

Hypernatremia

Sodium level is high because the body has too little water compared to salt.

THE STORY IN ONE SENTENCE

Sodium level is high because the body has too little water compared to salt.

“High sodium (Hypernatremia) means not enough water for the amount of sodium in your blood. Brain cells shrink because water is pulled out of them.”

Normal anatomy — the body's job

Your body keeps the balance between water and sodium in a narrow range by using thirst and a hormone called ADH (antidiuretic hormone). The hypothalamus (a control center at the base of your brain) senses when sodium is rising, triggers thirst so you drink, and makes ADH. ADH is stored and released by the pituitary gland (a pea-sized gland just below the hypothalamus) and travels through your blood to your kidneys. There, ADH tells the collecting ducts (the final tubes in each kidney that decide how much water leaves in urine) to open tiny water gates called aquaporin-2 channels, so water is pulled back into your blood instead of leaving as pee. This keeps sodium from climbing. Healthy people with access to water rarely develop high sodium because these systems work automatically and powerfully.

BODY'S JOB

The hypothalamus constantly checks the concentration of sodium in your blood. If sodium rises even a little, the hypothalamus makes you feel thirsty and releases ADH. ADH travels to your kidneys and inserts aquaporin-2 water channels into the walls of the collecting ducts, like opening thousands of tiny doors. Water flows back into your bloodstream, your urine becomes concentrated and dark, and sodium level drops back to normal. This cycle runs every minute of every day to keep your blood sodium between 135 and 145 milliequivalents per liter (mEq/L). Without this system, even normal daily water loss through breathing, sweating, and urine would quickly concentrate your blood and damage your cells.

1. Blood sodium rises slightly above 145 mEq/L.
2. Osmoreceptors (special sodium-sensing nerve cells) in the hypothalamus detect the change within seconds.

3. The hypothalamus sends a thirst signal to your conscious brain (you feel thirsty and want to drink) and releases ADH into the bloodstream.
4. ADH reaches the kidneys and binds to receptors on the collecting duct cells.
5. Aquaporin-2 water channels move into the cell walls of the collecting ducts, opening passages for water.
6. Water is pulled from the urine back into the blood, urine volume drops, and urine becomes more concentrated.
7. Blood sodium falls back into the normal range, the hypothalamus stops releasing ADH, and thirst fades.
8. This loop repeats constantly to keep sodium stable despite changes in what you eat, drink, or sweat out.

What breaks

High sodium (Hypernatremia) happens when water leaves your body faster than it comes in, or when you cannot drink enough to keep up with normal losses, or rarely when too much sodium is added. The most common causes are losing water (through diarrhea, vomiting, sweating, burns, or kidneys that cannot hold onto water) and not being able to replace it (because of broken thirst, no access to water, or being too sick or confused to drink). In older adults and people with dementia, the thirst signal is often blunted or ignored. In babies, thirst cannot be communicated. In diabetes insipidus—either central (the hypothalamus or pituitary stops making or releasing ADH) or nephrogenic (the kidneys ignore ADH)—the kidneys flush out huge volumes of dilute urine no matter how high the sodium climbs, and unless the person drinks enormous amounts of water constantly, sodium rises. Burns and severe diarrhea cause large water losses through the skin or stool. Rarely, someone drinks seawater, is given a wrongly concentrated sodium IV solution, or takes in baking soda or salt tablets in huge amounts, directly adding sodium. Once water is lost or sodium is added, the concentration of sodium in the blood climbs above 145 mEq/L.

