Lab Values
- Complete Blood Count, Blood Culture, Reticulocyte Count, Zinc protoporphyrin/heme ratio, Liver Function Tests, Thyroid Function, Blood Sugar, Alkaline Phosphatase, Albumin, Vitamin Levels, CSF Studies, etc.

Most Common Lab
- Electrolytes

Objectives
- Discuss physiologic changes in fluid and electrolyte balance in the neonatal period
- Discuss management of common fluid and electrolyte imbalances

Fluid and Electrolytes
- Transition from fetal to neonatal life creates major changes in fluid and electrolyte homeostasis
- Before birth fluid and electrolyte balance is mainly a maternal and placental function
- After birth newborns assume responsibility for their own fluid and electrolyte balance
Fetal Water Balance
- Water moves from maternal to fetal circulation across the placenta
- The fetus is dependent on the placental route for the majority of its water needs

Fetal Mineral Balance
- Minerals found in low concentrations in plasma are actively transported across placenta
  - K, Ca, Mg, phosphate
- Ions found in extracellular compartment are transported actively and passively
  - Na, Cl
- Sodium flux to the fetus is 10-100 times higher than fetal needs, most Na is returned to the mother by paracellular diffusion.

Preterm Infants and Renal Function
- GFR is diminished in the neonate
- Renal blood flow is decreased
- Not all glomeruli are functional
- Smaller glomerular pore size

Glomerular Filtration Rate
<table>
<thead>
<tr>
<th>Normal GFR mL/min/1.73m²</th>
<th>1 week</th>
<th>2-8 weeks</th>
<th>&gt; 8 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>25-32 weeks</td>
<td>11 ± 5.4</td>
<td>15.5 ± 6.2</td>
<td>47.4 ± 21.5</td>
</tr>
<tr>
<td>29-34 weeks</td>
<td>15.3 ± 5.6</td>
<td>28.7 ± 13.9</td>
<td>51.4 ± 21.7</td>
</tr>
<tr>
<td>38-42 weeks</td>
<td>40.6 ± 14.8</td>
<td>65.8 ± 24.8</td>
<td>95.7 ± 21.7</td>
</tr>
</tbody>
</table>

Water Balance
- Weight loss in first week of life 5% – 15%
- Contraction of the ECF space after birth
- Large increases in water and sodium intake can attenuate the contraction
- Higher intakes of sodium and water are associated with increase incidences of PDA, NEC, prolonged oxygen dependence, and BPD

Sodium
- Term infants conserve sodium effectively
- FE_{Na} is increased in the first hours of life
- Capacity to excrete a sodium load in term infant is limited
  - High renin and aldosterone levels
  - Increased capillary leak
  - Decreased GFR
Sodium
- Preterm infants do not conserve Na effectively
- Preterm infants waste Na in all segments of the nephron
- Proximal and distal tubules, and collecting duct
- Maturation of epithelial Na channels (ENaC) enhances Na reabsorption
- Positive sodium balance not achieved until 32 weeks gestation

ECF Contraction
- Sodium conservation will improve if ECF is allowed to contract.
- ECF expansion promotes loss of Na, Ca, PO4, glucose, and HCO3 in the urine

Potassium
- Serum potassium rises in the first 24 – 72 hours in premature infants
- Increased potassium results from K shifting from ICF space to ECF space
- Relative insensitivity to aldosterone in preterm infants
- Elevation in potassium does not usually occur after 30 – 32 weeks gestation.

Nonoliguric Hyperkalemia
- Hyperkalemia defined as plasma K > 5 mMol/L or serum K > 5.5 mMol/L
- Shift of K from the ICF to the ECF
- Occurs in 25% - 50% of infants < 1000 grams
- Usually occurs in the first 72 hours of life

Nonoliguric Hyperkalemia
- Clinical signs are rare with serum K < 6.5 mMol/L
- Clinical effects potentiated by hypocalcemia
- Cardiac arrhythmias can occur in 60% of preterm infants with hyperkalemia

Hyperkalemia
- Prompt treatment indicated for any infant with serum K > 7 mMol/L or infant with electrocardiographic changes
Nonoliguric Hyperkalemia Treatment

- Calcium – indicated for infants with ECG changes
- Insulin + glucose – effectively lowers serum K+
  - Stimulates membrane bound Na+K+ ATPase
- β agonist:
  - Stimulates membrane bound Na+K+ ATPase
  - RCT of inhaled albuterol in neonates
    - 400 mcg in 2 mL given q2h for max of 12 doses
    - Lowered K from ~7 meq/L to 6.34 then 5.93 meq/L

Prediuretic Phase

- First 12 to 48 hours urine flow rate is low regardless of intake
- Excretion of Na and K is low
- Insensible water loss is major route of water loss
- Low GFR limits infant’s ability to excrete water and electrolyte loads

