


26th Annual General Pediatric Review & Self-Assessment




FLUIDS & ELECTROLYTES

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Fluids and Electrolytes

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Topics to be Discussed

- Metabolic Acidosis
- Metabolic Alkalosis
- Sodium and Water balance
 - Hyponatremia
 - Hypernatremia
- Potassium Imbalance
- Calcium Imbalance

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Metabolic Acidosis

- Net gain of acid
- Loss of base (bicarbonate)
- Know your normals!

First step in determining the etiology of confirmed primary metabolic acidosis is to calculate the ANION GAP


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Metabolic Acidosis

- Serum Anion Gap
 - $\text{Na} - (\text{Cl} + \text{HCO}_3) = 10-14$ (normal)
 - Important to correct for hypoalbuminemia (an unmeasured anion)
- Causes of Positive Anion Gap Metabolic Acidosis:
 - Methanol
 - Uremia
 - Diabetic Ketoacidosis
 - Paraldehyde
 - INH
 - Lactic Acidosis
 - Ethylene Glycol
 - Salicylates

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Non Anion Gap Metabolic Acidosis

- Diarrhea 
 - Colonic bicarbonate loss
- Renal Tubular Acidosis
 - Type 1: Distal: Inability to secrete and excrete acid
 - Type 2: Proximal: Inability to reabsorb bicarbonate
 - Most Common Cause is Cystinosis
 - Type 4: Hyperkalemic: Aldosterone resistance

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Renal Tubular Acidosis

- A hyperchloremic non-anion gap metabolic acidosis
- Urinary Anion Gap (helpful; not diagnostic)
 - Assessment of distal production/secretion of NH_4Cl via random urine electrolytes
 - $[\text{Na} + \text{K}] - \text{Cl}$
 - In distal RTA the urine anion gap is POSITIVE
 - The urine pH is > 5.5
 - This means there is no distal acid secretion
 - In proximal RTA the urine anion gap is NEGATIVE
 - This means distal acid secretion is intact

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Lab Value	Type 1 RTA	Type 2 RTA	Type 4 RTA
Plasma K	Low-normal	Low-normal	Elevated
Urine pH	>5.5	<5.5	Variable
Urine AG	+	---	+
Stones/NC	Yes	No	No
Treatment	Responds	Challenging	Variable

Adapted from Soriano JR. Renal Tubular Acidosis: The Clinical Entity. JASN. 2002

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Metabolic Alkalosis

- Net gain of base
- Loss of acid

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- Common Causes Include:
 - Vomiting (pyloric stenosis) or NG suction
 - Diuretic Use
 - Salt
 - Post
 - Licor
 - Primary hyperaldosteronism
 - Mineralocorticoid excess
 - Cushings Syndrome
 - Liddle Syndrome
 - Excessive blood product transfusion (citrate)

First Step is to order.....
a URINE CHLORIDE!

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Salt Responsive Alkalosis UrCl < 20 mEq/L	Salt Resistant Alkalosis mEq/L	UrCl > 20
Contraction Alkalosis Vomiting/Suction Post-hypercapnea Chronic Diuretic Use (after depletion) Cystic Fibrosis Congenital CL diarrhea	With Normal BP -- Acute diuretic use --- Salt wasting tubulopathy With Elevated BP --- Mineralocorticoid --- Cushing's Syndrome --- Licorice (large amounts) --- Liddle's Syndrome	

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Salt-Water Imbalance

- Again do not let the lab plays tricks on you
 - Adjust for hyperlipidemia, hyperglycemia, hyperproteinemia
- Free Water Deficit Equation:
 - $\frac{Na_{current}}{Na_{desired}} \times 0.6kg - 0.6kg$
- Salt Deficit Equation
 - $0.6kg \times (Desired Na - Actual Na)$

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Hyponatremia Defined

- Definition: Serum Na⁺ <135 mmol/L
 - Associated with decreased measured osmolality to <275
- Usually caused by retention of water
 - A drop in osmolality SHOULD suppress ADH to allow excretion of the excess water via dilute urine
 - Most forms of hyponatremia are associated with elevated ADH (whether appropriate or inappropriate), which concentrates urine (via water reabsorption)

