3% sodium chloride solution. As in treatment of SIADH, hypertonic solutions (3% sodium chloride) must be administered slowly because too-rapid correction of hyponatremia can result in central pontine myelinolysis.^{3,13} In patients who tolerate oral intake, fluid can be replaced orally, often with salt tablet supplements.

Restriction of fluids is contraindicated in patients with CSWS. If fluids are restricted, patients will be at risk for cerebral vasospasm, cerebral ischemia, and/or infarction.³ Other medical interventions may include treatment with fludrocortisone acetate to increase absorption of sodium by the renal tubules.¹⁴

Nursing Management. Nursing management of patients with CSWS is comparable to that of patients with CNDI or SIADH. Isotonic or hypertonic fluids are administered intravenously to obtain positive fluid

balance and correct volume depletion. Sodium can also be replaced orally. Cardiac status should be monitored to detect side effects of medications and fluid volume status. The goal of treatment of CSWS is to replace sodium and fluid volume with intravenous saline or salt tablets. Fluid restriction is definitely contraindicated and can worsen neurological outcomes.

Summary

Caring for patients with complex neurological problems, specifically patients with TBI and electrolyte imbalances, can be challenging. Understanding and recognizing the signs and symptoms of CNDI, SIADH, and CSWS will guide nurses in the appropriate actions to take to avoid deterioration in a patient's condition. In summary, CNDI is a hyponatremia characterized by elevated serum levels of sodium and serum osmolality, but low urine specific gravity. Patients have large-volume urinary output and extreme thirst. Treatment is replacement of fluid volume either orally or intravenously or treatment with ADH.

SIADH and CSWS are both characterized by hyponatremia. Signs and symptoms are remarkably similar; however, patients with CSWS experiences a true loss of sodium and intravascular fluid. Both sodium and fluid must be replaced to correct the imbalance. In patients with SIADH, sodium is replaced, but fluid is restricted. Monitoring patients for trends in neurological status, laboratory findings, and physiological parameters will guide nurses in determining whether treatment is effective or not. **CCN**

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Financial Disclosures

None reported.

References

1. Faul M, Xu L, Wald MM, Coronado VG. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002-2006. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. http://www.cdc.gov/traumatic-braininjury/pdf/blue_book.pdf. Published March 2010. Accessed January 9, 2012.
2. Pangilinan PH Jr, Kelly BM, Hornyak JE IV, et al. Classification and complications of traumatic brain injury. <http://emedicine.medscape.com/article/326643>. Updated November 10, 2011. Accessed January 9, 2012.
3. Hickey JV. Fluid and metabolic disorders in neuroscience patients. In: *The Clinical Practice of Neurological and Neurosurgical Nursing*. 6th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2009:195-205.
4. Boughey J, Yost M, Bynoe R. Diabetes insipidus in the head-injured patient. *Am Surg*. 2004;70:500-503.
5. Byrum D, Kirkwood PL. Pituitary, thyroid, and adrenal disorders. In: Carlson KK, ed. *AACN Advanced Critical Care Nursing*. St Louis, MO: Saunders/Elsevier; 2009:939-965.
6. Klein MJ. Post head injury endocrine complications. <http://emedicine.medscape.com/article/326123>. Updated January 5, 2009. Accessed January 9, 2012.
7. Hadjizacharia P, Beale EO, Inaba K, Chan LS, Demetriades D. Acute diabetes insipidus in severe head injury: a prospective study. *J Am Coll Surg*. 2008;207(4):477-484.
8. Cooperman M. Diabetes insipidus. <http://emedicine.medscape.com/article/117648>. Updated June 17, 2011. Accessed January 9, 2012.
9. Urdan L, Stacy K, Lough M, eds. Endocrine disorders and therapeutic management. In: *Thelan's Critical Care Nursing: Diagnosis and Management*. 5th ed. St Louis, MO: Mosby-Elsevier; 2006:918-966.
10. Palmer B. Hyponatremia in patients with central nervous system disease: SIADH versus CSW. *Trends Endocrin Metab*. 2003;14(4):182-187.
11. Yee A, Burns J, Wijidicks E. Cerebral salt wasting: pathophysiology, diagnosis, and treatment. *Neurosurg Clin North Am*. 2010; 21(2):339-352.
12. Zhang W, Li S, Visocchi M, Wang X, Jiang J. Clinical analysis of hyponatremia in acute craniocerebral injury. *J Emerg Med*. 2010; 39(2):151-157.
13. Mortimer DS, Jancik J. Administering hypertonic saline to patients with severe traumatic brain injury. *J Neurosci Nurs*. 2006;38(3):142-146.
14. Momi J, Tang CM, Abcar AC, Kujubu DA, Sim JJ. Hyponatremia—what is cerebral salt wasting? *Perm J*. 2010;14(2):62-65.