CLINICAL TERM

High sodium (Hypernatremia) is defined as serum sodium above 145 mEq/L and represents a state of hyperosmolality in which there is insufficient free water relative to total body sodium. Causes are classified by volume status: hypovolemic high sodium (pure water loss exceeds sodium loss—seen in diabetes insipidus, osmotic diuresis, diarrhea, vomiting, burns, or sweating), euvoletic high sodium (pure water loss with normal total body sodium—seen in hypodipsic high sodium or insensible losses without adequate intake), and hypervolemic high sodium (sodium gain exceeds water gain—seen in hypertonic saline administration, sodium bicarbonate, or saltwater ingestion). Central diabetes insipidus results from inadequate ADH secretion due to damage to the hypothalamus or posterior pituitary (from trauma, tumor, surgery, or infiltration). Nephrogenic diabetes insipidus results from renal resistance to ADH (caused by lithium, high calcium (hypercalcemia), low potassium (hypokalemia), chronic kidney disease, or genetic mutations in the ADH receptor or aquaporin-2 channel). Hypodipsia (impaired thirst) is common in elderly patients, those with dementia, stroke affecting the hypothalamus, or intubated and sedated ICU patients who cannot access water. The break does not stay local: when extracellular sodium rises, water shifts out of cells down the osmotic gradient, leading to intracellular dehydration and cell shrinkage, most critically in the brain.

How the body compensates

When sodium rises, the body launches immediate protective responses: the hypothalamus triggers thirst and releases ADH to conserve water, and brain cells begin making organic osmolytes (small protective particles like taurine, glutamine, and myoinositol) within hours to days to pull water back into shrunken cells and prevent further collapse. These adaptations can keep a person looking fairly stable even as sodium climbs into the 150s or 160s, which makes it easy to miss how much danger the patient is in. The adaptations also create a trap: if you fix the sodium too fast by giving free water or hypotonic fluids, the blood sodium drops quickly but the brain's protective osmolytes are still inside the cells, so water rushes in too fast, the brain swells inside the rigid skull, and seizures, cerebral swelling (edema), and death can follow. The key clinical rule is to correct chronic high sodium (hypernatremia) (present more than 48 hours) slowly—no faster than 10–12 mEq/L per 24 hours, and ideally 6–8 mEq/L per 24 hours—to give the brain time to dismantle its protective particles as the sodium falls. Acute high sodium (under 48 hours) can be corrected faster because the brain has not yet adapted, but even then the rate should not exceed 1–2 mEq/L per hour. Watch closely for signs the brain is decompensating: confusion, seizures, lethargy, or focal neurologic deficits. Serial sodium

measurements every 2–4 hours during correction are standard. The danger zone is not just the high sodium itself, but the speed of change in either direction.

1. {'step': 'The hypothalamus releases ADH and triggers thirst immediately when sodium rises.', 'why': 'These are the body's first-line defenses to bring in water and stop the kidneys from losing more. If the person can drink and the kidneys respond, sodium will stabilize or fall without treatment.'}
2. {'step': 'Urine becomes concentrated and low in volume as the kidneys hold onto water.', 'why': 'ADH opens aquaporin-2 channels in the collecting ducts, so water is reabsorbed. Urine osmolality rises above 600–800 mOsm/kg, and urine sodium is typically low (under 25 mEq/L) unless the kidney itself is the problem (as in diabetes insipidus or salt-wasting).')}
3. {'step': 'Brain cells shrink as water is pulled out into the bloodstream.', 'why': 'High sodium in the blood creates an osmotic gradient, so water flows out of cells (including neurons and glia) to try to dilute the blood. Cell shrinkage stretches and can tear small bridging veins in the brain, causing intracerebral or subarachnoid bleeding (hemorrhage), especially in infants and older adults.'}
4. {'step': 'Within hours to days, brain cells make organic osmolytes (idiogenic osmoles) inside themselves.', 'why': 'Neurons and glia synthesize and import small particles—taurine, glutamine, glutamate, myoinositol, and glycerophosphorylcholine—that raise the osmolality inside the cell without disrupting protein function. These particles act like tiny magnets to pull water back into the cell from the blood, preventing further shrinkage and protecting the brain from mechanical damage. This is why chronic high sodium (hypernatremia) (over 48 hours) is often better tolerated than acute.'}
5. {'step': 'Vital signs may remain near normal, and the patient may appear stable or only mildly confused.', 'why': 'The brain's adaptive response buys time, so the person does not immediately seize or become unconscious (comatose) even with sodium in the 150s. This stability is deceptive—it hides how much the brain has had to adapt and how dangerous rapid correction will be.'}
6. {'step': 'If you correct sodium too fast, water floods into brain cells faster than the protective osmolytes can be cleared.', 'why': 'The blood sodium drops quickly with IV fluids or free water, but the brain still has high levels of organic osmolytes inside the cells. Water follows the osmotic gradient into the cell, the brain swells, intracranial pressure rises, and seizures or herniation can occur. This is called osmotic demyelination in reverse (cerebral swelling (edema) from overcorrection of high sodium (hypernatremia)) and is the main caused by medical care (iatrogenic) complication to avoid.'}
7. {'step': 'The safe correction rate is no more than 10–12 mEq/L per 24 hours (and ideally 6–8 mEq/L per 24 hours) for chronic high sodium (hypernatremia).', 'why': 'This slow rate gives the brain time to export or break down the organic osmolytes it made, so water entry into cells is gradual and controlled. Faster correction risks cerebral swelling (edema).')}