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Signs & Symptoms

- ▣ More profound when the decrease in sodium is very large or occurs rapidly
 - ▣ Generally asymptomatic if Na⁺ level >125 mmol/L
- ▣ Symptoms include:
 - ▣ Headache, nausea, vomiting
 - ▣ Disorientation, depressed reflexes, lethargy, restlessness, seizure, coma
- ▣ Important things to know
 - ▣ Recent fluid intake or losses
 - ▣ Changes in patients weight
 - ▣ Vital signs

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Approach to Hyponatremia

- Assess measured osmolality (via chemistry)
- Assess the patients volume status
- Assess random urinary sodium excretion and FeNa

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Osmolality

- **Hypertonic**
 - hyperglycemia, mannitol, glycerol
 - Check for osmolar gap
- **Isotonic** - 280-295
 - pseudo-hyponatremia from elevated lipids or protein
- **Hypotonic** - <280
 - excess fluid intake, low solute intake, SIADH, hypothyroidism, adrenal insufficiency, CHF, cirrhosis, nephrotic syndrome, etc..
 - This REQUIRES further work up and evaluation

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Volume Status

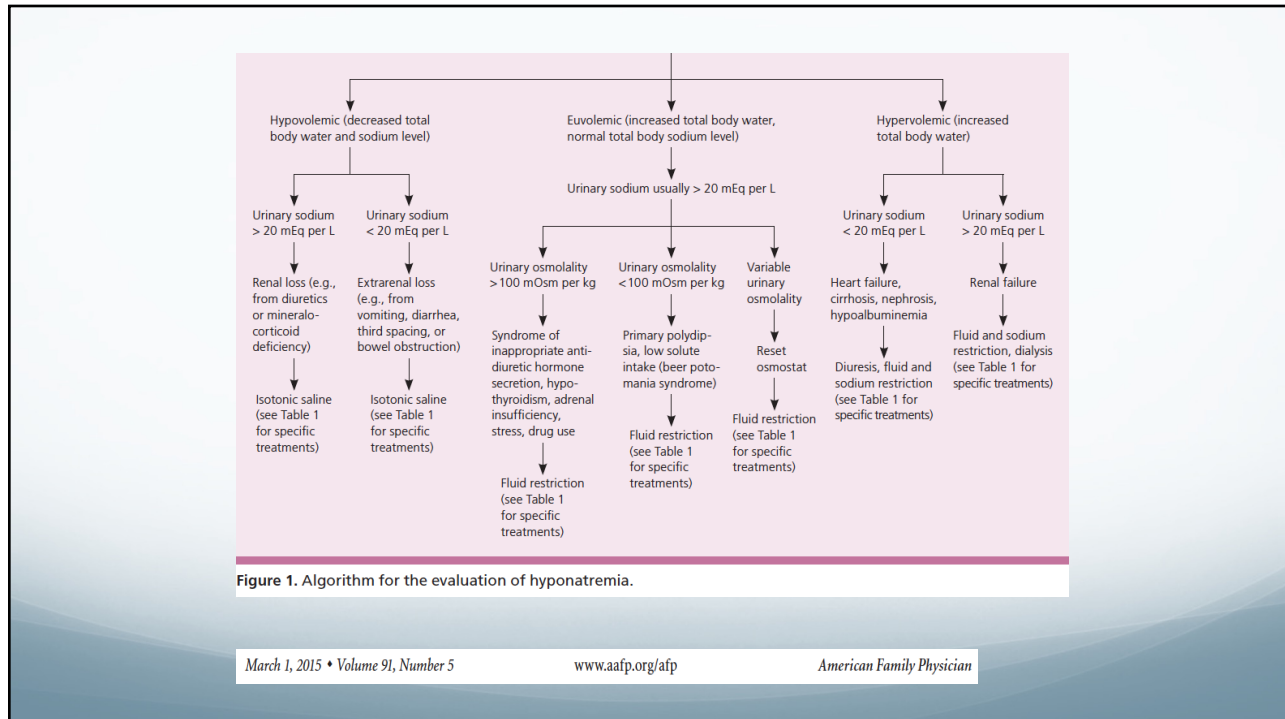
- **Hypotonic hyponatremia** has 3 main etiologies:
 - Hypovolemic – both H₂O and Na decreased (Na > H₂O)
 - Diarrhea, vomiting, dehydration, malnutrition, burns
 - Euvolemic – H₂O increased and Na stable
 - Consider SIADH, thyroid disease, cortisol deficiency, primary polydipsia
 - Hypervolemic – H₂O increased and Na increased (H₂O > Na)
 - Consider CHF, cirrhosis, Renal Failure and Nephrotic Syndrome