Diuretic/Natriuretic Phase

- Usually occurs abruptly
- Increase in urinary water and Na independent of water and Na intake
  - Indicates contraction of the ECF space
  - Sodium rises initially because water balance is more negative
  - Majority of body weight loss during this phase

Diuretic/Natriuretic Phase

- Fall in plasma K+ due to increased delivery of water and Na to distal nephron stimulates K secretion
- ECF space stabilizes at a reduced volume
  - Urinary water and electrolyte excretion decreased and begins to vary appropriately with intake
  - Onset of diuresis is delayed in infants with RDS
  - Marked delay in diuresis is a risk factor for BPD

Basic Principles for Fluid and Electrolyte Therapy

- Allow physiological contraction of ECF space
  - Fluid restriction
  - Na+ and K+ intake should be limited until after the diuresis is well established
- Anticipate and prevent
  - Minimize insensible water loss
  - Monitor carefully labs, weight, urine output

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Classroom 40577
Case Study 1

- Jane, a 650 gram 28-week infant born via cesarean section for maternal preeclampsia. On CPAP in an incubator with 50% humidity. IV fluids started with D5W at 140 mL/kg/day.
- 12 hours of age plasma Na is 128, urine output 1.1 mL/kg/hour

Questions

- Why is Jane hyponatremic?
- What are the risks of hyponatremia?
- What fluid and sodium intake should Jane receive?

Hyponatremia

- Plasma Na is determined by total body water (TBW) volume and total body Na content.
- Na may be low because TBW is increased or TBNa is low (could be combination of both)

Questions

- Why is Jane hyponatremic?
- What are the risks of hyponatremia?
- What fluid and sodium intake should Jane receive?

Risks of Hyponatremia

- Acute decrease in ECF osmolality causes water to move from ECF space to the ICF space, this can result in cerebral edema
- Acute hyponatremia may cause vomiting, seizures, impaired consciousness, brain stem herniation, long-term neurodevelopmental disability, or death
- In newborns cerebral palsy and sensorineural hearing loss have been associated with hyponatremia

Questions

- Why is Jane hyponatremic?
- What are the risks of hyponatremia?
- What fluid and sodium intake should Jane receive?
**Fluid and Sodium Intake**

- Jane should be fluid restricted (60 – 90 mL/kg/day) at least until the onset of diuresis.
- Because the likely cause is positive TBW balance not low TBNa no sodium intake is required.

**Hyponatremia Correction**

- Na should be followed every 8 hours to ensure it is rising but not too rapidly.
- Rapid correction can cause osmotic demyelination syndrome which can result in death or long term neurodevelopmental disability.
- Chronic, asymptomatic hyponatremia should be corrected even more slowly.

**Fluid Recommendations for Preterm Infants first 3 – 5 days**

<table>
<thead>
<tr>
<th>Weight</th>
<th>Weight Loss</th>
<th>Water mL/kg/d</th>
<th>Na</th>
<th>Cl</th>
<th>K</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1000 g</td>
<td>10%</td>
<td>90 - 140</td>
<td>0 - 1</td>
<td>0 - 1</td>
<td>0</td>
</tr>
<tr>
<td>1001 – 1500 g</td>
<td>8 – 10%</td>
<td>80 - 120</td>
<td>0 - 1</td>
<td>0 - 1</td>
<td>0 - 1</td>
</tr>
<tr>
<td>1501 – 2000 g</td>
<td>6 – 8%</td>
<td>70 - 100</td>
<td>0 - 1</td>
<td>0 - 1</td>
<td>0 - 1</td>
</tr>
<tr>
<td>&gt;2000 g</td>
<td>6 – 8%</td>
<td>60 - 80</td>
<td>0 - 1</td>
<td>0 - 1</td>
<td>0 - 1</td>
</tr>
</tbody>
</table>

**Case Study 2**

- James is a 4 day old 29 week infant delivered NSVD precipitously. Apgar scores 3 at 1 minute, 5 at 5 minutes and 7 at 10 minutes. On low ventilator settings.
- Plasma Na 155 mmol/L with normal neurologic status.

**Questions**

- Why is this baby hypernatremic?
- What are the risks of hypernatremia?
- What fluid and sodium intake should James receive today?
Hypernatremia

- TBW is decreased or TBNa increased or combination of both
- Decrease in James ECF volume does not seem excessive
- Infant received significant amounts of Na over first 3 days
- Plasma sodium increase from DOL2 to DOL3 likely due to water loss and positive Na balance
- Administration of sodium bicarb compounded the problem

Questions

- Why is this baby hypernatremic?
- What are the risks of hypernatremia?
- What fluid and sodium intake should James receive today?

