Chapter 5

When sodium tips the balance

Just the facts

In this chapter, you’ll learn:

- ways that sodium contributes to fluid and electrolyte balance
- the body’s mechanisms for regulating sodium balance
- causes, signs and symptoms, and treatments associated with sodium imbalances
- proper care for the patient with a sodium imbalance.

A look at sodium

Sodium is one of the most important elements in the body. It accounts for 90% of extracellular fluid cations (positively charged ions) and is the most abundant solute in extracellular fluid. In the body’s normal state, almost all sodium in the body is found in this fluid.

Why it’s important

The body needs sodium to maintain proper extracellular fluid osmolality (concentration). Sodium attracts fluid and helps preserve the extracellular fluid volume and fluid distribution in the body. It also helps transmit impulses in nerve and muscle fibers and combines with chloride and bicarbonate to regulate acid-base balance. Because the electrolyte compositions of serum and interstitial fluid are essentially equal, sodium concentration in extracellular fluid is measured in serum levels. The normal range for serum sodium level is 135 to 145 mEq/L. As a comparison, the amount of sodium inside a cell is 10 mEq/L (Deglin, Vallerand, & Sanoski, 2013; Smeltzer, Bare, Hinkle, & Cheever, 2010).
How the body regulates sodium

What a person eats and how the intestines absorb it determine a body’s sodium level. Sodium requirements vary according to the individual’s size and age. The minimum daily requirement is 0.5 to 2.7 g; however, a salty diet provides at least 6 g/day. The U.S. Department of Agriculture (2013) has released new guidelines suggesting no one should consume more than 2,300 mg of salt per day. They also suggest that people age 51 years and older and those who are African American or have high blood pressure, diabetes, or chronic kidney disease should restrict salt intake to 1,500 mg daily. One teaspoon of table salt has 2,325 mg of sodium (U.S. Department of Agriculture, 2013).

Kidneys naturally balance the amount of sodium stored in the body for optimal health. When sodium levels are low, kidneys essentially hold on to the sodium. When sodium levels are high, kidneys excrete the excess in urine.

If the kidneys can’t eliminate enough sodium, the sodium starts to accumulate in the bloodstream. Because sodium attracts and holds water, blood volume increases, which in turn increases workload of the heart and contributes to high blood pressure. (See Dietary sources of sodium.)

Dietary sources of sodium

Major dietary sources of sodium include:

• canned soups and vegetables
• cheese
• ketchup
• processed meats
• table salt
• salty snack foods
• seafood
• pickled foods
• seasonings such as monosodium glutamate, seasoned salt, and soy sauce
• baked goods with baking powder and baking soda.

Sodium is also excreted through the gastrointestinal (GI) tract and through the skin in sweat. When you think sodium, you should automatically think water as well—the two are that closely related in the body. The normal range of serum sodium levels reflects this close relationship. If sodium intake suddenly increases, extracellular fluid concentration also rises and vice versa (Smeltzer et al., 2010).

Not too much!

The body makes adjustments when the sodium level rises. Increased serum sodium levels cause the individual to feel thirsty and the posterior pituitary gland to release antidiuretic hormone (ADH). (For more information about ADH, see chapter 1, Balancing fluids.) ADH causes the kidneys to retain water, which dilutes the blood and normalizes serum osmolality.

When sodium levels decrease and serum osmolality decreases, thirst and ADH secretion are suppressed, and the kidneys excrete more water to restore normal osmolality. (See Regulating sodium and water, page 86.)
Regulating sodium and water

This flowchart shows two of the body’s compensatory mechanisms for restoring sodium and water balance.

Aldosterone also regulates extracellular sodium balance via a feedback loop. The adrenal cortex secretes aldosterone, which stimulates the renal tubules to conserve water and sodium when the body’s sodium level is low, thus helping to normalize extracellular fluid sodium levels (George, Majeed, Mackenzie, MacDonald, & Wei, 2013; Smeltzer et al., 2010).

The power of the pump

Normally, extracellular sodium levels are very high compared with intracellular sodium levels. The body contains an active transport mechanism, called the sodium-potassium pump, that helps maintain normal sodium levels. This is how the pump works:

In diffusion (a form of passive transport), a substance moves from an area of higher concentration to one of lower concentration. Sodium ions, normally most abundant outside the cells, tend to diffuse inward, and potassium (K) ions, normally most abundant inside the cells, tend to diffuse outward. To combat this ionic diffusion and maintain normal sodium and potassium levels, the sodium-potassium pump is constantly at work in every cell.

