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Concentration and Volume: Understanding Sodium and Water in the Body

The etiology of dysnatremia can be complex.

ABSTRACT

Nurses need to have a firm grasp of normal and pathophysiological mechanisms of sodium and water balance to fully understand assessment findings and establish a rationale for a patient's plan of care. While multiple mechanisms control sodium and water balance, antidiuretic hormone and aldosterone are the most important hormonal influences. This article, the first in a new series designed to improve nurses' understanding of the physiological abnormalities underlying many disorders, reviews the common etiologies and symptoms of hyponatremia and hypernatremia, as well as the role of nursing care in patients with imbalances of sodium and water. Case studies guide the reader through relevant medical history and examination findings to an understanding of both the nursing and medical plans of care.

Keywords: dysnatremia, fluid balance, hypernatremia, hyponatremia, nursing care

A 28-year-old ED patient has been admitted with a diagnosis of acute hyponatremia. The patient has no significant medical history but has been training for a half-marathon in 90° weather for the past week. You note the patient's serum sodium level is 125 mEq/L. The patient received a fluid bolus in the ED, and the admitting orders include a normal saline infusion. The patient is drowsy and confused, and his family is asking questions about the etiology and treatment of his hyponatremia.

Before you can begin to address the patient's needs and the family's questions, you need to consider how the patient landed in the ED with these symptoms and your role in ensuring his appropriate treatment and patient and family education. Diagnosing disorders of sodium and water balance is complex, requiring careful consideration of medical history and physical examination findings and, often, additional testing to determine the most likely etiology. Treatment for dysnatremia (either hyponatremia or hypernatremia) is also guided by volume status; and determining whether a patient is hypovolemic or hypervolemic can be complicated by multiple comorbidities and variations in the clinical picture. (For example, a patient with systolic heart failure who stopped taking furosemide and who has pneumonia and is hypotensive may be at risk for hypervolemia, but careful fluid resuscitation may still be needed to treat septic shock.) While managing medical diagnoses is ultimately the responsibility of the attending provider (NP, physician, or physician assistant), the nurse at the bedside who understands sodium and water balance is well equipped to recognize important changes in a patient's condition and make informed recommendations for ongoing care.

This article will provide an overview of the regulation of sodium and water balance in the body; guide the nurse through some of the common reasons for imbalances; and discuss major nursing responsibilities, including understanding signs, symptoms, and treatment approaches. This article is the first in *Back to Basics*, a new series devoted to improving acute care nurses' understanding of the physiological abnormalities underlying many disorders and applying that understanding to enhance physical assessment techniques and refine the nursing plan of care.

BACK TO BASICS: HOW THE BODY REGULATES SODIUM AND WATER BALANCE

Although sodium and water exist in the body as a solution, it's important to remember that distinct yet interrelated processes regulate the concentration and volume of this solution.

Osmolality and osmotic pressure. It's necessary to understand osmolality and osmotic pressure to understand the distribution of fluid and electrolytes in the body. The intracellular compartment is distinct from the extracellular compartment (which comprises both the interstitial and intravascular compartments), and each has its own osmolality, with tightly regulated concentrations of solutes and volumes of water. Cell membranes are semipermeable and do not provide an absolute barrier to the movement of water and solutes between compartments. When there is a difference in osmolality (concentration of solutes) between the two sides of a semipermeable membrane, this concentration gradient creates osmotic pressure, which tends to equalize osmolality on both sides of the membrane. Because of osmotic pressure and the semipermeable nature of cell membranes, water will move from an area of low to high solute concentration until osmolality is equilibrated on both sides of the membrane.[1,2] A hypotonic (low solute concentration) extracellular environment will result in water movement into cells and cells will swell, while a hypertonic (high solute concentration) extracellular environment will cause water movement out of cells and cell shrinkage.

Concentration can be altered by changing the amount of the solute (sodium) or by changing the volume of the solvent (water).

