

Hypernatremia

A plain-English guide for you and your family

WHAT IS HAPPENING

Your body tightly controls the balance between water and salt (sodium) in your blood. Two main systems do this job: thirst, which makes you drink when you need water, and a hormone called ADH (made in the hypothalamus at the base of your brain and released by the pituitary gland) that tells your kidneys to hold onto water instead of peeing it out. When sodium climbs too high, you feel thirsty and drink, and ADH kicks in to save water. But older adults, babies, and very sick people often cannot feel thirst clearly or cannot get to water on their own, and their ADH system may not respond as well. When these protective systems fail, you can lose more water than you take in—through sweating, diarrhea, vomiting, burns that weep fluid, or kidneys that dump too much water (as in diabetes insipidus, where the kidneys ignore ADH and flush out huge amounts of dilute urine). Rarely, someone takes in too much salt—like drinking seawater or getting a wrongly mixed IV solution. Once sodium rises, water is pulled out of every cell in your body (including brain cells) into the bloodstream to try to dilute the salt. Brain cells shrink. If this happens slowly over days, the brain protects itself by making tiny particles (organic osmolytes like taurine and glutamine) inside each cell that act like magnets to pull some water back in and keep the cell from collapsing completely. But if you then fix the sodium too fast by giving water or low-salt fluids, the sodium in the blood drops quickly while those protective particles are still sitting inside the brain cells, so water rushes into the brain too fast. The brain swells inside the rigid skull, which has no room to expand, and that causes seizures, coma, or even death. That is why high sodium (hypernatremia) must be corrected slowly and carefully—you are unwinding a chain the body built to protect itself.

What you might feel

- Thirst
- Confusion or trouble thinking clearly
- Muscle weakness or twitching
- Irritability or restlessness
- Seizures

- Extreme drowsiness or coma
- Very little urine output (in some causes)
- Very large amounts of urine (in diabetes insipidus)

Call the doctor right away if...

- 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.

What the care team will do

- 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
- Start seizure precautions: padded bed rails up, suction at bedside, oxygen ready, IV access secured
- 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
- Measure and record all urine output every hour; check urine specific gravity (concentration test) each void
- Monitor mental status, muscle strength, and deep tendon reflexes every 1 to 2 hours; use a standardized confusion scale
- Assess skin turgor (skin tent test on sternum), mucous membranes, and vital signs for signs of volume depletion (dehydration)
- Review all IV fluids and tube feedings with pharmacy; ensure patient gets adequate free water separate from nutrition
- 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

Medicines you may be given — and why

Free water (plain water by mouth or D5W intravenously)

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.

Desmopressin (DDAVP)

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.

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

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.

Amiloride

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.

Where this information comes from

- 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

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