However, moving sodium out of the cell and potassium back in can’t happen without some help. Each ion links with a carrier because it can’t get through the cell wall alone. This movement requires
energy (a form of active transport), which comes from adenosine triphosphate (ATP)—made up of phosphorus, another electrolyte—magnesium, and an enzyme. These substances help move sodium out of the cell and force potassium back into the cell.

The sodium-potassium pump allows the body to carry out its essential functions and helps prevent cellular swelling caused by too many ions inside the cell attracting excessive amounts of water. The pump also creates an electrical charge in the cell from the movement of ions, permitting transmission of neuromuscular impulses. (See Sodium-potassium pump.)

**Sodium-potassium pump**

This illustration shows how the sodium-potassium pump carries ions when their concentrations change.

**Normal placement**

Normally, more sodium (Na) ions exist outside cells than inside. More potassium (K) ions exist inside cells than outside.

**Increased permeability**

Certain stimuli increase the membrane’s permeability. When this occurs, sodium ions diffuse inward; potassium ions diffuse outward.

**Energy source**

The cell links each ion with a carrier molecule that helps the ion return through the cell wall. Energy for the ion’s return trip comes from ATP, magnesium (Mg), and an enzyme commonly found in cells.

**Hyponatremia**

A common electrolyte imbalance, *hyponatremia* is a term that describes a state when sodium concentration in the plasma (outside the cell) is lower than normal. In other words, body fluids are diluted and cells swell from decreased extracellular fluid osmolality. Severe hyponatremia can lead to seizures, coma, and permanent neurologic damage.
How it happens

Normally, the body gets rid of excess water by secreting less ADH; less ADH causes diuresis. For that to happen, the nephrons must be functioning normally, receiving and excreting excess water and reabsorbing sodium.

Hyponatremia develops when this regulatory function goes haywire. Serum sodium levels decrease, and fluid shifts occur. When the blood vessels contain more water and less sodium, fluid moves by osmosis from the extracellular area into the more concentrated intracellular area. With more fluid in the cells and less in the blood vessels, cerebral edema and hypovolemia (fluid volume deficit) can occur. (See Fluid movement in hyponatremia.)

Fluid movement in hyponatremia

This illustration shows fluid movement in hyponatremia. When serum osmolality decreases because of decreased sodium concentration, fluid moves by osmosis from the extracellular area to the intracellular area.
Deplete and dilute

Hyponatremia results from sodium loss, water gain (dilutional hyponatremia), or inadequate sodium intake (depletional hyponatremia). It may be classified according to whether extracellular fluid volume is abnormally decreased (hypovolemic hyponatremia), abnormally increased (hypervolemic hyponatremia), or equal to intracellular fluid volume (isovolemic hyponatremia).

Sodium slips lower

In hypovolemic hyponatremia, both sodium and water levels decrease in the extracellular area, but sodium loss is greater than water loss. Causes may be nonrenal or renal. Nonrenal causes include vomiting, diarrhea, fistulas, gastric suctioning, excessive sweating, cystic fibrosis, burns, and wound drainage. Renal causes include osmotic diuresis, salt-losing nephritis, adrenal insufficiency, and diuretic use.

Diuretics promote sodium loss and volume depletion from the blood vessels, causing the individual to feel thirsty and his kidneys to retain water. Drinking large quantities of water can worsen hyponatremia. Sodium deficits can also become more pronounced if the patient is on a sodium-restricted diet. Diuretics can cause potassium loss (hypokalemia), which is also linked to hyponatremia. (See Drugs associated with hyponatremia.)
Water rises higher

In hypervolemic hyponatremia, both water and sodium levels increase in the extracellular area, but the water gain is more impressive. Serum sodium levels are diluted and edema also occurs. Causes include heart failure, liver failure, nephrotic syndrome, excessive administration of hypotonic I.V. fluids, and hyperaldosteronism.

