

Hyponatremia & Hypernatremia

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Sodium Disorders

AKA "water disorders"

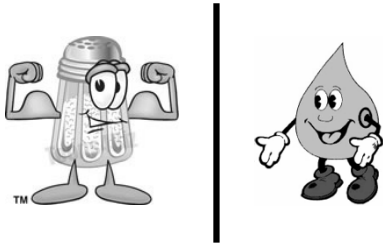
$H_2O \text{ in} > H_2O \text{ out} = \text{Hyponatremia}$

$H_2O \text{ out} > H_2O \text{ in} = \text{Hypernatremia}$

Outline

- Background & Definitions
- Hyponatremia
 - Diagnostic Approach
 - Treatment Considerations
- Hypernatremia
 - Diagnostic Approach
 - Treatment Considerations
- Questions...

Background:
Salt (NaCl) and Water (H₂O)



Principles of Electrolyte
Disturbances

- Clinical manifestations determine urgency of treatment, NOT absolute laboratory values
- Speed and magnitude of correction dependent on clinical circumstances
- Frequent reassessment of electrolytes required

Sodium



- Primary determinant of serum osmolality
- Primary extracellular electrolyte
- Regulates extracellular volume
 - If total body sodium goes UP -> extracellular volume goes UP
- Hyponatremia, serum Na < 135
- Hypernatremia, serum Na > 145

Water: Where Does it Go?

Intra-cellular 67% or 2/3 Extra-cellular 33% or 1/3

Intra-cellular 67% or 2/3	Interstitial 25% or 3/4
	Intravascular 8% 1/4

Everywhere!

Fast Facts: H₂O & Salt

- H₂O can move freely between all compartments
 - All compartments have equal osmolalities
- Salt cannot move freely between all compartments
 - Maintains extracellular (intravascular) volume
- Osmolality determines movement of H₂O

Serum Osmolality


- Osmolality = Osmoles/kg of water
- What makes of serum osmolality?


2 (Na⁺) + Glucose/18 + BUN/2.8
- Normal serum osmolality is 285-295
 - Kidney runs a pretty tight ship!


Maintaining Osmolality

- High plasma osmolality (osm)
 - H₂O flows OUT of cells and cells shrink
 - i.e. CPM (central pontine myelinolysis)
- Low plasma osm
 - H₂O flows INTO cells and cells swell
 - i.e. cerebral edema
- Both scenarios interfere with cell function

Definitions

Dehydration: Too little H₂O 

Volume Depletion: Too little NaCl 

Volume Overload: Too much NaCl 

Volume Status = Salt Status

Choosing a fluid...

Ask the question...

- Is this a **H₂O** problem?
- Is this a **NaCl** problem?
- Is this BOTH a **NaCl** and **H₂O** problem?

Remember... IVNS (or LR) ALWAYS fluid of choice for volume resuscitation

Background: IV Fluids

- Solute in a particular IVF determines where the IVF distributes
- For example...
 - D5W distributes to all compartments
 - IVNS (0.9%) - only to the extracellular space
 - LR - only to the extracellular space
 - Plasma expanders (pRBC, FFP, albumin, hetastarch) - only to the intravascular space

Water: Where Does it Go?

Intra-cellular 67% or 2/3 Extra-cellular 33% or 1/3

Intra-cellular 67% or 2/3	Interstitial 25% or 3/4
	Intravascular 8% 1/4

Fluid Selection

• Normal Saline (IVNS)
Isotonic (308 osm)
 Sodium : 154 meq/L
 Chloride : 154 meq/L

• 3% saline
Hypertonic (1027 osm)
 Sodium: 513 meq/L
 Chloride: 513 meq/L

• Lactated Ringers (LR)
Hypotonic (274 osm)
 Sodium : 130 meq
 Chloride : 109 meq
 Calcium : 3 meq
 Potassium : 4 meq
 **Lactate : 28 meq
 **Lactate metabolized to bicarbonate in the liver

IV Fluid Examples...

- 1 Liter of D5W
 - 67% or 670 cc to intracellular space
 - 25% or 250 cc to interstitial space
 - 8% or 80 cc to intravascular space
- 1 Liter of IVNS (0.9%)
 - ¾ or 75% or 750 cc to interstitial space
 - ¼ or 25% or 250 cc to intravascular space

IV Fluid Examples...