8. {'step': 'Watch for the crash point: seizures, worsening confusion, focal neurologic deficits, or sudden deterioration during correction.', 'why': 'These are signs that the brain is swelling or herniating from too-fast correction. If they appear, slow or stop free water replacement, check sodium immediately, and consider hypertonic saline to raise sodium slightly and pull water back out of the brain.'}
9. {'step': 'A patient who looks stable but whose sodium is rising or not improving is not truly stable.', 'why': 'Stability maintained by maximal physiologic compensation is fragile. The next stressor—infection, medication change, fluid restriction—can tip the patient into acute decompensation or make correction even riskier.'}

Domino effect

1. Water is lost from the body, or sodium is added, faster than replacement occurs. — Whether from diabetes insipidus dumping urine, diarrhea, burns weeping fluid, or too much salt given IV, the result is the same: the concentration of sodium in the blood climbs above 145 mEq/L because there is now too little water for the amount of sodium present.
2. Water moves out of cells into the bloodstream. — Water always flows toward higher concentration of particles (osmosis). High sodium in the blood pulls water out of every cell in the body, down the osmotic gradient, trying to dilute the blood back to normal.
3. Brain cells shrink. — Neurons and glial cells lose water and shrivel. The brain physically pulls away from the inside of the skull, stretching and sometimes tearing bridging veins (the small veins that connect the brain surface to the dura). This can cause subdural a pooled collection of blood (hematoma), intracerebral bleeding (hemorrhage), or subarachnoid bleeding, especially in infants and older adults.
4. The brain makes organic osmolytes (idiogenic osmoles) to protect itself. — To fight back against shrinking, brain cells synthesize or import small particles—taurine, glutamine, myoinositol, glutamate, and others—within hours to days. These raise the osmolality inside the cell so that water stops leaving and even flows back in slightly, cushioning the cell and preventing mechanical damage. This adaptation is protective but creates a trap for treatment.
5. If sodium is corrected too fast, water floods into brain cells. — When you give free water or hypotonic fluids, blood sodium falls quickly. But the brain still has high levels of organic osmolytes inside its cells from the adaptation. Now the inside of the cell is more concentrated than the blood, so water rushes into the brain cells down the new gradient, faster than the cell can clear out the osmolytes.
6. The brain swells inside the rigid skull. — The skull is a closed box with no room to expand. As brain cells take in water and swell, intracranial pressure rises. This compresses blood vessels (reducing oxygen delivery), squeezes the brainstem, and can cause herniation (the brain pushes down through the opening at the skull base), which is often fatal.