Water rises alone

In isovolemic hyponatremia, also called dilutional hyponatremia, sodium levels may appear low because too much fluid is in the body. However, these patients have no physical signs of fluid volume excess, and total body sodium remains stable. Causes include glucocorticoid deficiency (causing inadequate fluid filtration by the kidneys), hypothyroidism (causing limited water excretion), and renal failure (Smeltzer et al., 2010).
**Disturbing the balance**

Another cause of isovolemic hyponatremia is syndrome of inappropriate antidiuretic hormone (SIADH) secretion. SIADH causes excessive release of ADH, which causes inappropriate and excessive water retention, thereby disturbing fluid and electrolyte balance. This syndrome is a major cause of low sodium levels. ADH is released when the body doesn’t need it, which results in water retention and sodium excretion. (See *What happens in SIADH*.)

**What happens in SIADH**

This flowchart shows the events that occur in SIADH secretion.

![Flowchart of SIADH events](chart)

SIADH occurs with:
- cancers, especially cancer of the duodenum and pancreas and oat cell carcinoma of the lung
- central nervous system (CNS) disorders, such as trauma, tumors, and stroke
- pulmonary disorders, such as tumors, asthma, and chronic obstructive pulmonary disease
- medications, such as certain oral antidiabetics, chemotherapeutic drugs, psychoactive drugs, diuretics, synthetic hormones, and barbiturates.

**What lies beneath**

The patient is treated for the underlying cause of SIADH and for hyponatremia. For instance, if a tumor caused the syndrome, the patient would receive cancer treatment; if a medication caused it, the drug would be stopped. The low sodium levels are treated with fluid restriction (about 1 qt [1 L]/day) and diuretics, such as furosemide.
For many patients, initial treatment may be simple. The patient is placed on fluid restriction to lower water intake to match the low volume of urine caused by the increased ADH. Serum osmolality then increases, causing the ADH level to balance it. If this treatment is inadequate, then the patient may receive oral urea or be instructed to follow a high-sodium diet to increase the kidneys’ excretion of solutes (water follows). The patient may also receive medications, such as demeclocycline or lithium, to block ADH in the renal tubule. If fluid restriction doesn’t raise the patient’s sodium levels, he may need a hypertonic saline solution.

What to look for

As you look for signs of hyponatremia, remember that they vary from patient to patient. They also vary depending on how quickly the patient’s sodium level drops. If the level drops quickly, the patient will be more symptomatic than if the level drops slowly. Patients with sodium levels above 125 mEq/L may not show signs of hyponatremia—but, again, this depends on how quickly sodium levels drop. Usually, acute initial signs and symptoms of nausea, vomiting, and anorexia begin when the serum sodium levels fall between 115 and 120 mEq/L (Smeltzer et al., 2010).

When signs and symptoms start, they’re primarily neurologic. The patient may complain of a headache or irritability or he may become disoriented. He may experience muscle twitching, tremors, or weakness. Changes in level of consciousness (LOC) may start as a shortened attention span and progress to lethargy or confusion. When sodium levels drop to 110 mEq/L, the patient’s neurologic status deteriorates further (usually due to brain edema), leading to stupor, delirium, psychosis, ataxia, and possibly even coma. He may also develop seizures.

Low show

Patients with hypovolemia may have inelastic skin turgor and dry, cracked mucous membranes. Assessment of vital signs shows a weak, rapid pulse and low blood pressure or orthostatic hypotension. Central venous pressure (CVP), pulmonary artery pressure (PAP), and pulmonary artery
wedge pressure (PAWP) may be decreased.

**High signs**

Patients with hypervolemia (fluid volume excess) may experience edema, hypertension, weight gain, and rapid, bounding pulse. They may also have elevated CVP, PAP, and PAWP (George et al., 2013; Lindner & Funk, 2013; Smeltzer et al., 2010).

**What tests show**

Common diagnostic test results in patients with hyponatremia include:
- serum osmolality less than 280 mOsm/kg (dilute blood)
- serum sodium level less than 135 mEq/L (low sodium level in blood)
- urine specific gravity less than 1.010
- increased urine specific gravity and elevated urine sodium levels (above 20 mEq/L) in patients with SIADH
- elevated hematocrit and plasma protein levels.

**How it’s treated**

Generally, treatment varies with the cause and severity of hyponatremia. For example, the patient with an underlying endocrine disorder may require hormone therapy.