- 1 Liter of ½ NS (0.45%)
- Same as 500 cc of 0.9% and 500 cc of H₂O
 - 335 cc to intracellular space
 - 125 cc to interstitial space
 - 40 cc to intravascular space
- 500 cc of H₂O
- 500 cc of IVNS
 - 375 cc to interstitial space
 - 125 cc to intravascular space



Normal Kidney (and normal brain)

- Increased total body sodium (hypervolemia)
 - Kidney increases sodium excretion
- Decreased total body sodium (hypovolemia)
 - Kidney decreases sodium excretion
- Normal total body sodium (euvoemia)
 - You pee what you eat



Normal Volume Regulation

- Adequate GFR
- Adequate distal delivery of Na and H₂O to loop of Henle and collecting duct
- Intact tubular function for reabsorption
- Central ADH secretion
- ADH responsiveness in the kidney

What about Urine Electrolytes?

- There is no normal value
- Generally speaking...you pee what you eat
- Interpretation of urine electrolytes should be defined as *appropriate* or *inappropriate* NOT *normal* or *abnormal*

You pee what you eat...



Hyponatremia

Remember...

Hyponatremia is *ALWAYS* a H₂O problem and *SOMETIMES* a NaCl problem

Always *too much* H₂O!



Hyponatremia

- True Hyponatremia
 - Hypo-osmolar hyponatremia (*most common*)
 - Hypovolemic
 - Euvolemic
 - Hypervolemic
- Pseudohyponatremia

Pseudohyponatremia

- Hyper-osmolar hyponatremia (*less common*)
 - Hyperglycemia
 - HIGH measured and calculated osm
 - (Na ↓1.6meq/dL for every 100mg/dl ↑in glucose > 100)
 - Mannitol
 - HIGH measured osm but LOW calculated osm
- Normo-osmolar hyponatremia (*very uncommon*)
 - Hypertriglyceridemia
 - Hyperproteinemia (MM)
 - NORMAL measured osm but LOW calculated osm

Hyponatremia

- Occurs when H₂O in > H₂O out
 - *Can't get hyponatremic without water intake*
 - Watch for water "hiding" in gtt, flushes, meds, hypotonic fluids...
- Ingestion of too much H₂O with normal kidneys (i.e. psychogenic polydipsia)
 - Increased H₂O in
- Ingestion of "normal" H₂O with renal failure
 - Decreased H₂O out
- Ingestion of "normal" water with *increased ADH activity*
 - Decreased H₂O out

Hyponatremia: Signs & Symptoms

- Primarily due to osmotic shifts
- Water flows from hypotonic extra-cellular compartment to relatively hypertonic intracellular compartment -> cerebral edema
- Signs & symptoms primarily neurologic
- For non-neuro patients symptoms depend more on the *rate of change* NOT the absolute number (chronic > 48 hr)

Hyponatremia: Signs & Symptoms

ACUTE

- Severity of symptoms reflects severity of cerebral "overhydration"
- Malaise, Nausea, HA, lethargy, obtundation, seizures, coma, respiratory arrest

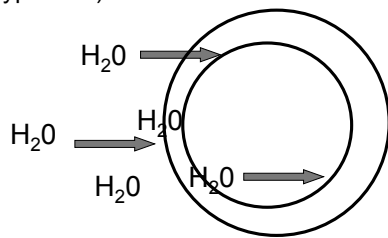
CHRONIC

- May be asymptomatic
- Nonspecific: fatigue, nausea, dizziness, gait disturbance, forgetfulness, confusion, lethargy, muscle cramps

Hyponatremia

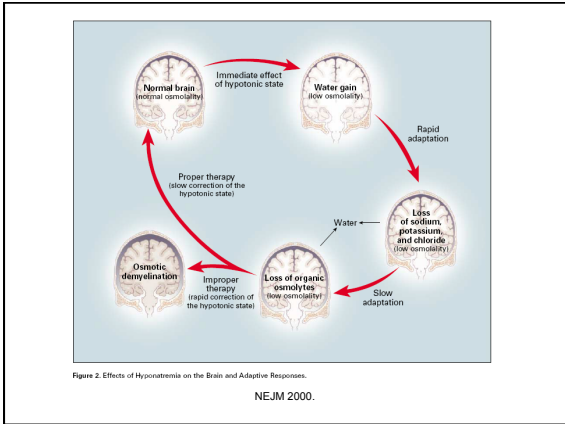
Extracellular
(hypotonic)

Intracellular
(hypertonic)



