

Hyponatremia

Low plasma [Na⁺] due to water retention & pt drinking excess water
Plasma [Na⁺] <133mmol/L (Normal: 133-145 mmol/L)

Note: urine is made at the same osmolality as plasma, but ADH can control the final osmolality of urine to suit the body's water needs. If there is no ADH, urine is usually 50mmol/kg.

Check Plasma Osm

Artifactual

True

-Artifactual = may just be lab error!

Plasma Osmolality high (>295 mmol/kg) or normal (280 - 295 mmol/kg)
(↑ TBW relative to Na⁺ & other effective osmoles)

Plasma Osmolality low (<280 mmol/kg)
(excess TBW relative to Na⁺ alone)

More common

Normal Posm (rare)

High POsm

Inappropriate urine osmolarity
Hyper-osmolar, or not hypo-osmolar enough (>100mmol/kg)

Check Urine Osmolarity & volume status

Appropriately Hypo-osmolar urine (<100mmol/kg)
(↑ TBW → appropriately ↓ ADH secretion → appropriate kidney response, intact H₂O excretion ability;) (No signs of hypovolemia)

Severe hyper-proteinemia Severe Hyper-triglyceridemia

Hyperglycemia (i.e. uncontrolled diabetes) Mannitol (used to treat cerebral edema)

(↑ ADH, concentrating urine, impairing H₂O excretion. May be appropriate (ie. Low EABV) or inappropriate)
Check urine [Na⁺], in mmol/L

→The abnormal excess of proteins or lipids (effective osmoles) in the plasma draw in water, ↑ plasma volume, diluting [Na⁺], while keeping plasma osmolality constant.
→↑ osmolal gap: calculated Posm < measured Posm

Non-Na⁺ effective osmoles in blood draw water from ICF into ECF, diluting [Na⁺]
→This effect is temporary!
Chronically, glucose/mannitol filtration will cause polyuria
→ ↑ water loss → hypernatremia!

Urine [Na⁺] <20 (LOW!):
RAAS is working to reabsorb Na⁺ (EABV is low)

Less common: Normal urine [Na⁺] = 25-250:
RAAS not that active, no need (EABV normal)

Hypervolemia:
Underfill Edema w/ low EABV:
1. ADH appropriately ↑ → ↑ water reabsorption → high urine osmolarity
2. ↑ RAAS → ↑ Na⁺ reabsorption → low urine [Na⁺]
• Heart failure
• Cirrhosis
• Nephrotic syndrome

Hypovolemia
Low plasma volume ↑ ADH (appropriately) → ↑ water reabsorption → ↑ urine osmolarity Na⁺ loss from excess Vomiting, Diarrhea, or sweating, combined with drinking only water (this is what most sick people present with)
• Thiazide diuretics (more Na⁺ loss than H₂O loss)

Euvolemia:
1. Adrenal insufficiency (cortisol normally inhibits ADH)
2. Hypothyroidism (T4 normally inhibits ADH)
3. SIADH: Syndrome of Inappropriate ADH
• Nausea, pain, vomiting
• Post-surgery
• Cancer (lung, pancreas/GI)
• CNS disease (stroke, infection, trauma)
• Pulmonary dx (i.e. pneumonia)
• Drugs (NSAIDs, etc)
• Idiopathic

Primary Polydipsia (Drinking too much water without ingesting Na⁺)
↓ **Osmole intake** (too little Na⁺ ingested per volume of water drank)
→ Beer potomania
→ "Tea + toast diet"

Clinical pearl: in sick inpatients with low Na⁺ and high Creatinine, its most likely just because they are sick (losing Na⁺ via vomiting/diarrhea, + hypovolemic)

Principle: to kidneys, preservation of water volume (EABV) is always more important than maintaining serum osmolality!

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Abbreviations:

TBW = Total body water
POsm = plasma osmolality
ICF = intracellular fluid
ECF = extracellular fluid (i.e. blood, interstitial fluid)

Practically speaking: In any electrolyte issue, ask yourself “what problem does the patient have, could this problem cause electrolyte issues, and is it worth investigating” before ordering tests to try to investigate the issue.

Hypernatremia

High plasma [Na⁺] due to excess free water loss & inadequate water intake
 Plasma [Na⁺] > 145mmol/L (Normal: 133-145 mmol/L)

Check urine output

LOW urine output (oliguria)

(< 500mL/24 hrs)

Hypodipsia (↓ water intake)

- ↓ **thirst drive**
(hypothalamic injury)
- ↓ **access to water**

↓ water ingestion, plus natural loss of hypotonic fluids over time (respiration, sweating) → hypernatremia

Extra-renal water loss

- GI**
(large-volume diarrhea)
- Resp**
(hyperventilation, mechanical ventilation)
- Skin**
(large burns, ↑ sweating)

Extensive Loss of hypotonic fluids from GI tract + skin (AND if pt drank less free water) → hypernatremia

→As a response: Functional kidneys reabsorb more Na⁺/water to boost blood volume; ↓ urine output.

→But urine is still “concentrated” (urine osmolality still >300) b/c of other, non-effective osmoles in the urine like urea & shit.

HIGH urine output (polyuria)

(>3L/24hr in adults, >2L/24hr in kids)

→Pt will be appropriately thirsty (**polydipsia**)

Check urine Osmolality

Osmotic diuresis Hyper-osmolar urine (>300mmol/kg)

→Presence of abnormally high [osmole] in the tubule ↓ water reabsorption, ↓ ECF water volume → Hypernatremia
(**more common**)

Uncontrolled hyperglycemia

→Diabetes mellitus

Excess Urea

Due to excess protein feeds in ICU patients

Mannitol

→Brain injury tx

Loop Diuretics

→Inhibit much Na⁺ & water reabsorption, causing massive Na⁺ & water loss
 →Also washes out medullary [Na⁺] gradient, ↓ urine concentration in collecting duct, causing further water loss.
 →Loss of water > loss of Na⁺ = hypernatremia

Diabetes Insipidus Hypo-osmolar urine (<300mmol/kg)

→ ↓ ADH effect ↓ water reabsorption in collecting duct → ↓ ECF water volume → Hypernatremia

Nephrogenic

(↓ ADH sensitivity)
 →Often congenital (genetic dx)
 →Acquired causes: drugs (lithium, etc), electrolyte imbalance (hypokalemia, etc), chronic kidney dx

Central

(↓ ADH production)
 →Head trauma, infection, surgery, cancer, or infiltrative dx affecting hypothalamus or posterior pituitary

→If hypernatremic at all, plasma [Na⁺] (and plasma osmolality) won't be too high due to functional thirst response/polydipsia.

→Distinguish eunatremic DI from eunatremic primary polydipsia via the water-deprivation test!

Note:

-Urine output changes based on the volume of water ingested, so guidelines of how much volume constitutes “Oliguria” or “polyuria” are not hard & fast.