**Milder measures**

Therapy for mild hyponatremia associated with hypervolemia or isovolemia usually consists of restricted fluid intake and possibly oral sodium supplements. If hyponatremia is related to hypovolemia, the patient may receive isotonic I.V. fluids such as normal saline solution to restore volume. High-sodium foods may also be offered.

**Critical steps**

When serum sodium levels fall below 120 mEq/L, treatment in the intensive care unit may include infusion of a hypertonic saline solution (such as 3% or 5% saline) if the patient is symptomatic (seizures, coma). Monitor the patient carefully during the infusion for signs of circulatory overload or worsening neurologic status. A hypertonic saline solution causes water to shift out of cells, which may lead to intravascular volume overload and serious brain damage (osmotic demyelination), especially in the pons.

Fluid volume overload can be fatal if untreated. To prevent fluid overload, a hypertonic sodium chloride solution is infused slowly and in small volumes. Furosemide is usually administered at the same time. Infusions of hypertonic solution (3% to 5%) are only done in the intensive care unit with cardiac monitoring available. Sodium levels should not be raised more than 25 mEq/L in the first 48 hours with the rate not exceeding 1 to 2 mEq/L/hour (Smeltzer et al., 2010).

Hypervolemic patients shouldn’t receive hypertonic sodium chloride solutions, except in rare instances of severe symptomatic hyponatremia. During treatment, monitor serum sodium levels and related diagnostic tests to follow the patient’s progress (George et al., 2013; Lindner & Funk, 2013; Smeltzer et al., 2010).
How you intervene

Watch patients at risk for hyponatremia, including those with heart failure, cancer, or GI disorders with fluid losses. Review your patient’s medications, noting those that are associated with hyponatremia. For patients who develop hyponatremia, you’ll want to take the following actions:

- Monitor and record vital signs, especially blood pressure and pulse, and watch for orthostatic hypotension and tachycardia.
- Accurately measure and record intake and output.
- Weigh the patient daily to monitor the success of fluid restriction.
- Assess skin turgor at least every 8 hours for signs of dehydration.
- Watch for and report extreme changes in serum sodium levels and accompanying serum chloride levels. Also monitor other test results, such as urine specific gravity and serum osmolality.
- Restrict fluid intake as ordered. (Fluid restriction is the primary treatment for dilutional hyponatremia.) Post a sign about fluid restriction in the patient’s room and make sure the staff, the patient, and his family are aware of the restrictions. (See Teaching about hyponatremia and hypernatremia, page 99.)

Teaching points

Teaching about hyponatremia and hypernatremia

When teaching a patient with hyponatremia or hypernatremia, be sure to cover the following topics and then evaluate your patient’s learning:

- explanation of hyponatremia or hypernatremia, including causes and treatment
- importance of increasing or restricting sodium intake, including both dietary sources and over-the-counter medications that contain sodium
- drug therapy and possible adverse effects
- signs and symptoms and when to report them.

- Administer oral sodium supplements, if prescribed, to treat mild hyponatremia. If the practitioner has instructed the patient to increase his intake of dietary sodium, teach him about foods high in sodium.
- For severe hyponatremia, make sure a patent I.V. line is in place, then administer prescribed I.V. isotonic or hypertonic saline solutions. Do so cautiously to avoid inducing hypernatremia, brain injury, or fluid volume overload from an excessive or too rapid infusion. Watch closely for signs of hypervolemia (dyspnea, crackles, engorged neck or hand veins), and report them immediately. Use an infusion pump to ensure that the patient receives only the prescribed volume of fluid.
- Keep the patient safe while he undergoes treatment. Provide a safe environment for a patient who has altered thought processes and reorient him as needed. If seizures are likely, pad the bed’s side
rails and keep suction equipment and an artificial airway handy. (See Documenting hyponatremia or hypernatremia, page 99.)

Chart smart

**Documenting hyponatremia or hypernatremia**

If your patient has hyponatremia or hypernatremia, make sure you document the following information:

- assessment findings (including neurologic status)
- vital signs
- types of seizures, if any
- daily weight
- serum sodium level and other pertinent laboratory test results
- intake and output
- medications given and I.V. therapy implemented
- notification of the practitioner when the patient’s condition changes
- nursing interventions and patient response
- patient compliance with fluid restrictions and dietary changes
- patient teaching provided and patient response to the teaching
- safety measures taken to protect the patient (seizure precautions).