CE Test Test ID C1222: Central Neurogenic Diabetes Insipidus, Syndrome of Inappropriate Secretion of Antidiuretic Hormone, and Cerebral Salt-Wasting Syndrome in Traumatic Brain Injury Learning objectives: 1. List potential causes of central neurogenic diabetes insipidus (CNDI), syndrome of inappropriate secretion of antidiuretic hormone (SIADH), and cerebral salt-wasting syndrome (CSWS) 2. Compare the signs, symptoms, and laboratory values for CNDI, SIADH, and CSWS 3. Discuss the treatment and nursing management for CNDI, SIADH, and CSWS

1. Which of the following groups is at greatest risk of death from a traumatic brain injury (TBI)?

- a. Young children (0-4 years old)
- b. Adolescents (15-19 years old)
- c. Young adults (20-27 years old)
- d. Older adults (older than 75 years)

2. Which of the following is the most common cause of TBI?

- a. Falls
- b. Motor vehicle crashes
- c. Sports-related injuries
- d. Penetrating trauma

3. Which of the following types of TBI is a patient injured in a rear end collision most likely to experience?

- a. Penetrating injury
- b. Acceleration-deceleration injury
- c. Direct injury
- d. Hemorrhagic injury

4. What is the role of glutamine in secondary brain injury?

- a. Decrease lactic acid buildup
- b. Decrease oxygen free radical release
- c. Increase calcium release into the neurons
- d. Increase adenosine triphosphate production

5. Which of the following disorders is associated with hypernatremia?

- a. Central neurogenic diabetes insipidus (CNDI)
- b. Syndrome of inappropriate secretion of antidiuretic hormone (SIADH)
- c. Cerebral salt-wasting syndrome (CSWS)
- d. TBI

6. Where is antidiuretic hormone (ADH) stored in the body?

- a. Infundibulum
- b. Hypothalamus
- c. Posterior pituitary gland
- d. Kidney

7. When the serum osmolality is 300 mOsm/kg, which of the following would occur?

- a. Adenosine triphosphate is produced.
- b. ADH is secreted.
- c. The collecting tubules decrease water reabsorption.
- d. Lactic acid is produced.

8. A patient with TBI has these symptoms: urine specific gravity, 1.003; urine osmolality, 298 mOsm/kg; serum osmolality, 305 mOsm/kg; and serum sodium, 150 mEq/L. Which of the following is the likely cause?

- a. CNDI
- b. SIADH
- c. CSWS

9. Which of the following is an appropriate intervention for a patient with CNDI?

- a. Replace urine output mL per mL with D5NS
- b. Desmopressin infusion at 5 µg/kg/h
- c. Vasopressin infusion 0.1 U/min
- d. Lypressin 5 units 7 times per day

10. Which of the following signs and symptoms are associated with SIADH?

- a. Decreased urinary output and weight gain
- b. High serum sodium and low urinary sodium
- c. Low serum osmolality and low urinary sodium
- d. High serum sodium and high urinary sodium

11. Which of the following interventions should the nurse question regarding SIADH treatment?

- a. Rapid infusion of 500 mL of 3% saline
- b. Frequent oral care
- c. Fluid restriction
- d. Intravenous administration of 40 mg of furosemide

12. Which of the following is the main difference between SIADH and CSWS?

- a. In CSWS the patient is volume depleted and in SIADH the patient is volume overloaded.
- b. In CSWS the patient is hyponatremic and in SIADH the patient is hypernatremic.
- c. In CSWS the patient is hypernatremic and in SIADH the patient is hyponatremic.
- d. In CSWS the patient is volume overloaded and in SIADH the patient is volume depleted.

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

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Test ID: C1222 Form expires: April 1, 2014 Contact hours: 1.0 Fee: AACN members, \$0; nonmembers, \$10 Passing score: 9 correct (75%) Synergy CERP: Category A
 Test writer: Marylee Bressie, MSN, RN, CCRN, CCNS, CEN

AMERICAN ASSOCIATION of CRITICAL-CARE NURSES

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Program evaluation

- | | | |
|--|--------------------------|--------------------------|
| | Yes | No |
| Objective 1 was met | <input type="checkbox"/> | <input type="checkbox"/> |
| Objective 2 was met | <input type="checkbox"/> | <input type="checkbox"/> |
| Objective 3 was met | <input type="checkbox"/> | <input type="checkbox"/> |
| Content was relevant to my nursing practice | <input type="checkbox"/> | <input type="checkbox"/> |
| My expectations were met | <input type="checkbox"/> | <input type="checkbox"/> |
| This method of CE is effective for this content | <input type="checkbox"/> | <input type="checkbox"/> |
| The level of difficulty of this test was: | | |
| <input type="checkbox"/> easy <input type="checkbox"/> medium <input type="checkbox"/> difficult | | |
| To complete this program, it took me _____ hours/minutes. | | |

Name _____ Member # _____
 Address _____
 City _____ State _____ ZIP _____
 Country _____ Phone _____
 E-mail _____
 RN Lic. 1/St _____ RN Lic. 2/St _____
 Payment by: Visa M/C AMEX Discover Check
 Card # _____ Expiration Date _____
 Signature _____

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