Cerebral Compensation

- 1st line of "self" defense
 - Increased hydrostatic pressure from cerebral edema causes fluid to shift to CSF, shunted into systemic circulation
- 2nd line of defense
 - Secretion of intracellular solutes (to decrease gradient drawing water into cells) - rapid
 - Secretion of osmolytes (after 24-48hr) -slower



ADH: Antidiuretic Hormone

ADH: Antidiuretic Hormone

- Produced in posterior pituitary gland
- Primary function -> maintain plasma osmolality
- Secondary function -> volume regulation
- Action via H₂O reabsorption in collecting tubules (V₂ receptors)

ADH: "Appropriate"

- Osmoreceptors in hypothalamus detect changes in osmolality
- Baroreceptors detect hypovolemia
- Increase in plasma osmolality stimulates ADH and thirst
- If plasma osm too high
 - ADH and thirst activated
- If plasma osm too low
 - ADH and thirst inactivated





ADH: "Appropriate"

SAADH – syndrome of "appropriate" ADH secretion

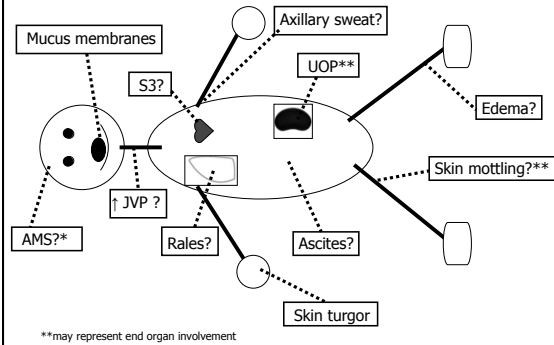
- Appropriate ADH secretion
 - Hyperosmolar state (e.g., hyponatremia)
 - Hypovolemic state
- ADH increases H₂O reabsorption in kidney
- ADH is last response to hypovolemia
 - Released after decrease in BP by 10-15mmHg
 - Not very effective for replacing volume
 - REMEMBER only 8% of H₂O stays in intravascular space

Approach to Hyponatremia







Hyponatremia

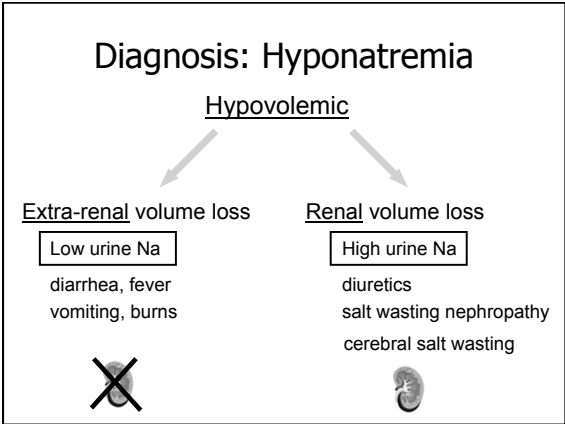
- 1st - Assess volume status
 - Determine whether it is a  problem,
 - a  problem or both
- 2nd - Check urine sodium
 - add urine potassium and osmolality in SIADH

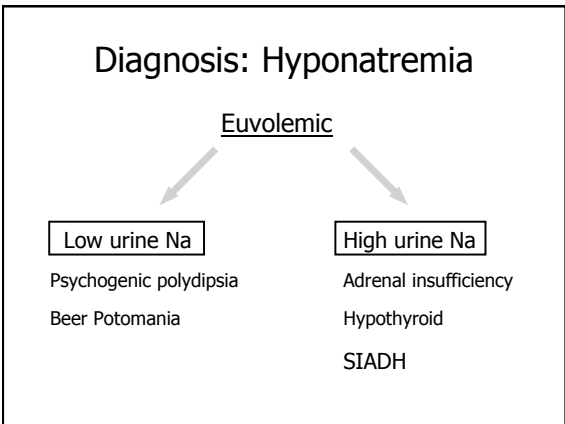
The "volume assessment" physical exam

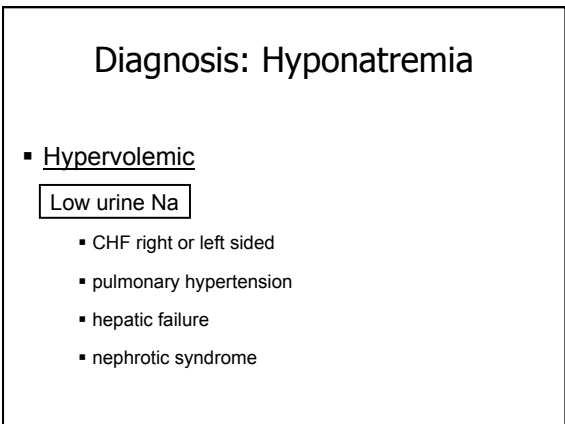


True Hyponatremia

- Hypovolemic hyponatremia
 - Too little  too much 
- Euvolemic hyponatremia
 - Just the right amount of  but too much 
- Hypervolemic hyponatremia
 - Too much  and too much 