**Hypernatremia**

*Hypernatremia*, a less common problem than hyponatremia, refers to an elevated sodium level in the plasma (blood). Severe hypernatremia can lead to seizures, coma, and permanent neurologic damage.

**How it happens**

Thirst is the body’s main defense against hypernatremia. The hypothalamus (with its osmoreceptors) is the brain’s thirst center. High serum osmolality (increased solute concentrations in the blood) stimulates the hypothalamus and initiates the sensation of thirst.

The drive to respond to thirst is so strong that severe, persistent hypernatremia usually occurs only in people who can’t drink voluntarily, such as infants, confused elderly patients, and immobile or unconscious patients. Hypothalamic disorders, such as a lesion on the hypothalamus, may cause a disturbance of the thirst mechanism, but this condition is rare. When hypernatremia occurs, it usually has a high mortality rate (>50%).
Striving for balance

The body strives to maintain a normal sodium level by secreting ADH from the posterior pituitary gland. This hormone causes water to be retained, which helps to lower serum sodium levels.

The cells also play a role in maintaining sodium balance. When serum osmolality increases because of hypernatremia, fluid moves by osmosis from inside the cell to outside the cell, to balance the concentrations in the two compartments. (For more information, see chapter 1, Balancing fluids.)

As fluid leaves the cells, they become dehydrated and shrink—especially those of the CNS. When this occurs, patients may show signs of neurologic impairment. They may also show signs of hypervolemia (fluid overload) from increased extracellular fluid volume in the blood vessels. (See Fluid movement in hypernatremia.) If the overload is severe enough, subarachnoid hemorrhage may occur.
Fluid movement in hypernatremia

With hypernatremia, the body tries to maintain balance by shifting fluid from the inside of cells to the outside. This illustration shows fluid movement in hypernatremia.

Increased concentration

A water deficit can cause hypernatremia—that is, more sodium relative to water in the body. Excessive intake of sodium can also cause it. Regardless of the cause, body fluids become hypertonic (more concentrated).

Water deficit

A water deficit can occur alone or with a sodium loss (but more water is lost than sodium). In either case, serum sodium levels are elevated. This elevation is more dangerous in debilitated patients and those with deficient water intake.

Insensible water losses of several liters per day can result from fever and heat stroke, with older adults and athletes being equally susceptible. Significant water losses also occur in patients with pulmonary infections, who lose water vapor from the lungs through hyperventilation, and in patients with extensive burns. Vomiting and severe, watery diarrhea are other causes of water loss and subsequent hypernatremia; either can be especially dangerous in children. (See Infants, children, elderly patients, and critically ill at risk.)
Infants, children, elderly patients, and critically ill at risk

Hypernatremia is more common in infants and children for two key reasons:

- They tend to lose more water as a result of diarrhea, vomiting, inadequate fluid intake, and fever.
- Their intake of water is generally inadequate because they lack access to fluids and can’t readily communicate their needs.

Elderly patients are also at increased risk for hypernatremia. They may have an impaired thirst response, or they may have limited access to water because of confusion, immobility, or a debilitating illness.

Critically ill patients who are unconscious, intubated, or sedated are at risk because they cannot express thirst or communicate they are thirsty (Lindner & Funk, 2013).

Patients with hyperosmolar hyperglycemic nonketotic syndrome can also develop hypernatremia due to severe water losses from osmotic diuresis. Urea diuresis, another cause of hypernatremia, occurs with administration of high-protein feedings or high-protein diets without adequate water supplementation (George et al., 2013; Lindner & Funk, 2013; Smeltzer et al., 2010).

Thirst to an extreme

Patients with diabetes insipidus have extreme thirst and enormous urinary losses, in many cases, more than 4 gal (15 L)/day. Usually, they can drink enough fluids to match the urinary losses; otherwise, severe dehydration and hypernatremia occur. Diabetes insipidus may result from a lack of ADH from the brain (central diabetes insipidus) or a lack of response from the kidneys to ADH (nephrogenic diabetes insipidus) (George et al., 2013; Lindner & Funk, 2013; Smeltzer et al., 2010).