Sodium gradient. There is a vastly different concentration of sodium in the intracellular and extracellular compartments, which creates an important sodium gradient across cell membranes. The sodium–potassium–ATPase pump maintains a much higher concentration of sodium in the extracellular compartment.[2] The sodium gradient is essential for optimal cell functioning as well as for maintaining stable intracellular and extracellular volume. In most cells, there is relatively free movement of both water and sodium across membranes, so changes in the extracellular osmolality are quickly equilibrated with the intracellular environment, and cell volume remains stable despite changes in serum sodium concentration (see Figure 1). However, in the central nervous system, the blood–brain barrier limits sodium movement relative to water movement. As a result, rapid changes in serum sodium affect brain tissue volume. Brain tissue shrinks in acute hypernatremia and swells in acute hyponatremia. Swelling of brain tissue can cause increased intracranial pressure, and therefore increases the risk of brain stem herniation and death.[2,3]

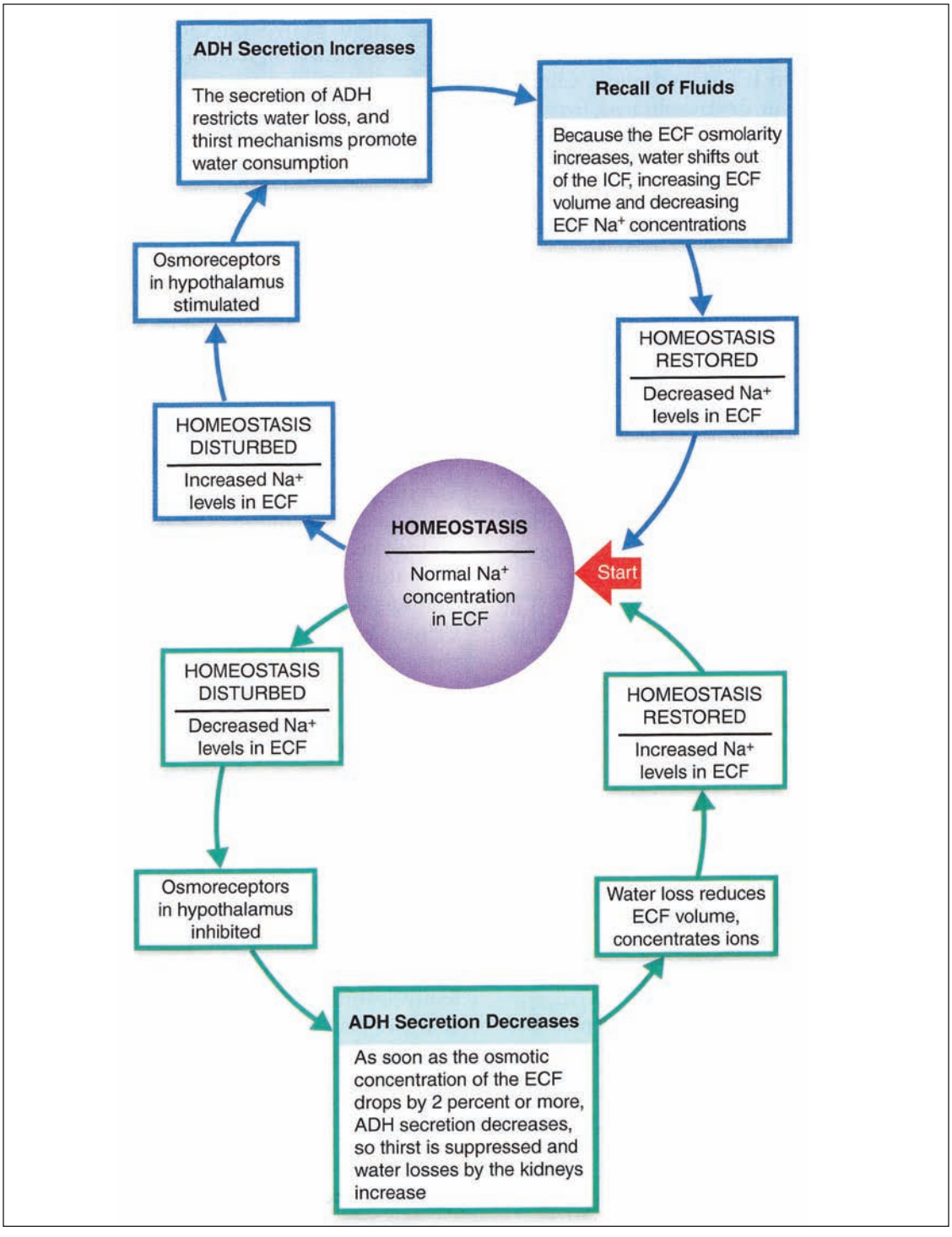
While multiple processes work together to regulate concentration and volume—primary among these being an intact thirst mechanism and the ability to drink water—this discussion will focus on the roles of antidiuretic hormone (ADH), also known as vasopressin, and aldosterone.