SIADH
Syndrome of "Inappropriate" ADH Secretion

ADH "Inappropriate" : SIADH

SIADH – syndrome of inappropriate ADH secretion
1st described in 1957

5 criteria:

- Hypotonic hyponatremia (low osmolality), $S_{Na} < 135$
- Euvolemia (not on diuretics)
- Urine Na > 20
- Urine osm $>$ plasma osm
- Rule out adrenal and thyroid dysfunction

Schwartz et al. Am J Med, 1957.

ADH "Inappropriate" : SIADH

- Due to excessive ADH release or increased sensitivity to ADH
- Characterized by hyponatremia in the setting of inappropriately concentrated urine
 - *i.e. should have dilute urine with hyponatremia*
- No defect in Na^+ handling in SIADH
 - High salt intake, high salt output (high urine output)
 - Low salt intake, low salt output (low urine output)

Common Etiologies of SIADH

- CNS disease (CVA, SAH, SDH, trauma, autoimmune, infection...)
- Ectopic (malignancy, classic small cell)
- Medications (TONS...)
- Pulmonary disease
- Stress, nausea, pain
- HIV

Cerebral Salt Wasting (CSW)

Syndrome of "Appropriate" ADH Secretion
"SAADH"

CSW: Diagnosis

- In the setting of CNS disease
- Clinical evidence of hypovolemia!
 - Evidence of net negative sodium balance (if possible)
- Hyponatremia (< 135 meq/L)
- Inappropriately elevated urine osmolality
 - > 100 mosmol/kg, usually > 300 mosmol/kg
- Urine sodium > 40 meq/L
- Low serum uric acid concentration due to urate wasting

Cerebral Salt Wasting (CSW)

- Usually occurs within first 10 days (2-10 days after event)
- Most commonly seen with SAH but has been described with meningitis, stroke, cerebral metastases
- 2 components
 - Renal salt wasting -> volume depletion
 - Hyponatremia

SIADH versus CSW

SIADH

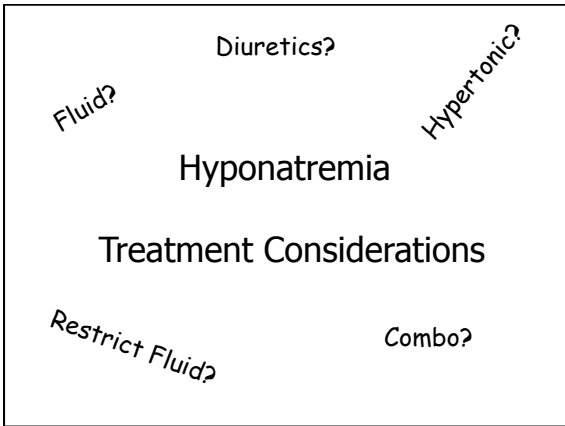
- Euvolemic
- Urine Na excretion equals urinary sodium intake
- Treatment is fluid restriction

CSW

- Hypovolemic
- Net negative sodium balance
- Treatment is IV saline

Hyponatremia in a Neuro ICU

- Most common electrolyte abnormality in a Neuro ICU
- Differential Diagnosis: consider SIADH, CSW, acute adrenal insufficiency
- Up to 43% of patients with SAH develop hyponatremia
- Hyponatremia after SAH is a risk factor for and predictor of vasospasm and cerebral ischemia
- Hyponatremia potentiates cerebral edema
- Early diagnosis and effective treatment is critical for hyponatremic patients with intracranial disease



Hyponatremia: Treatment

- Hypovolemic ? – replete volume deficit, rule out adrenal insufficiency
 - Give IVNS
- Hypervolemic ? – increase free H₂O loss and NaCl loss
 - Give diuretics
- Euvolemic?
 - Restrict water intake
 - Increase water loss

? symptomatic

SIADH: Treatment

- Restrict free H₂O intake
 - Calculate Free H₂O Clearance
- Increase free H₂O loss
 - Hypertonic saline + diuretics
 - Vaptans (V₂ antagonists) – pure aquaresis
 - Salt (or urea) tablets + diuretics
- Correct slowly due to possibility of demyelinating syndromes (CPM)