Central diabetes insipidus may be caused by a tumor or head trauma (injury or surgery), or it may be idiopathic (no known cause). It responds well to vasopressin (another name for ADH). On the other hand, nephrogenic diabetes insipidus doesn’t respond well to vasopressin and is more likely to occur with an electrolyte imbalance such as hypokalemia or with certain medications such as lithium.

Excessive sodium intake

Like water losses from the body, sodium gains can cause hypernatremia. Several factors can contribute to a high sodium level, including salt tablets, high-sodium foods, and medications such as sodium polystyrene sulfonate (Kayexalate). Excessive parenteral administration of sodium solutions, such as hypertonic saline solutions or sodium bicarbonate preparations, and gastric or enteral tube feedings can also cause hypernatremia. (See Drugs associated with hypernatremia.)
### Drugs associated with hypernatremia

The drugs listed below can cause high sodium levels. Ask your patient if any of these medications are part of his drug therapy:

- antacids with sodium bicarbonate
- antibiotics such as ticarcillin disodium-clavulanate potassium (Timentin)
- salt tablets
- sodium bicarbonate injections (such as those given during cardiac arrest)
- I.V. sodium chloride preparations
- sodium polystyrene sulfonate (Kayexalate)
- corticosteroids

(Deuling et al., 2013; Institute for Safe Medicine Practices, 2012)

Other causes of increased sodium levels include inadvertent introduction of hypertonic saline solution into maternal circulation during therapeutic abortion and near drowning in salt water. Excessive amounts of adrenocortical hormones (as in Cushing’s syndrome and hyperaldosteronism) also affect water and sodium balance.

### What to look for

The most important signs of hypernatremia are neurologic because fluid shifts have a significant effect on brain cells. The hyperosmolarity causes a shift of free water from the intracellular to the extracellular space, leading to brain cell shrinkage. Vascular rupture can occur with permanent neurologic deficits if severe. Remember, the body can tolerate a high sodium level that develops over time better than one that occurs rapidly. Early signs and symptoms of hypernatremia may include restlessness or agitation, anorexia, nausea, and vomiting. These may be followed by weakness, lethargy, confusion, stupor, seizures, and coma. Neuromuscular signs also commonly occur, including twitching, hyperreflexia, ataxia, and tremors.

### That flushed, fevered feeling

You may also observe a low-grade fever and flushed skin. The patient may complain of intense thirst from stimulation of the hypothalamus from increased osmolality.

Other signs and symptoms vary depending on the cause of the high sodium levels. If a sodium gain has occurred, fluid may be drawn into the blood vessels and the patient will appear hypervolemic, with an elevated blood pressure, bounding pulse, and dyspnea.

If water loss occurs, fluid leaves the blood vessels and you’ll notice signs of hypovolemia, such as dry mucous membranes, oliguria, and orthostatic hypotension (blood pressure drop and heart rate increase with position changes).

### What tests show

Now that you know how hypernatremia progresses, you can better understand how it causes these
common diagnostic findings:
• serum sodium level greater than 145 mEq/L
• urine specific gravity greater than 1.030 (except in diabetes insipidus, where urine specific gravity is decreased)
• serum osmolality greater than 300 mOsm/kg.

Memory jogger
To remember some common signs and symptoms of hypernatremia, think of the word SALT:
Skin flushed
Agitation
Low-grade fever
Thirst.

How it’s treated
Treatment for hypernatremia varies with the cause. The underlying disorder must be corrected, and serum sodium levels and related diagnostic tests must be monitored. If too little water in the body is causing the hypernatremia, treatment may include oral fluid replacement. Note that the fluids should be given gradually over 48 hours to avoid shifting water into brain cells.

Brain drain
Remember, as sodium levels in the blood vessels rise, fluid shifts out of the cells—including the brain cells—to dilute the blood and equalize concentrations. If too much water is introduced into the body too quickly, water moves into brain cells and they get bigger, causing cerebral edema.