ADH. The serum concentration of sodium is regulated primarily by ADH, which is secreted by the hypothalamus and released from the posterior pituitary gland. ADH acts in the distal tubules and collecting ducts of the nephrons of the kidney to control the reabsorption of water (through production of special water channels called aquaporins), which in turn regulates the concentration of solutes in urine and overall urine volume. As the nephrons produce filtrate, certain hormones can act to alter its concentration and composition before it passes out into the collecting system as urine. As serum sodium and osmolality increase, more ADH is released, the nephrons reabsorb more water from the filtrate, and the kidneys produce a lesser volume of more concentrated urine. The water that is reabsorbed into the bloodstream from the filtrate increases the extracellular volume and dilutes sodium, so the sodium concentration returns to normal. If the serum sodium decreases, less ADH is released, less water is reabsorbed by the kidneys, and a larger volume of dilute urine is produced. By losing more water relative to sodium, sodium concentration increases, returning to normal.[1]

In addition to the osmolality-related release described above, ADH release is also mediated by stretch receptors in the heart, carotid sinus, and aortic arch. For example, when intravascular volume is low, there is less stretch on these receptors, so more ADH is released and more water is retained in the kidneys to increase intravascular volume.[4]

Aldosterone is secreted from the adrenal cortex as the final step in the renin–angiotensin–aldosterone pathway. In response to reduced pressure in the afferent arterioles of the kidneys (such as happens in hypotension), the juxtaglomerular cells produce renin. Renin leads to increased angiotensin II, which causes both vasoconstriction and the release of aldosterone. Aldosterone acts in the renal tubules to increase reabsorption of both sodium and water. In essence, aldosterone causes the body to retain

Figure 1. Mechanisms Involved in Maintaining Sodium Homeostasis



These two cycles illustrate the response of ADH to fluctuating levels of sodium in extracellular fluid. The top (blue) cycle shows the body's response to increased sodium levels, while the bottom (green) cycle shows the body's response to decreased sodium levels. ADH = antidiuretic hormone; ECF = extracellular fluid; ICF = intracellular fluid; Na+ = sodium. From Sorenson M, et al. *Pathophysiology: Concepts of Human Disease*. 2018. Reprinted with permission by Pearson Education, Inc., New York, NY.

more salt water (that is, saline), thereby increasing extracellular volume without altering sodium concentration.[1]

Assessing volume status. The interplay of hormones like ADH and aldosterone, as well as other factors such as the thirst mechanism; environmental fluid loss through sweating, breathing, and stool; comorbidities; and certain medications, can lead to one of several clinical pictures. A patient with hyponatremia can have hypovolemia, hypervolemia, or even euvolemia (normal fluid volume status), and the same can be said of a patient with hypernatremia. Because sodium concentration and volume status are connected but distinct, symptoms of dysnatremia will vary depending on the patient's volume status.[5] For example, a patient with hypervolemia will have acute weight gain (1 kg = 1 liter of water) and possibly other signs of excessive volume, such as edema, bounding pulses (a greater than normal amplitude), jugular venous distention, hypertension, and pulmonary symptoms (such as orthopnea, crackles, and impaired oxygenation). A patient with hypovolemia may have acute weight loss, poor skin turgor, dry mucus membranes, orthostatic tachycardia and hypotension, and concentrated urine and/or low urinary output. The nurse must consider all such examination findings, which are indicative of volume status, in order to fully understand a patient with dysnatremia.

Recognizing dysnatremia. Table 1[1, 2, 4, 6-9] provides a basic summary of etiologies and clinical manifestations of altered sodium concentration. Note that most of the purely sodium concentration–related symptoms are neurologic, because rapid increases or decreases in serum sodium (those occurring in 24 to 48 hours) quickly affect brain tissue volume.[3]

BACK TO THE BEDSIDE: CASE STUDIES

When we test serum sodium levels, it's important to remember that we are measuring the concentration of sodium in a solution, which is only one part of understanding concentration and volume. Concentration can be altered by changing the amount of the solute (sodium) or by changing the volume of the solvent (water). What does this mean for your patient?

A laboratory value of sodium obtained from a blood test is a snapshot of the serum sodium concentration at a given moment; to understand the patient's volume status (hypovolemia, hypervolemia, or euvolemia), nurses must also consider medical history and physical examination findings. Consider the following two cases to understand the significance of these factors (both cases are composites based on my clinical experience).