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Urine Studies

- For euvolemic hyponatremia, check urine osmolality
 - Urine osmolality <100 - excess water intake
 - Urine osmolality >100 - impaired renal concentration
 - SIADH, hypothyroidism, cortisol deficiency
- Check urine sodium & calculate FeNa %
 - A low urine sodium (<10) and low FeNa (<1%) implies the kidneys are appropriately reabsorbing sodium
 - A high urine sodium (>20) and high FeNa (>1%) implies the kidneys are not functioning as they should

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Treatment of Hyponatremia

- Depending on the cause:
 - Water restriction
 - Saline
 - Saline and Lasix
 - 3% NS IF symptomatic
- Be **CAUTIOUS** with correction:
 - Do not increase Na more than 10-12 meq/L in 24 hrs
 - Central pontine demyelination syndrome

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Hypernatremia

- Serum Na > 150 mmol/L
- Intact thirst and free access to water should prevent this
- Causes include
 - Water losses from kidneys, GI system, skin
 - Weight loss
 - GI losses are the most common cause
 - Salt loading/ high solute load

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Most Important Lab Test

After assessing volume status → Urine Na and Urine osmolality

	Urine Na > 20 mEq/L	Urine Na < 20meq/L
Hypovolemic	Renal losses --Dysplasia --Post-obstructive diuresis	Extrarenal losses --burns, sweat --diarrhea
Hypervolemic	Salt Loading	

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Hypernatremia WITH Euvolemia

- Concentrated Urine
 - Treat with water replacement
- Dilute Urine (Ur osm <300)
 - Diabetes Insipidus
 - Depending on results of water deprivation test
 - If responds → Central DI (treat with DDAVP)
 - No response → Nephrogenic DI (treat with water, thiazides)

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Hypokalemia

- Etiology
 - Urine losses
 - Diuretics
 - Tubular dysfunction (Fanconi Syndrome, Bartter/Gitelman Syndrome)
 - GI losses
 - Diarrhea, NG suction
 - Magnesium depletion
 - Displacement (transcellular shifts)
 - Abnormal RAS pathway (usually associated with elevated BP)
 - Alkalosis
- Manifestations
 - EKG: U waves, flat and inverted T waves
 - Muscle weakness, cramping, paresthesia
 - Paralytic ileus
- Treatment
 - Replace: oral if possible
 - Avoid medications that push K intracellularly

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Hyperkalemia

- Etiology
 - Renal failure
 - Cell lysis /muscle injury
 - Medications (K sparing diuretics, ACEI/ARB, BB, NSAIDs)
 - Metabolic acidosis
 - Adrenal insufficiency
 - Fictitious (heel stick / tourniquet)***
- EKG Findings
 - Wide QRS, peaked T waves, prolonged PR interval

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Hyperkalemia

- Treatment:
 - Calcium → does not treat but stabilizes the myocardium
 - Temporary Treatment/Shifters (fast acting)
 - Triple albuterol neb
 - Insulin (need to give dextrose prior)
 - Bicarbonate
 - Stop K containing IVF/TPN, wash blood products
 - True Removal (these take time)
 - Sodium polystyrene sulfonate (increase GI losses)
 - Diuretics (urine urine K losses +/- fluids)
 - Dialysis

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Hypocalcemia

- Etiology:
 - False; Hypoalbuminemia: corrected calcium:
 - Hypoparathyroidism
 - Transient in neonates (especially infant of diabetic mother)
 - DiGeorge anomaly (22q11)
 - Hypomagnesemia (impairs PTH secretion/CaSR resistance)
 - Vitamin D insufficiency (exclusively breastfed, measure 25OH Vitamin D level)
 - Chelation or precipitation (hyperphosphatemia, tumor lysis syndrome)
 - Alkalosis (increased albumin binding)
- Manifestations:
 - Neuromuscular: tetany, paresthesia, weakness, seizures, muscle spasm.
 - Cardiovascular: depressed myocardial contractility (prolonged QT)
 - Respiratory: laryngo/brochospasm

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Hypocalcemia

- Work Up (prior to treatment)
 - ical
 - CMP (albumin for correction), Phosphorus, Magnesium
 - PTH (appropriate response is for it to be elevated)
 - Vitamin D levels (25OH, 1,25 OH)
- Treatment:
 - Replace (IV is very caustic)
 - May need: Vitamin D or calcitriol (IV or PO)
 - Do not give oral calcium with food (then it becomes a phosphorus binder!)