If the patient can’t drink enough fluids, it will be necessary to provide I.V. fluid replacement. A patient may receive salt-free solutions (such as dextrose 5% in water) to return serum sodium levels to normal, followed by infusion of half-normal saline solution to prevent hyponatremia and cerebral edema. Other treatments include restricting sodium intake and administering diuretics along with oral or I.V. fluid replacement to increase sodium loss.
Treatment for diabetes insipidus may include vasopressin, hypotonic I.V. fluids, and thiazide diuretics to decrease free water loss from the kidneys. The underlying cause should also be treated.

How you intervene

Try to prevent hypernatremia in high-risk patients (such as those recovering from surgery near the pituitary gland) by observing them closely. Also, find out if the patient is taking medications that may cause hypernatremia. If your patient does develop hypernatremia, take the following measures:

• Monitor and record vital signs, especially blood pressure and pulse.
• If the patient needs I.V. fluid replacement, monitor fluid delivery and his response to the therapy. Watch for signs of cerebral edema and check his neurologic status frequently. Report any deterioration in LOC.
• Carefully measure and record intake and output. Weigh the patient daily to check for body fluid loss. (See Documenting hyponatremia or hypernatremia.)
• Assess skin and mucous membranes for signs of breakdown and infection as well as water loss from perspiration.
• Monitor the patient’s serum sodium level and report any increase. Monitor urine specific gravity and other laboratory test results.
• If the patient can’t take oral fluids, recommend the I.V. route to the practitioner. If the patient can drink and is alert and responsible, involve him in his treatment. Give him a target amount of fluid to drink each shift, mark cups with the volume they hold, leave fluids within easy reach, and provide paper and pen to record amounts. If family members want to help the patient drink, give them specific instructions as well. (See Teaching about hyponatremia and hypernatremia.)
• Insert and maintain a patent I.V. as ordered. Use an infusion pump to control delivery of I.V. fluids to prevent cerebral edema.
• Assist with oral hygiene. Lubricate the patient’s lips frequently with a water-based lubricant and provide mouthwash or gargle if he’s alert. Good mouth care helps keep mucous membranes moist and decreases mouth odor.
• Provide a safe environment for confused or agitated patients. If seizures are likely, pad the bed’s side rails and keep an artificial airway and suction equipment handy. Reorient the patient as needed, and reduce environmental stimuli (Smeltzer et al., 2010).
Sodium imbalances review

**Sodium basics**
- Major cation in extracellular fluid (90%)
- Attracts fluids
- Helps transmit impulses in nerve and muscle fibers
- Combines with chloride and bicarbonate to regulate acid-base balance
- Normal serum level: 135 to 145 mEq/L

**Sodium balance**
- Balance is maintained by ADH, which is secreted from the posterior pituitary gland.
- The balance depends on what’s eaten and how sodium is absorbed in the intestines.
- Increased sodium intake results in increased extracellular fluid volume.
- Decreased sodium intake results in decreased extracellular fluid volume.
- Increased sodium levels result in increased thirst, release of ADH, retention of water by the kidneys, and dilution of blood.
- Decreased sodium levels result in suppressed thirst, suppressed ADH secretion, excretion of water by the kidneys, and secretion of aldosterone to conserve sodium.
- Balance is maintained by diffusion, which moves sodium ions into cells and potassium out.
- Sodium-potassium pump uses energy to move sodium ions back out of cells and return potassium to cells; it also creates an electrical charge within the cell from the movement of ions, allowing transmission of nerve impulses.

**Hyponatremia**
- Common electrolyte imbalance
- Caused by an inadequate sodium intake, excessive water loss, or water gain
- Serum sodium level less than 135 mEq/L
- Varied signs and symptoms, depending on the individual
- Results from decreased serum osmolality
- Fluid shifts into intracellular areas: neurologic symptoms are related to cerebral edema
- May cause stupor and coma if serum sodium level drops to 110 mEq/L

**Types**
- *Hypovolemic*—both sodium and water are decreased in extracellular area, but sodium loss is greater than water loss
- *Hypervolemic*—both sodium and water are increased in extracellular area, but water gain is more than sodium gain
- *Isovolemic*—water increases, but total sodium levels remain stable; may also be caused by SIADH

**Signs and symptoms**
• Abdominal cramps
• Lethargy and confusion (altered LOC)
• Headache
• Muscle twitching
• Nausea and vomiting
• Anorexia

