Hypernatremia

$Na^+ > 145$ · Almost always a water problem, not a salt problem

≤ 10–12 mEq / 24 h
if chronic

WHY IT HAPPENS *thirst normally protects you — so ask why it did not*

WATER LOSS

Renal: diabetes insipidus, **osmotic diuresis** (hyperglycemia, mannitol, high-protein feeds, post-obstructive or recovery-phase polyuria. **Extrarenal:** fever, burns, ventilation, sweating, and **osmotic diarrhea** including lactulose.

NO ACCESS TO WATER — the commonest hospital cause

Intubated, sedated, or delirious patients cannot ask for water. Also impaired thirst in the elderly, dementia, and anyone NPO without maintenance free water. **This is usually iatrogenic and preventable.**

SODIUM GAIN

Hypertonic saline, sodium bicarbonate boluses, hypertonic feeds, and salt-water ingestion. Less common, and usually accompanied by volume overload rather than depletion.

CALCULATE THE DEFICIT *then add ongoing and insensible losses*

FREE WATER DEFICIT

Deficit (L) = TBW × ($[Na^+]$ / 140 − 1)
TBW = weight (kg) × **0.6** in men, **0.5** in women; use **0.5** and **0.45** in the elderly.
Example: 70 kg man, Na^+ 160 → 0.6 × 70 × (160/140 − 1) = 42 × 0.143 ≈ ***6 L*** *deficit.*

THEN ADD

Ongoing losses (urine output, diarrhea, drains) and **insensible losses** of roughly **30–50 mL/h**. The deficit calculation alone **under-replaces** in a polyuric patient, which is the usual reason the sodium fails to budge.

CORRECT SAFELY *the brain adapts to chronic hypernatremia — undo it slowly*

1. **Restore circulation first.** If the patient is hypotensive or shocked, give **isotonic saline** until perfusion is restored, then switch to free water. Perfusion beats tonicity.
2. **Chronic (> 48 h or unknown): do not exceed 10–12 mEq/L per 24 h**, roughly **0.5 mEq/L per hour**. Correcting faster risks **cerebral edema and seizures** as water rushes into brain cells that have shed osmolytes.
3. **Acute (< 48 h, clearly documented):** can be corrected faster, up to about **1 mEq/L per hour**, because the brain has not yet adapted.
4. **Route: enteral water is safest and cheapest** if the gut works — via NG or PO. Otherwise **D5W** intravenously; watch the glucose, since dextrose loads cause osmotic diuresis and can worsen the problem.
5. **Recheck sodium every 4–6 hours** and recalculate. Formulas predict the first few hours; the patient's ongoing losses decide the rest.

DIABETES INSIPIDUS *hypernatremia with dilute urine despite a high serum osmolality*

	Central DI	Nephrogenic DI
Problem	Deficient **ADH release** — pituitary surgery, trauma, tumor, infiltrative disease	**Renal resistance to ADH** — **lithium**, hypercalcemia, hypokalemia, chronic kidney disease, congenital
Urine	Both give **large-volume dilute urine** (osm often < 300) with a rising serum sodium and osmolality.	
Distinguish	**Desmopressin trial:** urine osmolality **rises substantially in central** DI, and changes **little in nephrogenic** DI. Copeptin assays are increasingly used in place of formal water deprivation testing.	
Treat	**Desmopressin** plus free water	**Remove the cause** (stop lithium, correct calcium), low-solute diet, **thiazide** ± amiloride, and free water. Thiazides work by inducing mild volume depletion and enhancing proximal reabsorption.

A WORKED PLAN *what the actual orders look like*

Step	Order
1 · Decide acuity	Chronic or unknown duration in almost every ward patient → **ceiling 10–12 mEq/L per 24 h**. Document the ceiling and the target explicitly.
2 · Calculate	Free water deficit from the formula, **plus insensible losses** (~30–50 mL/h), **plus measured ongoing losses** such as urine output and diarrhea.
3 · Choose the route	**Enteral water via NG or PO if the gut works**, in divided volumes. Otherwise **D5W** infusion. Add isotonic fluid separately if the patient is also volume-depleted.
4 · Address the cause	**Stop or reduce the driver**: control hyperglycemia, review lactulose and high-protein feeds, treat fever, stop offending drugs, and **fix water access** for the patient who simply cannot ask.
5 · Monitor	**Sodium every 4–6 h**, strict input/output, daily weight. Recalculate rather than trusting the initial estimate — formulas assume no ongoing losses, which is rarely true.

THE HIGH-RISK GROUPS *where this becomes lethal*

WHO GETS INTO TROUBLE

Intubated and sedated ICU patients · **frail elderly** with impaired thirst and limited mobility · anyone **NPO** on saline-only maintenance · **enteral-feed patients** without a free-water order · osmotic diuresis in **hyperglycemia or after mannitol**.

WHY IT MATTERS

Hospital-acquired hypernatremia carries **substantially increased mortality** and largely reflects care processes rather than disease severity. **Prescribe free water as deliberately as you prescribe sodium**, and review the sodium trend on every patient who cannot drink to thirst.

PEARLS & PITFALLS

- **Most inpatient hypernatremia is a nursing and prescribing failure**, not a disease — someone stopped free water. Check the maintenance fluids and the feed regimen.
- **Perfusion before tonicity.** In shock, give isotonic fluid first; a normal blood pressure is more urgent than a normal sodium.
- **Do not forget insensible and ongoing losses.** Replacing only the calculated deficit is the commonest reason sodium plateaus.
- **Correct hyperglycemia and hypernatremia together carefully** — both drive osmotic shifts, and treating glucose alone will drop the effective osmolality fast.
- **Thiazides treat nephrogenic DI** — counterintuitive, but the induced volume contraction reduces free water delivery.
- **Enteral water beats D5W** whenever the gut is usable: no dextrose load, no line, no osmotic diuresis.

References: Adrogué-Madias formulae · NEJM reviews on hypernatremia and disorders of water balance · Endocrine Society guidance on diabetes insipidus / arginine vasopressin deficiency.