Hyponatremia & Hypernatremia

9th Annual Rocky Mountain Hospital Medicine Symposium
Denver, Colorado
Paula Dennen, MD
Assistant Professor of Medicine
Nephrology and Critical Care Medicine
October 6, 2011

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?

<table>
<thead>
<tr>
<th>Intra-cellular 67% or 2/3</th>
<th>Extra-cellular 33% or 1/3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interstitial 25% or 3/4</td>
<td></td>
</tr>
<tr>
<td>Intravascular 8% 1/4</td>
<td></td>
</tr>
</tbody>
</table>

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?
  \[ 2 (Na^+) + \text{Glucose/18} + \text{BUN/2.8} \]
- 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?

<table>
<thead>
<tr>
<th>Intra-cellular</th>
<th>Extra-cellular</th>
</tr>
</thead>
<tbody>
<tr>
<td>67% or 2/3</td>
<td>33% or 1/3</td>
</tr>
<tr>
<td>67% or 2/3</td>
<td>25% or 3/4</td>
</tr>
<tr>
<td>8% or 1/4</td>
<td></td>
</tr>
</tbody>
</table>

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%)
  - 75% or 750 cc to interstitial space
  - 25% or 250 cc to intravascular space

- 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
    - 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 (euvolemia)
  - 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

<table>
<thead>
<tr>
<th>Extracellular (hypotonic)</th>
<th>Intracellular (hypertonic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( H_2O )</td>
<td>( H_2O )</td>
</tr>
<tr>
<td>( H_2O )</td>
<td>( H_2O )</td>
</tr>
</tbody>
</table>

Cerebral Compensation

- 1\textsuperscript{st} line of "self" defense
  - Increased hydrostatic pressure from cerebral edema causes fluid to shift to CSF, shunted into systemic circulation

- 2\textsuperscript{nd} 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

- 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., hypernatremia)
  - 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

- Mucus membranes
- S3?
- JVP ?
- Rales?
- AMS?*
- Axillary sweat?
- UOP**
- Ascites?
- Edema?
- Skin mottling***
- Skin turgor

**may represent organ involvement

True Hyponatremia

- Hypovolemic hyponatremia
  - Too little too much
- Euvolemic hyponatremia
  - Just the right amount but too much
- Hypervolemic hyponatremia
  - Too much and too much
Diagnosis: Hyponatremia

Hypovolemic

**Extra-renal** volume loss
- Low urine Na
- Diarrhea, fever, vomiting, burns

**Renal** volume loss
- High urine Na
- Diuretics
- Salt wasting nephropathy
- Cerebral salt wasting

Hypovolemic

Euvolemic

**Low** urine Na
- Psychogenic polydipsia
- Beer Potomania

**High** urine Na
- Adrenal insufficiency
- Hypothyroid
- SIADH

Diagnosis: Hyponatremia

- Hypervolemic
  - Low urine Na
    - CHF right or left sided
    - Pulmonary hypertension
    - Hepatic failure
    - Nephrotic syndrome
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), SNa <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

<table>
<thead>
<tr>
<th>SIADH</th>
<th>CSW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euvolemic</td>
<td>Hypovolemic</td>
</tr>
<tr>
<td>Urine Na excretion equals urinary sodium intake</td>
<td>Net negative sodium balance</td>
</tr>
<tr>
<td>Treatment is fluid restriction</td>
<td>Treatment is IV saline</td>
</tr>
</tbody>
</table>

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 Considerations

- Fluid?
- Diuretics?
- Hypertonic?
- Restrict Fluid?
- Combo?

- 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

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

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

Max daily urine vol = \( \frac{\text{daily solute load}}{\text{minimum urine}} \) \left[ \frac{10\text{mmol x kg}}{\text{normal} \sim 50} \right]

Examples: "normal" intake
80 kg man with min urine \( \geq 50 \) can drink 16 L
80 kg man with min urine \( \geq 400 \) can drink 2 L
80 kg man with min urine \( \geq 600 \) can drink 1.3 L
SIADH: Acute Symptomatic Hyponatremia

- Using 3% saline + lasix for SIADH = net aquaresis
  - Goal salt in = salt out (euvoletic 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)

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

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

*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 \( [\text{Na}^+] = 513 \text{ meq/L} \)
  - 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
     • Urine Na⁺ x Urine Volume = meq of ongoing loss

   Example of ongoing losses: UNa 150 (meq/L) x 5 L UOP (750 meq) = ~ 4.8L IVNS or 200cc/hr IVNS

CSW vs. SIADH: Treatment

• Response to isotonic saline
  • SIADH - worsening of hyponatremia
    • Lose the salt, hold on to the H₂O
  • 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

• 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

- 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 ½ 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.

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

What’s the treatment?

1) IVNS bolus 1 liter
2) IVNS 125 cc/hr
3) D5 ½ 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 ½ 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 (SNa > 160) independently associated with increased mortality in NSICU
Hypernatremia

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

Hypernatremia: Associated Mortality

- 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 H\(_2\)O in collecting tubule
  - Excrete concentrated urine
    - High urine Na, High urine Osm, low UOP

- **WITHOUT ADH**
  - Unable to reabsorb H\(_2\)O in collecting tubule
  - Excrete dilute urine
    - Low urine Na, Low urine Osm, high UOP

Hypernatremia

- H\(_2\)O flows **OUT** of cells and cells shrink
- **GOAL**: Appropriate rate of correction, H\(_2\)O flows **IN** and cells return to normal size
- **PROBLEM**: Overly rapid correction of hypernatremia
- **COMPLICATION**: H\(_2\)O flows **IN** cells and cells swell leading to cerebral edema

![Hypernatremia Diagram]

- **Extracellular** (hypertonic)
  - e.g. Serum Na\(^+\) 150

- **Intracellular** (hypotonic)
  - H\(_2\)O flows **IN**
Hypernatremia: Causes

- **Decreased H\(_2\)O intake** (*most common*)
  - Poor access (elderly, altered mental status)
  - Iatrogenic (inadequate water prescription)

- **Increased H\(_2\)O 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 H\(_2\)O problem or is it a salt and a H\(_2\)O problem???
    - Gain of Sodium???
    - Loss of Water?? (i.e. HONK, DI, mannitol)
    - Inadequate water intake???

- **Step 2 – Calculate H\(_2\)O deficit**

Hypernatremia: H\(_2\)O deficit

\[
H_2O \text{ deficit (L)} = \left[ 0.6 \times \text{wt (kg)} \right] \times \left[ \frac{\text{obs Na} - 1}{140} \right]
\]

Example: 80 kg male with serum Na 150

\[
0.6 \times 80 \times (150/140 - 1) \sim 3.4L
\]

+ "insensible" losses (skin, respiratory, GI)
+ "sensible" losses (urine output)
Hyponatremia: H₂O deficit

\[ \text{H₂O deficit (L)} = \left[ 0.6 \times \text{wt (kg)} \right] \times \left[ \frac{\text{obs Na} - 1}{140} \right] \]

Example: 80 kg male with serum Na 145
0.6 x 80 x (145/140 – 1) \approx 1.7L

+ “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
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

• 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 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 euvoelemic 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) There is no such thing!
   d) 70

POST-TEST

8) What is the primary determinant of a person’s volume status (i.e. hypovolemia, euvoolemia, hypervolemia)?
   a) salt
   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) central pontine myelinosis (CPM)
   c) volume overload
   d) none of the above
10) What is the risk of rapidly correcting hypernatremia?

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

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

12) Dehydration refers to which of the following?

a) too little salt
b) too little water
c) neither
d) both
Thanks!

Paula.Dennen@ucdenver.edu