7. Seizures, altered mental status, coma, or death occur. — Rising intracranial pressure and brain swelling disrupt normal electrical signaling in neurons, triggering seizures. Brainstem compression shuts down breathing and heart rate control. This is why overcorrection of chronic high sodium (hypernatremia) is as dangerous as the high sodium itself.
8. Every step in this chain is the body trying to protect itself, not random damage. — Pulling water out of cells protects blood volume and tries to dilute sodium. Making osmolytes protects the brain from collapsing. Even the final swelling is water following physical laws, not a disease process. The job of the clinician is to understand the chain, correct the upstream break (restore water or remove excess sodium) at a safe speed, and not trigger the downstream disaster (cerebral swelling (edema) from overcorrection).

Symptoms · what the patient feels, and why

- Thirst: When sodium (salt) in the blood climbs too high, special sensors in the brain (the hypothalamus, the body's water-balance control center) notice the blood has become too salty and trigger a powerful thirst drive to make you drink and dilute the blood back down.
- Confusion or trouble thinking clearly: High sodium pulls water out of brain cells (osmosis—water moves from where there's less salt to where there's more salt), shrinking the cells slightly and slowing the electrical signals between them, which makes thinking foggy.
- Muscle weakness or twitching: Muscles need the right balance of sodium and water to contract smoothly; when sodium is too high and cells are dehydrated, the electrical signals that tell muscles to fire become erratic, causing weakness or twitches.
- Irritability or restlessness: Brain cells that regulate mood and behavior are especially sensitive to dehydration; as they shrink, their normal signaling is disrupted, making a person feel cranky, on edge, or unable to settle.
- Seizures: If sodium rises very fast or very high, brain cells shrink so much that the normal brakes on electrical activity fail, letting waves of uncontrolled signals fire across the brain—a seizure.
- Extreme drowsiness or coma: Severe dehydration of brain cells shuts down the arousal centers (the reticular activating system deep in the brainstem, which keeps you awake and aware), causing deep sleepiness or complete loss of consciousness.
- Very little urine output (in some causes): When high sodium (hypernatremia) comes from losing more water than sodium (like sweating heavily or having severe diarrhea without drinking), the kidneys try to save every drop of water by making very concentrated, dark urine—so you pee much less.
- Very large amounts of urine (in diabetes insipidus): In diabetes insipidus, the kidneys can't respond to ADH (antidiuretic hormone, the signal that tells kidneys to hold onto water) or the brain doesn't make enough ADH, so the kidneys dump huge volumes of dilute urine—sometimes many liters a day—leaving the body dehydrated and sodium climbing.

Focused exam

- Severe thirst or no thirst at all: Thirst is the body's alarm that says 'drink now.' In high sodium (hypernatremia), this alarm is either broken (older adults, damage to the hypothalamus — the brain's control center for thirst) or the person cannot respond (sedation, paralysis, no access to water). No thirst means the upstream break is water intake.
- Dry mouth, dry tongue, and sunken eyes: Water left the cells to dilute the salty blood. The mouth and eyes lose water first. This is the body trying to keep enough fluid in the blood vessels so blood pressure does not crash, but it means cells everywhere are shriveling.
- Confusion, lethargy, or restlessness: Water leaving brain cells makes the brain shrink. A shrinking brain pulls on the small blood vessels anchored to the skull. This can tear vessels and cause bleeding. Confusion is the first sign the brain is in danger.
- Muscle weakness or muscle twitching: Water left muscle cells too. Muscles need water to contract and relax smoothly. Weakness or twitching means high sodium (hypernatremia) is affecting the whole body, not just the brain.
- Seizures (especially if sodium > 160 mEq/L or if corrected too fast): The brain made special protective particles called osmolytes to pull water back in. If you fix high sodium (hypernatremia) too fast, water rushes into brain cells faster than the brain can dump those particles. The brain swells. Seizures mean the brain is swelling right now — the most dangerous complication of high sodium or its treatment.
- Urine output — very low (oliguria) or very high (polyuria, > 3 liters per day): Low urine means the kidneys are trying to save water (normal response). High urine means the kidneys cannot hold water — either central diabetes insipidus (the hypothalamus stopped making ADH, the hormone that tells kidneys to hold water) or nephrogenic diabetes insipidus (the kidneys cannot hear ADH). Which one tells you the upstream cause.