Free H₂O Clearance

Urine volume = C_E + C_{EF}

$$C_E = \frac{U_{Na} + U_K}{S_{Na} + S_K}$$

Example 1: 60 yo woman with metastatic melanoma (to brain)
Serum Na 115, Urine Na 80, Urine K 24, Uosm 500

$$U_{vol} = (C_E) 0.9 \left(\frac{80 + 24}{115} \right) + (C_{EF}) 0.1$$

Only 10% of all urine output is free water, 90% is electrolyte clearance

EXAMPLE: IF UOP 1 liter, only 100cc is H₂O. Any H₂O intake > 100cc + insensible losses will cause serum Na⁺ to fall

Free H₂O Clearance

Urine volume = C_E + C_{EF}

$$C_E = \frac{U_{Na} + U_K}{S_{Na} + S_K}$$

Example 2: 50 year old woman s/p large hemorrhagic
MCA CVA with Serum Na 119, Urine Na 140, Urine
K 20, Uosm >700

$$U_{vol} = (C_E) > 1 + (C_{EF})$$

NONE of urine output is free H₂O, any intake
exceeding insensible losses will drop serum Na⁺

Maximum urine volume

$$\text{Max daily urine vol} = \frac{\text{daily solute load}}{\text{minimum urine []}} \rightarrow \frac{10\text{mmol} \times \text{kg}}{\text{normal} \sim 50}$$

Examples: "normal" intake

80 kg man with min urine [] of 50 can drink 16 L

80 kg man with min urine [] of 400 can drink 2 L

80 kg man with min urine [] of 600 can drink 1.3 L

SIADH: Acute Symptomatic Hyponatremia

- Using 3% saline + lasix for SIADH = net aquaresis
 - Goal salt in = salt out (euvolemic to start)
- Replace all Na lost in the urine with 3%
 - 3% saline Na⁺ content 513 meq/L
 - 1 mL of 3% = 0.5 meq of Na⁺
- Example: If urine Na⁺ is 80 meq/L and urine volume is 500cc over 2 hr, 40meq of Na⁺ has been lost and must be replaced with 80cc of 3%

Vaptans

- Provide option for net aquaresis
- Tolvaptan & Conivaptan available in U.S.
- For use in heart failure, cirrhosis, SIADH
- Caution: overly rapid correction is a risk
- Caution: avoid use in hypovolemic patients
- Increased thirst side effect in studies (in absence of hypernatremia)
- Often requires nephrology approval

Why use hypertonic saline?

- To avoid worsening hyponatremia in SIADH, osmolality of fluid must exceed that of urine
- Risk of missing CSW -> increased vasospasm and cerebral ischemia

Why use hypertonic saline?

Example: Urine osm 600 (max ability to dilute)

- 1 liter of IVNS (308 osm)
 - 308 osm/616 osm = 0.5 L UOP (308 osm will be excreted in 500cc of urine or 616 osm urine) and 500cc of water will be retained, SNa will fall
- 1 liter of 3% saline (1026 osm)
 - 1026 osm/616 osm = 1.6 L UOP (1026 osm will be excreted in 1.6L of urine or 616 osm urine) and no water will be retained, SNa will rise

Using 3% Saline for Hyponatremia (SIADH or CSW)

Na^+ deficit = total body water x (target Na^+ - serum Na^+)
e.g. $(0.6 \times 80\text{kg} \times (130 - 120)) = 480 \text{ meq}$

Rate of infusion (cc/hr) = $\frac{\text{Na}^+ \text{ deficit (meq)} \times 1000}{\text{infusate Na}^+ \text{ (meq/L)} \times \text{time (hours)}}$
e.g. $(480 \times 1000) / (513 \times 24) = 39 \text{ cc/hr}$ of 3% saline

***Total Body Water**

- Women 0.5 x weight in kg (~0.4 in elderly)
- Men 0.6 x weight in kg (~0.5 in elderly)

Treatment: Hyponatremia

$$\Delta \text{ serum Na} = \frac{\text{Infusate Na} - \text{Serum Na}}{\text{Total Body Water}^* + 1}$$

- 1 liter 3% saline [Na^+] = 513 meq/L

Example: If SNa 120 and 1 liter 3% given, Na^+ will rise by 9.8 meq

***Total Body Water**

- Women 0.5 x weight in kg (~0.4 in elderly)
- Men 0.6 x weight in kg (~0.5 in elderly)

