

Central DI

Central DI — the brain stops sending the 'hold water' signal, so the kidneys flush out gallons of dilute urine and the patient must drink constantly or sodium will spike and cause confusion, seizures, or coma.

THE ONE-LINE TRUTH

The back part of the pituitary gland stops releasing enough ADH (the hormone that tells your kidneys to save water), so the kidneys dump huge amounts of pale, watery urine and the body gets dangerously thirsty.

The chain — how it unfolds

1. ADH production or release stops because the magnocellular neurons in the hypothalamus or the pituitary stalk are damaged or destroyed. — why: Without ADH traveling through the blood to the kidneys, the collecting ducts never get the signal to insert aquaporin-2 water channels into their walls, so water that should return to the blood is dumped into the bladder and lost as urine.
2. Urine output jumps suddenly and dramatically to three to twenty liters per day (polyuria), and the urine looks clear, pale, and watery with a specific gravity under 1.005 and osmolality under 200 milliosmoles per kilogram (normal concentrated urine is 600 to 1200). — why: The kidney filters about 180 liters of blood every day and normally pulls almost all the water back, leaving one to two liters as urine. Without ADH, the kidney cannot pull water back in the collecting ducts, so nearly all the filtered water leaves the body, flooding the bladder every hour or two.
3. Massive water loss shrinks the blood volume and makes the blood more concentrated, raising plasma sodium above 145 milliequivalents per liter and plasma osmolality above 295 milliosmoles per kilogram. — why: Water leaves the bloodstream much faster than sodium does (the kidneys still hold onto sodium through other hormones like aldosterone), so the ratio of sodium to water climbs. Blood becomes saltier and thicker.

4. Rising blood osmolality triggers osmoreceptors in the hypothalamus, which fire signals that create intense, unrelenting thirst (polydipsia), and the person drinks constantly—often preferring ice-cold water or ice chips. — why: The hypothalamus detects the high sodium and turns on the thirst drive to pull water back into the body from the outside and dilute the blood back to normal. Thirst in central DI is often described as more intense and specific than ordinary thirst.
5. If the person can drink freely and has a normal thirst mechanism, sodium may stay near normal or only mildly elevated (145 to 150 milliequivalents per liter), but they are trapped in an exhausting cycle of urinating and drinking around the clock, often every one to two hours, day and night. — why: Water intake roughly matches water output (input equals output), so the body stays in a precarious, fragile balance. The person cannot leave the house for long, cannot sleep through the night, and must always be near a bathroom and a water source.
6. If water intake stops or falls behind output—during sleep, anesthesia, surgery, nausea, vomiting, loss of consciousness, or in infants, young children, and debilitated adults who cannot ask for or reach water—serum sodium climbs rapidly, often spiking from 145 to 160 milliequivalents per liter or higher within hours to one day. — why: Urine output does not slow down at all (there is still no ADH), so water keeps pouring out at several hundred milliliters per hour while intake drops to zero. Blood sodium rises at a rate of 1 to 2 milliequivalents per liter per hour, much faster than the brain can adapt.
7. Severe high sodium (hypernatremia) (sodium above 155 to 160 milliequivalents per liter) shrinks brain cells by pulling water out of them, stretches and tears bridging veins between the brain and skull, and disrupts electrical signaling in neurons, causing early symptoms of irritability, restlessness, and intense thirst, then confusion, lethargy, muscle twitching, weakness, fever, seizures, and coma. If untreated or corrected too fast, permanent brain injury or death can occur. — why: Brain cells cannot tolerate sudden dehydration. The mechanical stress of shrinking tears blood vessels (causing subdural a pooled collection of blood (hematoma) or subarachnoid bleeding (hemorrhage)), and the high sodium disrupts the electrical gradients neurons need to fire properly. If sodium is corrected too quickly (faster than 10 to 12 milliequivalents per liter per day), brain cells swell rapidly as water rushes back in, causing cerebral swelling (edema), brainstem herniation, and death.