Diagnostics · every test answers a question

- Serum sodium (part of basic metabolic panel) → Is sodium concentration in the blood too high, confirming high sodium (hypernatremia)?
- Serum glucose → Is high blood sugar pulling water out of cells and falsely raising sodium?
- Urine osmolality → Are the kidneys concentrating urine properly to save water?
- Urine sodium → Is the body losing salt in the urine or holding it?
- Serum osmolality → Is the blood too concentrated overall (confirming true high sodium (hypernatremia)) or is something else raising osmolality?
- Water deprivation test (if diabetes insipidus suspected) → Can the body concentrate urine when deprived of water, or does it keep dumping dilute urine?

- Medication review → Is a medication causing high sodium (hypernatremia) by blocking ADH or increasing water loss?

Treatment logic

- Give free water by mouth, or give D5W (5% dextrose in water) IV if patient cannot drink → Water enters the bloodstream and dilutes the sodium back down. Water then moves back into shrunken brain and body cells. This reverses the cell shrinkage causing confusion and weakness.
- Correct sodium slowly — drop sodium by no more than 10 mEq/L per 24 hours (goal 0.5 mEq/L per hour maximum) → Gives the brain time to dump the protective osmolytes it made when it was shrinking. Slow correction prevents water from rushing into brain cells too fast, which would cause brain swelling (cerebral swelling (edema)) and seizures.
- For central diabetes insipidus, give desmopressin (DDAVP) — synthetic ADH → DDAVP replaces the missing ADH (antidiuretic hormone normally made by the hypothalamus). It tells the kidneys to hold water instead of dumping it. Water stays in the body, diluting sodium naturally.
- For nephrogenic diabetes insipidus, give thiazide diuretic (hydrochlorothiazide or amiloride) plus low-sodium, low-protein diet → Thiazide diuretics make the kidneys reabsorb more water in the early part of the kidney tubule, even though the kidneys cannot hear ADH. Low sodium and low protein mean fewer particles (solutes) in the urine, so less water is dragged out with them.
- Find and fix the root cause — stop medications causing water loss (lithium, diuretics), treat fever, cover burns, restore access to water, or treat the brain injury affecting thirst → Stops the ongoing water loss or restores the thirst signal. This prevents high sodium (hypernatremia) from coming back after you dilute the sodium.
- Replace ongoing insensible water losses (breathing, sweating, fever) with scheduled water intake or IV fluids → Prevents new high sodium (hypernatremia) from forming as the body continues to lose water through skin, lungs, and normal urine. Keeps water balance neutral while the underlying cause is being fixed.
- Monitor sodium every 2–4 hours during correction to avoid overcorrection → Catches the sodium level if it drops too fast. Allows you to slow or stop free water if sodium is falling faster than the safe 0.5 mEq/L per hour limit.

Medications

Free water (plain water by mouth or D5W intravenously) · Hypotonic fluid replacement

Free water means water without salt. When the blood has too much sodium compared to water, giving plain water (or dextrose 5% in water, which the body uses like plain water) dilutes the blood slowly and gently brings the sodium level back down. The key safety rule is to lower sodium no faster than 10 milliequivalents per liter in 24 hours—ideally around 0.5 per hour—because dropping it too fast can cause the brain to swell dangerously.

1. Plain water enters the bloodstream (if given by vein) or is absorbed from the gut (if given by mouth).
2. The extra water spreads into the blood and then moves into cells that have been shrunk by the high salt.
3. As water enters cells, the sodium concentration in the blood slowly falls toward normal.
4. The kidneys sense the change and adjust urine concentration to keep things balanced.
5. Brain cells—which are very sensitive to fast sodium swings—gradually return to their normal size without swelling too quickly.