CSW: Treatment

1. Avoid Volume Depletion!
2. Treat hypovolemic hyponatremia
 - Calculate & replace deficit (use 3% if symptomatic)
 - Match I's & O's for ongoing losses
 - $\text{Urine Na}^+ \times \text{Urine Volume} = \text{meq of ongoing loss}$

Example of ongoing losses: $\text{UNa } 150 \text{ (meq/L)} \times 5 \text{ L UOP (750 meq)} = \sim 4.8\text{L IVNS or } 200\text{cc/hr IVNS}$

CSW vs. SIADH: Treatment

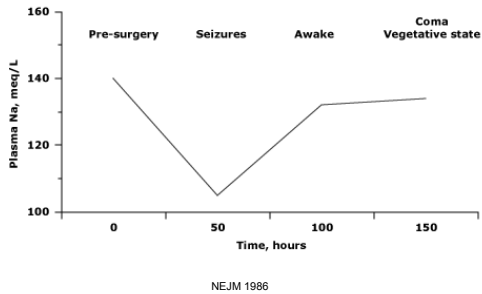
- Response to isotonic saline
 - SIADH - worsening of hyponatremia
 - Lose the salt, hold on to the H_2O
 - CSW – improvement in hyponatremia (unless coexistence of CSW and SIADH)
- Response to fluid restriction
 - SIADH – appropriate
 - CSW – clinical deterioration, high risk of significant complications

Central Pontine Myelinosis

Watch Out!!

- Complication of overly rapid correction of hyponatremia
- Caused by severe damage to the myelin sheath in the pons
- Alcoholics, malnourished and elderly females are at highest risk

Hyponatremia: Complications of Rapid Correction



Hyponatremia: Treatment

- Rate of correction is critical
- Correction not to exceed ~10meq/24 hr
 - On occasion will need to give water back to slow rate of correction
- **WATCH OUT** for significant increase in urine output with correction of hyponatremia
- **CAUTION: SOMETIMES TREATMENT CAN BE RISKIER THAN THE PROBLEM ITSELF!**

It Can Be Complicated...

- A patient's condition may change over time
- Causes of hyponatremia may coexist
- Treatment of one problem may unmask another (e.g. volume depletion and SIADH)
- Always go back and reassess volume status, follow urine osm and urine electrolytes
- Slow correction!!!

Case #1

45 yo alcoholic admitted with AMS after being found at home in his own vomit with multiple empty bottles of beer. SNa 125, UNa 10. HR 110, BP 80/50. Exam: confused, nonfocal, dry MM, no edema, no axillary sweat. Unable to provide any history.

Case #1

What's the cause of his hyponatremia?

- 1) Hypovolemic hyponatremia from vomiting
- 2) Beer potomania
- 3) Euvolemic hyponatremia 2/2 SIADH
- 4) Hypervolemic hyponatremia 2/2 alcoholic cardiomyopathy

Case # 1

What's the treatment?

- 1) IVNS bolus 1 liter
- 2) IVNS 125 cc/hr
- 3) D5 1/2 NS 125 cc/hr
- 4) Fluid restriction
- 5) Lasix 20mg IV x 1

Case #2

45 yo alcoholic admitted with AMS after being found at home confused with multiple empty bottles of beer. SNa 125, UNa 10. HR 90, BP 100/50. Exam: confused, nonfocal, moist MM, 2+ edema, elevated JVP, rales and S3 on exam. Unable to provide any history.

Case #2

What's the cause of his hyponatremia?

- 1) Hypovolemic hyponatremia from vomiting
- 2) Beer potomania
- 3) Euvolemic hyponatremia 2/2 SIADH
- 4) Hypervolemic hyponatremia 2/2 alcoholic cardiomyopathy

Case # 2

What's the treatment?

- 1) IVNS bolus 1 liter
- 2) IVNS 125 cc/hr
- 3) D5 1/2 NS 125 cc/hr
- 4) Fluid restriction
- 5) Lasix 20mg IV x 1

Case #3

45 yo alcoholic admitted with AMS after being found at home confused with multiple empty bottles of beer. SNa 125, UNa 110. HR 80, BP 120/70. Exam: confused, nonfocal, moist MM, no edema, no elevated JVP, cardiopulm exam without rales or S3. Unable to provide any history.

Case #3

What's the cause of his hyponatremia?