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Hypercalcemia

- Manifestations:
 - Anorexia, nausea
 - Polyuria (nephrocalcinosis)
 - Hypertension
 - Bone pain
 - Pyschosis
- Work Up
 - Same as hypocalcemia
 - If PTH suppressed there is an “exogenous” source

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Etiology

Suppressed PTH	Normal/Elevated PTH
Neoplasm (PTHrp mediated)	Familial hypocalciuric hypercalcemia (no treatment needed)
Granulomatous Disease (+1,25 VitD)	
Immobility (bone resorption)	Hyperparathyroidism (transient neonatal, primary, tertiary)
Hypervitaminosis A and D	
Fat necrosis (newborns)	

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Hypercalcemia

- Treatment:
 - Volume expansion (induce calciuria)
 - Loop diuretics ONLY (induce calciuria)
 - Calcitonin (SQ) or biphosphonates (IV) (inhibit osteoclastic activity)
 - Dialysis (low calcium bath to induce removal)

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Question 1

- A 5 week old male infant presents to the emergency department with a vomiting x 4 days. His mother states that the vomiting has gotten progressively worse and now seems to "shoot out of his mouth." The vomitus is non-bilious and non-bloody. He is exclusively bottle fed with formula. There is no history of fever, URI symptoms, or diarrhea. He is less active than normal. He is making fewer wet diapers and less stool than usual. You suspect pyloric stenosis. What constellation of labs findings to most expect to find on the BMP in process?
 - A: Hyperkalemic, hypochloremic, metabolic acidosis
 - B: Normokalemic, normochloremic, metabolic acidosis
 - C: Hyperkalemic, hyperchloremic, metabolic acidosis
 - D.: Hypokalemic, hypochloremic, metabolic alkalosis

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 - C: Hyperkalemic, hyperchloremic, metabolic acidosis
 - D.: **Hypokalemic, hypochloremic, metabolic alkalosis**

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Question # 2

- A 10 year old boy presents with new onset bed wetting after previously being dry for the last 5 years. He denies dysuria or hematuria but does have increased urinary frequency and morning headaches. No stooling issues. He has recently started a new school but grades are above average. His vitals are normal. In office UA demonstrates a SG 1.002, no RBC, no WBC, no glucose, no protein
The most likely cause of his new symptoms is:
 - A. UTI
 - B. Stress
 - C. Urine concentration defect
 - D. Constipation

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The most likely cause of his new symptoms is:

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- B. Stress
- C. **Urine concentration defect**
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Questions #3

- Which of the following is most consistent with renal tubular acidosis?
 - A. Low anion gap
 - B. Height above the 75%
 - C. Hyperchloremic
 - D. Hyperphosphatemia

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- Which of the following is most consistent with renal tubular acidosis?
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 - C. Hyperchloremic**
 - D. Hyperphosphatemia

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Question #4

- A mother brings her 6 month old exclusively breastfed boy to the office for constipation and extreme fussiness. The mother has not had any recent diet changes and the infant does not have a fever, emesis or recent exposure to a sick contact. The mother states she recently has started giving Vitamin D to avoid him getting sick during the Flu season. What lab findings to you expect to see in this patient?
 - A: Hypercalcemia, high PTH, high 25 Vitamin D
 - B: Hypercalcemia, low PTH, high 25 Vitamin D
 - C: Normocalcemic, normal PTH, high 25 Vitamin D
 - D: Hypocalcemic, high PTH, high 25 Vitamin D

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Question #4

- A mother brings her 6 month old exclusively breastfed boy to the office for constipation and extreme fussiness. The mother has not had any recent diet changes and the infant does not have a fever, emesis or recent exposure to a sick contact. The mother states she recently has started giving Vitamin D to avoid him getting sick during the Flu season. What lab findings to you expect to see in this patient?
 - A: Hypercalcemia, high PTH, high 25 Vitamin D
 - B: **Hypercalcemia, low PTH, high 25 Vitamin D**
 - C: Normocalcemic, normal PTH, high 25 Vitamin D
 - D: Hypocalcemic, high PTH, high 25 Vitamin D

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Thank You!

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