SAFETY WATCH

Drop sodium no faster than 10 mEq/L in 24 hours (about 0.5 mEq/L per hour) to avoid cerebral swelling (edema) (brain swelling). · Check sodium levels every 2 to 4 hours at first to make sure correction is on track. · If the patient cannot drink safely, use D5W by vein—never plain water by vein, because that would burst red blood cells. · Keep replacing ongoing losses (urine, sweat, diarrhea) on top of the initial deficit.

Desmopressin (DDAVP) · Synthetic vasopressin analog (antidiuretic hormone replacement)

Desmopressin is a lab-made copy of vasopressin (also called antidiuretic hormone or ADH), the natural signal the brain sends to tell the kidneys to hold onto water. In central diabetes insipidus, the pituitary gland (a pea-sized gland at the base of the brain) does not make enough vasopressin, so the kidneys let huge amounts of dilute urine leave the body. Giving desmopressin replaces the missing hormone and tells the kidneys to pull water back into the blood, which stops the flood of urine and lets sodium levels normalize.

1. Desmopressin enters the bloodstream (given as a nasal spray, pill, or injection).
2. It travels to the kidneys and binds to V2 receptors on the cells of the collecting duct.

3. Binding triggers the cells to insert aquaporin-2 channels (tiny water gates) into the tube wall.
4. Water from the forming urine flows back through these channels into the blood.
5. Urine becomes more concentrated, urine volume drops sharply, and blood sodium gradually falls as the body holds onto water.

SAFETY WATCH

Watch for low sodium (hyponatremia) (sodium dropping too low) if the dose is too high or the patient drinks too much water—check sodium regularly. · Use cautiously in older adults and anyone with heart failure or kidney disease, because holding onto too much water can overload the circulation. · Nasal spray absorption can be unpredictable if the nose is congested or inflamed; oral or injection routes may be more reliable. · Do NOT use if the patient has nephrogenic DI—it will not help and may delay correct treatment.

Thiazide diuretic (e.g., hydrochlorothiazide or chlorthalidone) · Thiazide diuretic

In nephrogenic diabetes insipidus, the kidneys cannot respond to the brain's signal to hold onto water, so they flush out huge amounts of dilute urine. Thiazide diuretics—which usually make people urinate more—work paradoxically here: they lower blood volume slightly, which triggers the kidneys' early filtering tubes (the proximal tubules) to pull more salt and water back into the body before urine even reaches the broken part. This reduces total urine volume and helps the blood hold onto water, gently lowering sodium levels.

1. The thiazide blocks the sodium-chloride cotransporter (NCC) in the distal tubule, so a little extra salt and water leave as urine at first.
2. This mild salt and water loss shrinks blood volume slightly.
3. The body senses the drop and signals the proximal tubule to pull more sodium and water back upstream.
4. Because more water is reabsorbed early, less reaches the broken collecting duct, so total urine volume falls.
5. With less water lost in urine, blood sodium gradually normalizes.

SAFETY WATCH

Monitor potassium closely—thiazides can cause low potassium (hypokalemia) (low potassium), which leads to muscle weakness, cramps, and dangerous heart rhythms. · Check sodium regularly; overdoing it can flip high sodium (hypernatremia) into low sodium (hyponatremia). · Can raise blood sugar and uric acid—watch in patients with diabetes or gout. · May cause dehydration or dizziness, especially when starting; encourage steady fluid intake and slow position changes.

Amiloride · Potassium-sparing diuretic (epithelial sodium channel blocker)

Amiloride is sometimes used in lithium-induced nephrogenic diabetes insipidus. Lithium (a mood-stabilizing drug) enters kidney cells through epithelial sodium channels (ENaC) in the collecting duct and over time damages the cells so they cannot respond to vasopressin, leading to huge urine output and high sodium. Amiloride blocks those channels, preventing lithium from getting into the cells and reducing the damage, which helps the kidneys hold onto water and brings sodium down.

