

STEP 1 · ARE THERE SEVERE SYMPTOMS *this overrides all workup*

SEVERE — TREAT NOW
Seizure, coma, obtundation, respiratory arrest. 3% saline 100 mL IV over 10 min, repeat up to 2 more times until symptoms resolve. Goal is a rapid rise of 4–6 mEq/L, which is enough to reverse cerebral edema. You do not need the full workup first.

MILD / MODERATE
Nausea, headache, confusion, unsteadiness. Work through the diagnosis before treating, and correct slowly. Chronic asymptomatic hyponatremia is not an emergency — the danger is in correcting it fast.

THEN CHECK TONICITY
Measure serum osmolality. Normal or high means pseudohyponatremia (severe hyperlipidemia, paraproteinemia) or translocational: hyperglycemia, or mannitol given for raised ICP, cerebral edema, glaucoma, or as irrigant. Mannitol patients are hypertonic — no hypertonic saline.

STEP 2 · VOLUME STATUS + TWO URINE TESTS *this is the whole diagnostic algorithm*

Volume	Urine studies	Cause & treatment
Hypovolemic	Urine Na < 20 mEq/L	Extrarenal loss — vomiting, diarrhea, third-spacing, poor intake. Treat with isotonic saline; the sodium corrects as ADH switches off.
Hypovolemic	Urine Na > 20 mEq/L	Renal loss — diuretics (especially thiazides), adrenal insufficiency, cerebral salt wasting, salt-wasting nephropathy.
Euvolemic	Urine osm > 100, urine Na > 30	SIADH — malignancy, pneumonia, CNS disease, drugs (SSRIs, carbamazepine, cyclophosphamide). Exclude hypothyroidism and cortisol deficiency first.
Euvolemic	Urine osm < 100	Primary polydipsia, beer potomania, or low solute intake. The kidney is diluting appropriately; restrict water.
Hypervolemic	Urine Na < 20 (usually)	Heart failure, cirrhosis, nephrotic syndrome — effective arterial volume is low despite total overload. Treat the disease, restrict water, use loop diuretics. Saline makes it worse.

STEP 3 · CORRECT SAFELY *the ceiling matters more than the target*

- 1

Do not exceed 8 mEq/L in any 24-hour period in patients at risk of osmotic demyelination. The minimum useful daily correction is 4–6 mEq/L — so the therapeutic window is narrow, and faster is not better.
- 2

Overcorrection usually happens by accident, when the underlying stimulus resolves and the kidney suddenly dumps free water.
- 3

High ODS risk: Na⁺ ≤ 105, alcohol use disorder, hypokalemia, malnutrition, or advanced liver disease. In these patients stay at the low end of the range.
- 4

Replace potassium at the same time. Giving K⁺ raises the sodium too, and unaccounted-for potassium repletion is a classic cause of inadvertent overcorrection.
- 5

Overcorrecting? Stop hypertonic saline, give free water as D5W, and add desmopressin to re-establish water retention. Co-administering desmopressin proactively is one of the best ways to prevent overly rapid correction in high-risk patients.

TREATING SIADH *the water is the problem, not the salt*

FIRST LINE
Fluid restriction plus increased solute intake is the most effective therapy — oral urea or salt tablets alongside restriction. Restriction alone frequently fails because it is hard to sustain and slow to work.

IF THAT FAILS
Loop diuretic with salt tablets, or a vasopressin receptor antagonist (tolvaptan) with close monitoring, since it can overcorrect. Avoid saline in SIADH — the kidney excretes the salt and retains the water, so the sodium can fall further.

DRUGS & SCENARIOS *the causes you will actually meet on the wards*

Scenario	What is happening, and what to do
Thiazide-induced	Impairs urinary dilution while thirst persists. Classically an older woman within weeks of starting. Stop the thiazide — and it may recur on rechallenge, so record it as an intolerance.
Drug-induced SIADH	SSRIs and venlafaxine, carbamazepine and oxcarbazepine, cyclophosphamide, vincristine, antipsychotics, NSAIDs, and MDMA. Review every new medication started in the preceding month.
Post-operative	Pain, nausea, and opioids all drive ADH, and hypotonic maintenance fluid then supplies the water. Use isotonic maintenance fluid in surgical patients — this is a preventable iatrogenic cause.
Post-TURP / hysteroscopy	Absorbed hypotonic glycine or sorbitol irrigant during monopolar transurethral resection of the prostate. Suspect it on hypertension with bradycardia, the reverse of the usual intraoperative crash. Glycine also gives transient blindness and hyperammonemia. Bipolar uses saline and avoids it.
Beer potomania / low solute	High fluid, minimal solute intake, so there is nothing to excrete the water with. High ODS risk from coexisting alcohol use, malnutrition, and hypokalemia. Sodium can rise dangerously fast once nutrition starts.
Exercise-associated	Endurance athlete who drank large volumes of hypotonic fluid. Can cause fatal cerebral edema with a modest number. Treat with hypertonic saline, never with more water.
Adrenal insufficiency	Hyponatremia with hyperkalemia and hypotension. Check a cortisol before labelling it SIADH — the treatment is completely different.

MONITORING WHILE YOU CORRECT *write these into the plan explicitly*

THE ORDER SET
Sodium every 2–4 h while on hypertonic saline, then 6-hourly. Strict input and output — a sudden large dilute urine output is the warning sign of imminent overcorrection. Check potassium, magnesium, and glucose alongside.

THE HANDOVER
State the starting sodium, the target for the next 24 h, and the maximum permitted. Overcorrection almost always happens across a shift change, when the underlying stimulus resolves overnight and nobody has written down the ceiling.

PEARLS & PITFALLS

- Thiazides, not loops, are the classic diuretic cause — loops impair concentrating ability, thiazides impair dilution.
- In severe symptomatic disease, bolus 3% saline is safer than an infusion — you get the rapid 4–6 mEq rise you need without drifting past the ceiling.