- 1) Hypovolemic hyponatremia from vomiting
- 2) Beer potomania
- 3) Euvolemic hyponatremia 2/2 SIADH
- 4) Hypervolemic hyponatremia 2/2 alcoholic cardiomyopathy

Case # 3

What's the treatment?

- 1) IVNS bolus 1 liter
- 2) IVNS 125 cc/hr
- 3) D5 1/2 NS 125 cc/hr
- 4) Fluid restriction
- 5) Lasix 20mg IV x 1

Hypernatremia

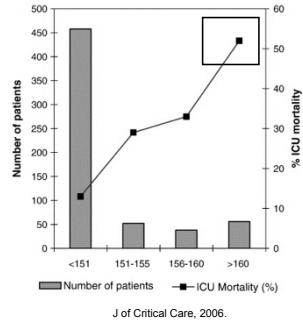
I'm Thirsty!!!!









Hypernatremia: Background

- *Associated* with increased mortality in hospitalized patients (MICU/SICU)
- Commonly part of the "treatment" in NSICU
 - Osmotic therapy (i.e. mannitol)
- Only severe hypernatremia ($S_{Na} > 160$) independently associated with increased mortality in NSICU

Hypernatremia Associated Mortality



Hypernatremia

- It is *ALWAYS* a water problem (& sometimes a salt problem)
- Hypovolemic hypernatremia
 - ↓↓  and ↓ 
- Euvolemic hypernatremia
 - ↓  and normal 
- Hypervolemic hypernatremia
 - Not enough  and ↑↑ 

Hypernatremia

- Protection against hypernatremia
 - Thirst
 - ADH
- Water moves *toward* increased tonicity
- Leads to cell shrinkage
- Impairs cellular function
- Can be therapeutic in cerebral edema

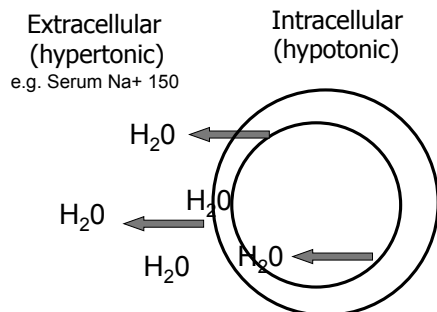
ADH in Hypernatremia

- WITH ADH
 - Reabsorb H₂O in collecting tubule
 - Excrete concentrated urine
 - High urine Na, High urine Osm, low UOP
- WITHOUT ADH
 - Unable to reabsorb H₂O in collecting tubule
 - Excrete dilute urine
 - Low urine Na, Low urine Osm, high UOP

Hypernatremia

- H₂O flows OUT of cells and cells shrink
- **GOAL**: Appropriate rate of correction, H₂O flows IN and cells return to normal size
- **PROBLEM**: Overly rapid correction of hypernatremia
- **COMPLICATION**: H₂O flows IN cells and cells swell leading to cerebral edema

Hypernatremia



Hypernatremia: Causes

- Decreased H₂O intake (*most common*)
 - Poor access (elderly, altered mental status)
 - Iatrogenic (inadequate water prescription)
- Increased H₂O loss
 - DI (nephrogenic or cerebral)
 - Iatrogenic (diuretics)
 - Osmotic loss (hyperglycemia, mannitol, post ATN)
 - Post obstructive AKI
- Gain of sodium (*uncommon*)
 - Massive hypertonic solutions

Hypernatremia: Diagnosis

- Step 1 – Assess volume status
 - Is it only a H₂O problem or is it a salt and a H₂O problem???
 - Gain of Sodium???
 - Loss of Water???. (i.e. HONK, DI, mannitol)
 - Inadequate water intake???
- Step 2 – Calculate H₂O deficit

Hypernatremia: H₂O deficit

$$\text{H}_2\text{O deficit (L)} = [0.6 \times \text{wt (kg)}] \times [\frac{\text{obs Na} - 1}{140}]$$

Example: 80 kg male with serum Na 150

$$0.6 \times 80 \times (150/140 - 1) \sim 3.4\text{L}$$

+ "insensible" losses (skin, respiratory, GI)

+ "sensible" losses (urine output)

Hypernatremia: H₂O deficit

$$\text{H}_2\text{O deficit (L)} = [0.6 \times \text{wt (kg)}] \times [\frac{\text{obs Na} - 1}{140}]$$

Example: 80 kg male with serum Na 145
 $0.6 \times 80 \times (145/140 - 1) \sim 1.7\text{L}$

- + "insensible" losses (skin, respiratory, GI)
- + "sensible" losses (urine output)