8. Once desmopressin (DDAVP, a synthetic analog of ADH) is given—usually as a nasal spray, sublingual tablet, or subcutaneous injection—the kidneys immediately start holding water again. Within one to two hours, urine output drops from liters per hour to normal (50 to 100 milliliters per hour), urine becomes concentrated and yellow, and serum sodium drifts back toward normal over 24 to 48 hours. Close monitoring with frequent sodium checks (every 4 to 6 hours initially) is essential to avoid overcorrection and caused by medical care (iatrogenic) low sodium (hyponatremia). — why: DDAVP replaces the missing ADH signal by binding to the same V2 receptors in the kidney that natural ADH uses, restoring the kidney's ability to insert aquaporin-2 channels and concentrate urine. Careful dosing (starting low, often 0.05 to 0.1 milligrams DDAVP by mouth or 1 to 2 micrograms subcutaneously) and fluid balance (matching intake to output, not forcing fluids) prevent the sodium from swinging from too high to dangerously low. Patients often need one to two doses per day for life, adjusted based on urine output, thirst, and sodium levels.

Spot it — what you see, and why

- Making huge amounts of urine every day (often 3–20 liters, which is like 3–20 big soda bottles): The brain normally makes a hormone called ADH (anti-diuretic hormone, or the 'save water' signal) in a tiny region called the hypothalamus and stores it in the pituitary gland (a pea-sized gland at the base of the brain). ADH travels through the blood to the kidneys and tells them to hold on to water. In central DI, the hypothalamus or pituitary is damaged by surgery, a tumor, head injury, infection, or sometimes for no clear reason, so the brain cannot make or release enough ADH. Without this signal, the kidneys never get the command to stop water from pouring out, and the body loses water like a leaky bucket.
- Constant, strong thirst and drinking water all day and night: When you lose so much water in the urine, your blood becomes more concentrated (like juice concentrate before you add water). Sensors in the hypothalamus (the brain's thirst center) notice this change and trigger a powerful thirst signal so you will drink and replace the lost water. People who are awake and can drink usually do this automatically and stay safe, but babies, very sick people, or anyone who cannot drink enough can become dangerously dehydrated.
- Getting up many times at night to pee and drink (nocturia): Because the kidneys keep making huge amounts of urine around the clock—even at night—the bladder fills up over and over, waking the person to empty it and then to drink more water to keep up with the loss. This cycle can happen four, five, or more times every night.
- Urine that looks almost clear or very pale, like water: Normal urine is yellow because the kidneys concentrate waste into a smaller amount of water. In central DI, the kidneys are flushing out huge volumes of water that are barely concentrated at all, so the urine is very dilute and pale—its specific gravity (a measure of how concentrated urine is) is usually very low, under 1.005.

- Feeling tired, weak, or foggy: Constantly losing water and having to drink and pee day and night interrupts sleep and wears the body down. Also, if the thirst response cannot keep up—or if someone cannot drink freely—the blood can become too concentrated, which slows down how cells work and makes the person feel sluggish and confused.
- Dry mouth and dry skin: When the body is losing more water than it is taking in, the skin and mucous membranes (the moist linings of the mouth and lips) lose moisture, like a plant that isn't watered enough starts to wilt. This is an early sign that the body is getting behind on replacing fluids.

Red flags — act now

- Making huge amounts of urine right after brain surgery, a head injury, or while very sick—this can mean the hypothalamus or pituitary gland is damaged and the brain's water-control system needs fast treatment.
- Confusion, extreme sleepiness, seizures, or muscle twitching along with massive urination—this means the blood sodium may be climbing dangerously high (hypernatremia) because the person cannot drink enough to keep up.
- A baby or young child who is always thirsty, soaking many diapers with clear urine, irritable, and not growing well—central DI in infants is easy to miss and can cause serious dehydration and brain damage very quickly.
- Sudden onset of uncontrollable thirst and urination in someone with a known pituitary tumor, a history of brain infection (such as meningitis or encephalitis), or autoimmune inflammation of the pituitary—this may signal new or worsening pituitary damage.
- Signs of severe dehydration: sunken eyes, dry lips and tongue, skin that stays pinched when you pull it up (tenting), very little or no tears when crying, or feeling dizzy and faint—this means the body is critically low on water and needs emergency fluids.