Case 1: Acute hyponatremia. Let's revisit the case presented at the beginning of this article. In discussion with the patient's family, you learn that he has been drinking water after running, started vomiting yesterday, and hasn't been able to keep anything down since. Examination findings are key to completing the clinical picture, and you note stable vital signs, drowsiness, confusion, poor skin turgor, and dry mucus membranes, all of which are consistent with hypovolemic hyponatremia. You can now explain to the family that the patient's sodium is low because he has been diluting his body's electrolytes with water and not replacing electrolytes after heavy sweating. Because of the rapid onset of hyponatremia, the patient may have experienced water movement into brain tissue, which could cause his neurologic symptoms of lethargy, confusion, nausea, and vomiting. In addition, he is hypovolemic because of the fluid volume lost through sweating, vomiting, and poor oral intake. Until he is awake and able to drink, isotonic IV fluids will be important to replace fluid and sodium losses.

Case 2: Acute hypernatremia. Let's consider a slightly more complex case. An 80-year-old female patient is on day 3 of an admission for septic shock and pyelonephritis. Her medical history includes systolic heart failure, type 2 diabetes, and Alzheimer's disease. The patient transferred out of intensive care the day before and is currently hemodynamically stable and requiring 3 L/min of oxygen via nasal cannula. Relevant orders include 100 mL/hr of normal saline and nothing by mouth. You note a morning sodium value of 149 mEq/L compared to 144 mEq/L the day before and 139 mEq/L two days prior.

Further consideration of this patient's hospitalization and current status is warranted before developing a plan of care for the day. As part of treatment for septic shock, the patient received 30 mL/kg of normal saline on admission, and IV fluids have been running for three days. Poor ejection fraction associated with systolic heart failure places the patient at risk for volume overload, and the need for supplemental oxygen also points to possible volume overload. However, the patient is making urine and kidney function is stable. You ask yourself: Is the patient hypovolemic and in need of ongoing volume administration and free water? Or is the patient volume and sodium overloaded and in need of diuresis and reduced sodium intake with free water replacement?

Your relevant physical examination findings include the following:

- vital signs–temperature, 98.3°F; pulse, 88 beats per minute; respiratory rate, 20 breaths per min-

Table 1. Common Etiologies and Symptoms of Hypernatremia and Hyponatremia

	Hypernatremia (serum sodium > 145 mEq/L)	Hyponatremia (serum sodium < 135 mEq/L)
Common etiologies	Water loss as a result of • Vomiting and diarrhea • Sweating • Osmotic diuresis as in hyperglycemia with glycosuria • Loop diuretics • Diabetes insipidus (inadequate ADH) • Impaired thirst and/or limited access to water[6] Sodium gain as a result of • Oral ingestion of sodium • Iatrogenic causes, as in the administration of excessive sodium-containing solutions or enemas[6-8]	• Chronic conditions (heart, kidney, and liver failure) associated with increased fluid volume[4] • High intake of low-solute fluids, as in primary polydipsia, beer potomania,[4] and iatrogenic causes (excessive hypotonic IV fluid administration) • Syndrome of inappropriate ADH[4] ○ CNS disorders, such as subarachnoid hemorrhage[4] ○ Other conditions that increase ADH secretion or produce ADH ectopically (for example, nausea, pain, stress, small cell lung cancer, pneumonia)[4] ○ Drugs that stimulate ADH (for example, SSRIs, carbamazepine, lamotrigine)[9] • Other drugs: thiazide diuretics, ACE inhibitors, ARBs[9] • Vomiting and diarrhea[4] • Heavy sweating leading to electrolyte losses[4] • Adrenal insufficiency[4] • Renal salt wasting[4]
Symptoms	Acute: • Confusion[1] • Lethargy[1] • Seizures[2] • Coma[2] • Intracranial hemorrhage[2] Chronic: • Obtundation or coma[1, 2]	Acute: • Confusion or delirium[2, 4] • Headache[2, 4] • Nausea and vomiting[4] • Malaise, fatigue, somnolence[2, 4] • Seizures[2, 4] • Coma[2, 4] Chronic: • Malaise and fatigue[2] • Gait disturbances and falls[2, 4] • Cognitive impairment[4] • Osteoporosis and fractures[4] • Seizures (in cases of very low serum sodium)[2]

ACE = angiotensin-converting enzyme; ADH = antidiuretic hormone; ARB = angiotensin receptor blocker; CNS = central nervous system; SSRI = selective serotonin reuptake inhibitor.