**Signs and symptoms with depletional hyponatremia**
• Dry mucous membranes
• Orthostatic hypotension
• Poor skin turgor
• Tachycardia

**Signs and symptoms with dilutional hyponatremia**
• Hypertension
• Rapid, bounding pulse
• Weight gain

**Hyponatremia**
• Caused by water loss, inadequate water intake (rarely from failure of the thirst mechanism), excessive sodium intake, or diabetes insipidus
• Patients at increased risk: infants, elderly, immobile and comatose patients
• Always results in increased serum osmolality
• Fluid shifts out of the cells, causing cells to shrink
• Must be corrected slowly to prevent a rapid shift of water back into the cells, which could cause cerebral edema

**Signs and symptoms**
• Agitation
• Confusion
• Flushed skin
• Lethargy
• Low-grade fever
• Thirst
• Restlessness
• Muscle twitching
• Weakness

**Signs and symptoms of hypervolemia with sodium gain**
• Bounding pulse
• Dyspnea
• Hypertension

**Signs and symptoms of hypervolemia with water loss**
• Dry mucous membranes
• Oliguria
Quick quiz

1. In addition to its responsibility for fluid balance, sodium is also responsible for:
   A. good eyesight and vitamin balance.
   B. bone structure.
   C. impulse transmission.
   D. muscle mass.

   **Answer:** C. Besides its role as the main extracellular cation responsible for regulating fluid balance in the body, sodium is also involved in impulse transmission in nerve and muscle fibers.

2. Signs and symptoms of hyponatremia include:
   A. change in LOC, abdominal cramps, and muscle twitching.
   B. headache, rapid breathing, and high energy level.
   C. chest pain, fever, and pericardial rub.
   D. weight loss, slow pulse, and vision changes.

   **Answer:** A. The signs and symptoms of hyponatremia include change in LOC, abdominal cramps, and muscle twitching. A patient with hyponatremia may also exhibit headache, nausea, coma, blood pressure changes, and tachycardia.

3. The minimum daily requirement of sodium for an average adult is:
   A. 2 g.
   B. 4 g.
   C. 5 g.
   D. 8 g.

   **Answer:** A. Although the minimum daily requirement is 2 g, most people consume more than 6 g/day.

4. Increased serum sodium levels cause thirst and the release of:
   A. potassium into the cells.
   B. fluid into the interstitium.
   C. ADH into the bloodstream.
   D. aldosterone into the kidneys.

   **Answer:** C. Higher blood sodium levels prompt the release of ADH from the posterior pituitary gland.

5. The sodium-potassium pump transports sodium ions:
   A. into cells.
B. out of cells.
C. into and out of cells in equal amounts.
D. into skeletal muscles.

**Answer:** B. Normally most abundant outside of cells, sodium tends to diffuse inward. The sodium-potassium pump returns sodium to the extracellular area. Potassium ions tend to diffuse out of the cells and require transport back into cells.

6. You’re teaching a patient with hypernatremia that he needs to restrict daily intake of sodium. Which foods high in sodium should you tell him to avoid?
   A. Bananas, peaches, and broccoli
   B. Red meat, chicken, and pork
   C. Milk, nuts, and liver
   D. Canned soups, ketchup, and cheese

**Answer:** D. Major dietary sources of sodium include canned soups and vegetables, cheese, ketchup, processed meats, table salt, salty snack foods, and seafood.

7. Which of the following disorders causes isovolemic hyponatremia?
   A. Hyperthyroidism
   B. SIADH
   C. Heart failure
   D. Dementia

**Answer:** B. Causes of isovolemic hyponatremia include glucocorticoid deficiency, hypothyroidism, renal failure, and SIADH.

8. Drugs that may cause high sodium levels include:
   A. antacids.
   B. diuretics.
   C. antipsychotics.
   D. antidepressants.

**Answer:** A. If taken on a regular basis, antacids with sodium bicarbonate may cause high sodium levels.

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**Scoring**

🌟🌟🌟 If you answered all eight questions correctly, congratulations! You’re a Sodium Somebody!
🌟🌟 If you answered six or seven correctly, good job. You’re a pillar of strength and intelligence (and not salt)!
🌟 If you answered fewer than six correctly, don’t fret. You’ll strike the proper balance in the following chapters!
References