1. Amiloride binds to and blocks the ENaC channels in the collecting duct.
2. Blocking ENaC stops lithium from entering the cells through that pathway.
3. With less lithium inside, the cells suffer less damage and can better respond to any remaining vasopressin signal.
4. The kidneys pull more water back into the blood, urine volume drops, and blood sodium falls toward normal.
5. Amiloride also prevents potassium loss, which is why it is called potassium-sparing.

SAFETY WATCH

Watch for high potassium (hyperkalemia) (potassium rising too high), especially in patients with kidney disease or those taking ACE inhibitors, ARBs, or potassium supplements—high potassium can cause dangerous heart rhythms. · Check potassium and kidney function regularly. · May cause dizziness or headache. · Do NOT combine with other potassium-sparing diuretics or high-dose potassium supplements without close monitoring.

Red flags

- Seizure or sudden collapse—means the brain is in immediate danger from severe cell shrinkage.
- Cannot wake the person or they are deeply confused—sign of life-threatening brain dysfunction.

- Sodium above 160 mEq/L or rising faster than 0.5 mEq/L per hour—rapid or extreme rises can tear small blood vessels in the brain.
- No urine for many hours despite drinking, or urine output suddenly stops—may mean the kidneys are failing.

Nursing priorities

1. Check sodium level and calculate how fast it rose; notify provider immediately if sodium is above 160 mEq/L or patient has altered mental status — Sodium above 160 mEq/L or rapid rise causes brain cells to shrink and bleed; seizures and coma can happen; you must know the baseline to calculate safe correction speed of 10 mEq/L per 24 hours maximum
2. Start seizure precautions: padded bed rails up, suction at bedside, oxygen ready, IV access secured — Shrinking brain cells from high sodium lower the seizure threshold (make seizures more likely); aspiration (breathing in vomit) during a seizure can cause death
3. Give free water by mouth if patient can swallow safely, or start D5W (5% dextrose in water) IV if ordered; recheck sodium every 2 to 4 hours during correction — Free water dilutes the high sodium concentration; D5W provides pure water because body cells use up the sugar quickly and only water enters cells; dropping sodium faster than 0.5 mEq/L per hour causes cerebral swelling (edema) (brain swelling) and death
4. Measure and record all urine output every hour; check urine specific gravity (concentration test) each void — Dilute urine with high output (more than 200 mL per hour) means diabetes insipidus; concentrated urine with low output means dehydration from not enough water intake; this difference determines whether patient needs DDAVP or just fluids
5. Monitor mental status, muscle strength, and deep tendon reflexes every 1 to 2 hours; use a standardized confusion scale — Improving confusion and strength mean sodium is correcting safely; worsening symptoms during treatment mean cerebral swelling (edema) from too-fast correction or continued sodium rise from untreated cause
6. Assess skin turgor (skin tent test on sternum), mucous membranes, and vital signs for signs of volume depletion (dehydration) — Dry mucous membranes, poor skin turgor, low blood pressure, and fast heart rate mean the body has lost total body water; euvoletic (normal fluid volume) patients with pure water loss need different treatment than hypovolemic (low volume) patients
7. Review all IV fluids and tube feedings with pharmacy; ensure patient gets adequate free water separate from nutrition — Hypertonic fluids (fluids with high salt or sugar) and high-protein tube feeds without water flushes are common causes of hospital high sodium (hypernatremia); prevention stops the problem from getting worse

8. If central diabetes insipidus is diagnosed, give DDAVP (desmopressin) as ordered and watch for overcorrection and low sodium (hyponatremia) (low sodium) within 4 to 6 hours — DDAVP replaces antidiuretic hormone and makes kidneys hold water immediately; too much DDAVP causes the opposite problem where sodium drops dangerously low and causes brain swelling

Sources

- KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease
- UpToDate: Hypernatremia
- American Journal of Medicine systematic reviews on dysnatremias
- Cochrane Database of Systematic Reviews

Last reviewed: 2026-07-07