ute; blood pressure, 132/86 mmHg; and oxygen saturation, 93% on 3 L/min oxygen
- neurologic–awake and asking for a drink, oriented to person only
- cardiovascular–1+ pretibial edema; weight is up 2.5 kg since admission
- respiratory–bilateral lower lobe crackles anterior and posterior, but no respiratory distress
- intake and output–net positive 1 L in the previous 24 hours; urinary output, 70 to 80 mL/hr

Although the patient doesn't have acute neurologic symptoms from hypernatremia, considering the multiple indicators of mild hypervolemia (the need for supplemental oxygen, edema, weight gain, and pulmonary crackles), you conclude she has hypervolemic hypernatremia. The patient is likely suffering from both volume overload and sodium gain from excessive normal saline administration in the setting of chronic systolic heart failure. The patient might need free water replacement (by changing IV fluids to something hypotonic or allowing the patient to drink or both). Since the patient is making urine and doesn't require much oxygen, it may be fine to wait for her kidneys to eliminate the excess volume. In situations in which the hypervolemia is causing more of a problem (for example, severe hypoxemia or hemodynamic instability), careful administration of loop diuretics such as furosemide (Lasix) may be needed, along with replacement of the free water deficit.

Since this patient can drink to supplement free water and is making adequate urine, the attending provider orders a sodium-restricted diet and a basic metabolic

panel for the morning, discontinues the IV fluids, and requests careful tracking of intake and output and daily weight.

If you had found dry mucous membranes, poor skin turgor, and tachycardia or hypotension with position changes, the patient might still need volume, but the fluids would be changed to something hypotonic, and the patient would be allowed to start drinking, if appropriate.[5]

NURSING'S ROLE

Nurses have several important roles in assessing and managing the patient with a sodium and water imbalance, including the following:

Help discover the cause. As described in the cases above, nurses should integrate medical history and physical assessment findings to help inform the clinical picture. For many patients, additional diagnostic testing is warranted. Recall that ADH affects how much water is eliminated in urine, thereby regulating the concentration of serum sodium. For example, in a patient with hyponatremia, the appropriate compensatory response of the posterior pituitary should be to release ADH, which should result in urine that is dilute relative to the serum as the body eliminates more water than solute. By evaluating the concentrations of sodium in both serum and urine, the attending provider can determine whether the problem is with the compensatory mechanism itself (regulation of ADH secretion by the posterior pituitary) or because the compensatory mechanism cannot keep up with another primary problem affecting sodium concentration. If you receive an order to test to compare serum and urine osmolality and sodium concentration, make sure to maximize diagnostic usefulness by collecting samples at the same time, preferably before diuretics are given.

Help guide safe treatment. Treatment of dysnatremias is generally a slow process with serial measurement of sodium levels, especially if the onset was gradual or chronic. Rapidly decreasing serum sodium levels can result in cerebral edema, which can lead to neurologic damage, brain herniation, and death. Rapidly increasing sodium levels, especially in chronic hyponatremia, can result in astrocyte damage and osmotic demyelination syndrome, a severe condition that can also lead to permanent brain damage or death.[3] If you are caring for patients with dysnatremia, advocate for your patient through timely sample collection and communication of laboratory findings. Most importantly, be alert to even subtle changes in neurologic status and notify an attending provider promptly.

Prevention is key. Dysnatremias often develop in hospitalized patients, and nurses can play a critical role in prevention. You are already recording your patients' laboratory test results every day, so take it a step further: Is there an upward or downward trend in the serum sodium? What is the patient's volume status? Are there neurologic signs that may indicate complications of the dysnatremia? Is there relevant information from your physical examination that might explain why a change in sodium concentration is developing? Are there any orders or treatments that can account for this? Look at IV fluids and oral intake. For hypernatremia, consider that many drugs are diluted in 0.9% saline by default, and 0.9% saline is often used for flushes or as slow infusions to "keep vein open." Nurses can monitor sodium trends and work with pharmacists to change diluents to dextrose 5%, which may help reduce risk of sodium overload and hypernatremia.[7]

Apply what you know. When faced with a patient with hyponatremia or hypernatremia, the nurse has an important role in determining the etiology through careful integration of history and physical findings and interpretation of volume status. Understanding the etiology guides the nurse's understanding of the treatment approach and maximizes the role of the nurse in correcting, managing, and preventing disorders of sodium and water balance. ▼

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