



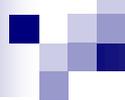
# Abnormal Sodium

National Pediatric Nighttime Curriculum

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# Learning objectives

After this module learners will be able to:

- Describe principles of acute fluid management in the correction of hypernatremia and hyponatremia
- Recognize the signs and symptoms that require immediate attention in patients with disordered sodium
- Consider the level of care appropriate for patients requiring correction of hypernatremia and hyponatremia

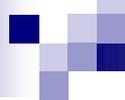
# Case #1 (intern)

- You have just finished sign out and you are reviewing your patient list to prioritize the most ill patients when your pager goes off:
- “Lab called with critical value for patient in 735: sodium 160. Please advise.  
–Kevin”



# You review your sign out...

- 7 month old otherwise healthy male admitted directly from clinic in the late afternoon with gastroenteritis and dehydration. He has had minimal PO intake and decreased urine output.
- Tachycardic and febrile when the admitting team saw him but otherwise stable.
- Overnight plan: floor staff is placing an IV, giving a 20cc/kg NS bolus and will call night team to reassess when complete.



# You head to room 735

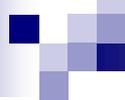
As you go to the bedside to assess the patient, you review some questions:

- What are possible etiologies of hypernatremia?
- What about in this patient specifically?
- What do I need to worry about immediately?
- Should I call my senior?
- Can I take care of this patient on the floor or does he need a higher level of care?

# At the bedside

- VS: T 38.5, HR 120, RR 30, BP 90/60, O2 sat 99% RA
- His nurse, Kevin, tells you that the NS bolus is almost complete and that the patient has been irritable since arriving to the floor
- Physical exam: General: irritable infant; HEENT: mucous membranes dry, anterior fontanelle slightly sunken; Chest: clear; CV: tachycardic, regular rhythm, II/VI systolic ejection murmur; Abdomen: soft, hyperactive bowel sounds; Extremities: normal skin turgor, cap refill 3 seconds
- What is your overall assessment of this patient?

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- What is your next step?
    - A) Stop the bolus—this patient is hypernatremic and NS is an inappropriate fluid choice
    - B) Give another 20cc/kg bolus of NS
    - C) Call a renal consult



# Next steps

You give another normal saline bolus and the patient's perfusion, heart rate and mental status start to normalize

Kevin asks you what fluids you want to hang now....

- What do you need to consider when correcting the sodium in hypernatremic dehydration?
- What do you need to worry about if correcting too fast?

# Calculating free water deficit

- Free water deficit is the minimum amount of fluid necessary to correct serum sodium
- Estimate of free water deficit:  
*4mL x body weight x desired change in sodium*
- Goal is to correct sodium at a rate no faster than 0.5 mEq/L/hour
- Add maintenance fluid needs and account for any ongoing losses



# Ongoing management

- What fluid should you choose?
- When should you recheck a sodium?

# Hypernatremia

- Defined as serum sodium  $\geq 145\text{mEq/L}$

- Causes:

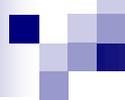
Excess sodium intake	Concentrated formula, salt ingestion (seawater, accidental, Munchausen-by-proxy), hypertonic IV fluids, sodium bicarbonate, blood products
Increased free water losses	<ol style="list-style-type: none"><li>1) Renal: diabetes insipidus, diuretics, tubular disorder</li><li>2) GI: diarrhea, vomiting, colostomy/ileostomy output, malabsorption</li><li>3) Insensible: fever, tachypnea, burns</li></ol>
Decreased free water intake	Ineffective breastfeeding, poor access to water, blunted thirst mechanisms, fluid restriction

# Clinical Manifestations and Evaluation of Hypernatremia

- Early neurologic signs include agitation and irritability → can progress to seizure and coma
- Neurologic exam can reveal increased tone, brisk reflexes and nuchal rigidity
- Lab evaluation can include:
  - Serum osmolarity
  - Serum glucose
  - Urine osmolarity and specific gravity

# Neurologic Sequelae

- In acute phase:
  - Intracellular fluid moves to extracellular space →  
volume loss in brain → separation from meninges
- If hypernatremia has existed for >2-3 days:
  - Neurons protect themselves by making osmolytes to maintain gradient
  - With rapid correction, neurons can swell leading to cerebral edema
- Mortality estimated at 10-16% despite correct rate of rehydration



## Case #2 (senior)

You are doing your late evening rounds on the ward when one of the nurses pulls you aside:

- “One of the post-op orthopedic patients has a sodium of 115 and I can’t reach the primary team. Can you help me?”