Diabetes Insipidus

- It's an ADH issue
- Can be Central or Nephrogenic
- Central
 - Absence of ADH secretion or inadequate ADH secretion from hypothalamus
 - Treatment: Replace ADH using ddAVP
- Nephrogenic
 - Absent or decreased renal responsiveness to ADH
 - No treatment, just give lots and lots of water

Diabetes Insipidus

- Polyuric state
- Low urine osm
- Lots and lots of water loss in the urine
- *Inappropriate* to have low urine osm and high serum osm

Hypernatremia: Treatment

- Hypovolemic ? – replete free H₂O deficit and replete volume deficit
 - Give IVNS and D5W (replete salt & water)
- Hypervolemic ? – replete free H₂O deficit and increase NaCl loss
 - Give D5W (or enteral H₂O) and diuretics (replete H₂O and remove salt)
- Euvolemic? – replete free H₂O deficit
 - Give D5W or enteral H₂O

Hypernatremia: Treatment

- Provide intravascular volume replacement if indicated
- Consider giving 1/2 of free H₂O deficit initially over 1st 24 hours
- Reduce Na cautiously:
 - 1 meq/hr if ACUTE
 - 0.5 meq/hr if timing unknown or chronic
- Secondary neurologic syndromes with rapid correction
 - Water moves back into cells -> cerebral edema

Hypernatremia: Treatment

- Watch for high urine output (inappropriate)
 - Suggests DI, other osmotic loss or recovering AKI
- Give "enough", uncomfortable to be "thirsty"
- Can replete IV and/or PO

Approach to Hypernatremia

- Step 1. Determine volume status. (? Salt problem too)
- Step 2. Calculate free water deficit.
- Step 3. Choose a fluid type.
- Step 4. Choose a rate.
- Step 5. Estimate ongoing "sensible" losses.
- Step 6. Estimate ongoing "insensible" losses.
- Step 7. Determine underlying cause if possible.

Hypernatremia: Clinical Pearls

- Hypernatremia is *always* a water problem and *sometimes* a salt problem.
- Calculation of H₂O deficit represents only a snapshot in time.
- Hypernatremia *always* reflects a hyperosmolar state.
- Patients must have a defect in their thirst mechanism or limited access to H₂O in order for hypernatremia to persist.
- Failure to consider ongoing sensible and insensible losses is the *most common cause of undercorrection*.
- Hypernatremia does not provide any information about total body salt or volume status.



Questions?????



POST-TEST

1) Hyponatremia is *ALWAYS* a disorder of which of the following?

- a) salt
- b) water
- c) neither
- d) both

POST-TEST

2) Hypernatremia is *ALWAYS* a disorder of which of the following?

- a) salt
- b) water
- c) neither
- d) both

POST-TEST

3) Which condition is characterized by a volume depleted state?

- a) Cerebral Salt Wasting (CSW)
- b) SIADH
- c) Neither
- d) Both

POST-TEST

4) Which condition is characterized by a euvolemic or "normal" volume state?

- a) Cerebral Salt Wasting (CSW)
- b) SIADH
- c) Neither
- d) Both

POST-TEST

5) Water can move freely between the intracellular and extra-cellular space.

- a) True
- b) False

POST-TEST

6) Which of the following is NOT a cause of SIADH?

- a) Medications
- b) Pulmonary disease
- c) CNS disease
- d) Congestive heart failure
- e) Pain

POST-TEST

7) What is a normal urine sodium?

- a) 20
- b) 40
- c)
- d) 70

POST-TEST

8) What is the primary determinant of a person's volume status (i.e. hypovolemia, euvoemia, hypervolemia)?

- a)
- b) water
- c) blood pressure
- d) pulse
- e) none of the above

POST-TEST

9) What is the risk of rapidly correcting hyponatremia?

- a) cerebral edema
- b)
- c) volume overload
- d) none of the above

POST-TEST

10) What is the risk of rapidly correcting hyponatremia?

- a) cerebral edema
- b) central pontine myelinosis (CPM)
- c) volume overload
- d) none of the above

POST-TEST

11) Which of the following is NOT commonly associated with polyuria?

- a) CSW (cerebral salt wasting)
- b) SIADH
- c) Diabetes Insipidus (central or nephrogenic)
- d) Psychogenic polydipsia

POST-TEST

12) Dehydration refers to which of the following

- a) too little salt
- b) too little water = volume depletion
- c) neither
- d) both

Thanks!

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