Treat it — in this order

1. DDAVP (desmopressin) — nasal spray, tablet under the tongue (sublingual), oral tablet, or IV/subcutaneous injection → Replaces the missing ADH signal, so the kidneys start holding onto water again instead of dumping it. Urine output drops from over 3 liters back down to normal (1-2 liters per day), and extreme thirst goes away. Blood sodium stabilizes in a safe range. DDAVP is a synthetic version of ADH that lasts longer and is easier to dose — typical starting doses are 5-10 mcg nasal spray, 0.1-0.2 mg oral, or 1-2 mcg IV/subcutaneous.

2. Let the patient drink freely when thirsty — do not force fluids or restrict them → An awake patient's thirst tells them exactly how much water their body is losing, so they drink just enough to stay balanced. The brain's thirst center (in the hypothalamus) is very precise — it tracks blood salt level and tells the patient to drink when sodium climbs. This prevents both dehydration (from not drinking enough) and water overload (from drinking too much).
3. Check blood sodium every 6–12 hours in the first few days, then every 1–2 weeks once stable → Catches high sodium (from dehydration or not enough DDAVP) or low sodium (from too much DDAVP or giving too much free water through an IV) before the patient has seizures or brain swelling. Sodium swings can happen fast in the first days after surgery or injury, so frequent checks keep you ahead of trouble.
4. Find and treat the underlying cause — MRI to look for tumors, infection, or inflammation; follow up after pituitary surgery; treat autoimmune hypophysitis with steroids if present → If a tumor is pressing on the hypothalamus or pituitary, removing it can relieve the pressure and let the nerve cells heal. If inflammation (hypophysitis) is damaging the gland, steroids (like prednisone) can stop the attack. In some cases, ADH production can restart on its own over weeks to months after the cause is treated.
5. Avoid giving hypotonic (low-salt) IV fluids like half-normal saline (0.45% NaCl) or D5W (5% dextrose in water) in patients taking DDAVP — use normal saline (0.9% NaCl) or let them drink free water instead → Prevents the kidneys from diluting the blood dangerously while DDAVP is forcing them to hold onto water. Hypotonic fluids flood the body with free water when the kidneys can't get rid of it, causing low sodium (hyponatremia below 135 mEq/L), which can lead to seizures or brain swelling. Normal saline matches the blood's salt, so it doesn't dilute anything.

Your top priorities at the bedside

1. Check blood sodium level (and serum osmolality if available) immediately and then every 6–12 hours until stable, watching for values above 145 mEq/L. — why: Without ADH, the kidneys dump free water and blood sodium climbs fast (hypernatremia). Sodium above 150 mEq/L shrinks brain cells, causing confusion, seizures, coma, and bleeding inside the brain. Early detection lets us replace water and give DDAVP before dangerous levels occur.
2. Measure and record every drop of urine output and all fluid intake every hour, using a urometer or measuring container at the bedside. — why: Central DI can cause 500–1000 mL of urine per hour. Tracking input and output shows whether DDAVP is working (urine volume should drop) and whether the patient is drinking enough to match losses. Large negative fluid balance signals worsening dehydration.

3. Give DDAVP (desmopressin) exactly as ordered—nasal spray, sublingual tablet, or IV injection—and watch urine output drop within 1-2 hours. — why: DDAVP replaces the missing ADH, telling the kidneys to hold onto water. Urine volume should fall and urine concentration (specific gravity or osmolality) should rise. If output stays high, the dose may be too low or the diagnosis may be nephrogenic DI (kidney does not respond to ADH).