# His nurse gives you more info...

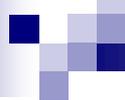
- Patient is a 16yo with cerebral palsy and global developmental delay who is post-operative day #2 from posterior spinal fusion.
- He has been wretching and not tolerating g-tube feeds so has been on maintenance IV fluids of D5 ½NS + 20mEq/L KCl all day.
- His mother is at the bedside and feels he is not himself.

# At the bedside

- VS: T 38.0, HR 90, BP 100/75, RR 20, O2 98%RA
- General: neurologically impaired child moaning in bed, less responsive to voice/touch per mother; HEENT: lips dry, mucous membranes slightly dry; Chest: CTAB; CV: RRR, nl S1, S2; Abdomen: g-tube intact, hypoactive bowel sounds; Extremities: well perfused; Neuro: increased tone and spasticity in extremities, responds to voice with a moan, responsive to painful stimuli

# Next steps

- You initiate a rapid response and transfer to the PICU should happen shortly. Your immediate next step should be:
  - A) Prompt administration of hypertonic saline (3%)
  - B) Emergent head CT
  - C) Fluid restriction due concern for SIADH



# Your patient stabilizes...

Your patient is returning to baseline mental status and you stop the hypertonic saline.

- What general guidelines do you use to think about ongoing fluid management?
- Why are you worried about the rate of correction?

# Hyponatremia

- Defined as serum sodium  $\leq 135$
- Occurs in 3% of hospitalized patients
- Kidney protects against hyponatremia by excreting free water as dilute urine
  - Hyponatremia is an increase in total body water rather than a decrease in serum sodium

# Causes of hyponatremia

Decreased total body water	GI losses (diarrhea, emesis), diuretics, RTA, 3 <sup>rd</sup> spacing
Increased total body water	CHF, acute renal failure, SIADH, water intoxication (dilute formula feeding)
Normal total body water	Hypoglycemia
Pseudohyponatremia	Severe hyperlipidemia or hypoproteinemia

- Hyperglycemia leads to hyperosmolarity with translocation of fluids from intracellular to extracellular space
- Pseudohyponatremia: displacement of plasma water resulting in falsely low serum by laboratory measurement

# Clinical manifestations of hyponatremia

- Neurologic symptoms related to edema caused by hypo-osmolarity
  - Children at higher risk due to higher brain-to-skull ratio
- Symptoms include headache, nausea, emesis, weakness
- Severity worsens as edema increases leading to signs of cerebral herniation
  - Respiratory changes, posturing, pupillary changes, seizure

# Lab evaluation of hyponatremia

- Serum osmolarity if concerned for pseudohyponatremia
- Urine osmolarity to evaluate for impaired ability to excrete free water
- Urine sodium
  - $<25$  mEq/L consistent with volume depletion
  - $>25$  Meq/L consistent with renal tubular dysfunction, SIADH, diuretic use
  - Must be interpreted with caution since affected by IV fluids, fluid restriction, diuretic use

# Fluid management goals

- Hyponatremia with neurologic symptoms is a **medical emergency**

Clinical picture	Fluid	Rate
Seizure	3% hypertonic saline	raise serum sodium by 4-8 mEq/L/hour until seizure activity stops
No seizure activity but not at neurologic baseline	3% hypertonic saline	raise serum sodium by 1mEq/L/hour until: -patient at baseline -plasma sodium increases by 20-25mEq/L <u>OR</u> -serum sodium increases to 125-130mEq/L
Asymptomatic	0.9% normal saline	raise sodium no faster than 0.5 mEq/L/hour

# Why are we concerned about the rate of correction?

- Excessive changes in serum sodium can lead to cerebral demyelination (central pontine myelinolysis)
  - Usually occurs several days after correction
  - Presents with confusion, quadriplegia, confusion or pseudocoma
- Recent data shows rate of correction may have little affect on development of demyelination
  - Magnitude of correction and underlying illness more important contributing factors
- Risk of untreated hyponatremia far exceeds that of rapid correction so **do not hesitate to use hypertonic saline for symptomatic patients**



# Key learning points

- Always prioritize hemodynamic stability over sodium correction
- Correction calculations for both hypernatremia and hyponatremia are general guidelines—sodium should be monitored frequently to ensure safe rate of correction
- Symptomatic hyponatremia is a medical emergency and should be managed in a closely monitored setting with 3% hypertonic saline

# References

- Chung C, Zimmerman D. Hyponatremia and hypernatremia: current understanding and management. *Clin Ped Emerg Med*. 2009; 10: 272-278.
- Moritz M and Ayus JC. Disorders of water metabolism in children: hyponatremia and hypernatremia. *Pediatr Rev*. 2002; 23: 371-380.
- Schwaderer AL, Schwartz GJ. Treating hypernatremic dehydration. *Pediatr Rev*. 2005; 26: 148-150.
- Waseem M, Hussain A. Index of suspicion. *Pediatr Rev*. 2004; 25: 397-399.