Hypernatremia Risks

- Distribution of water between ECF and ICF space determined by ECF osmolality
- Increased ECF osmolality (here due to elevated Na) causes water to move from ICF space to ECF space
- Problematic in the brain where ICF volume may result in neurologic abnormalities – high pitched cry, irritability, seizures, impaired consciousness, intracranial hemorrhage, cerebral infarction, long term neurodevelopmental disability, or death

Questions

- Why is this baby hypernatremic?
- What are the risks of hypernatremia?
- What fluid and sodium intake should James receive today?

Hypernatremia Treatment

- Eliminate sodium intake
- Further decrease in TBW should be prevented
- Consider increasing TBW – especially if there were neurologic symptoms
- Follow neurologic status closely and reassess labs in 6 – 8 hours
- Rapid fall in Na can cause cerebral edema

Questions

- Why is this baby hypernatremic?
- What are the risks of hypernatremia?
- What fluid and sodium intake should James receive today?

Case Study 3

- Cody born at 24 wks for placental abruption. Did not receive antenatal steroids. Apgars 1/3/5. Intubated, PPV, compressions, NS bolus. Birth weight 680 grams. 1st hematocrit 30% – PRBCs x2, 7u hct 45%. Mechanical ventilation. During 1st day of life received 172 mL/kg/day of fluid intake (not including bolus and PRBCs) without Na or K. There has been no urine since birth.
Case Study 3 continued

- Current weight is 623 grams now at 28 hours of age. HR, BP, and perfusion are normal. Plasma sodium went from 135 at 8 hours to 146 mmol/L at 28 hours. Plasma K values at 12, 24, and 28 hours of age were 6.2, 7.2, and 7 mmol/L. Plasma creatinine 1.1mg/dL at 12 hours, and 1.3mg/dL at 24 hours. Blood gas 7.36/39/70 and base excess of -2.7. Ionized calcium 1.23.

Case 3 Questions

- Is this baby in acute renal failure?
- How should his renal failure be treated?
- Is this nonoliguric hyperkalemia?
- Should his hyperkalemia be treated, and if so how?
- What adjustments should be made in fluid and electrolyte administration?

Acute Renal Failure?

- Plasma creatinine is still a function of maternal creatinine at this age
- Best indication for ARF in the 1st weeks of life is change in plasma creatinine over time
- Rate of change of plasma creatinine depends on what is normal GFR
  - Rise in creatinine of 0.1 – 0.3 in the first few days can be normal in a very preterm infant

Normal Creatinine Values

<table>
<thead>
<tr>
<th>Gestational Age</th>
<th>1 week</th>
<th>2-8 weeks</th>
<th>&gt; 8 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>25-28 weeks</td>
<td>1.4 ± 0.9</td>
<td>0.9 ± 0.6</td>
<td>0.4 ± 0.2</td>
</tr>
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<td>29-34 weeks</td>
<td>0.9 ± 0.3</td>
<td>0.7 ± 0.3</td>
<td>0.3</td>
</tr>
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<td>35-42 weeks</td>
<td>0.5 ± 0.1</td>
<td>0.4 ± 0.1</td>
<td>0.4 ± 0.1</td>
</tr>
</tbody>
</table>

Prerenal ARF

- Hypovolemia, decreased effective blood volume, decreased cardiac output
- May result in intrinsic renal failure if cause is not treated promptly
Intrinsic Renal Failure
- Due to renal parenchymal maldevelopment or injury

Postrenal failure
- Due to congenital obstruction of the urinary tract

Acute Renal Failure?
- Infant appears normovolemic now and history of hypovolemia due to acute blood loss (maternal abruption) likely intrinsic acute renal failure.

Case 3 Questions
- Is this baby in acute renal failure?
- How should his renal failure be treated?
- Is this nonoliguric hyperkalemia?
- Should his hyperkalemia be treated, and if so how?
- What adjustments should be made in fluid and electrolyte administration?

Prerenal ARF Treatment
- NS bolus – unless volume expansion is strongly contraindicated
- No response to NS, can give furosemide
- If no response to NS or furosemide with a catheter in place can start low dose dopamine at 0.5 – 5 mcg/kg/min

Intrinsic Acute Renal Failure Treatment
- Observe for polyuric phase of ARF – may need to increase Na intake to replace urinary losses as well as an increase in total fluids
- Goal is to administer fluid and electrolytes to maintain 85 – 90% of birth weight and normal electrolyte values until tubular function recovers
Case 3 Questions

- Is this baby in acute renal failure?
- How should his renal failure be treated?
- Is this nonoliguric hyperkalemia?
- Should his hyperkalemia be treated, and if so how?
- What adjustments should be made in fluid and electrolyte administration?

Case Study 3 continued

- Current weight is 623 grams now at 28 hours of age. HR, BP, and perfusion are normal. Plasma sodium went from 135 at 8 hours to 146 mmol/L at 28 hours. Plasma K values at 12, 24, and 28 hours of age were 6.2, 7.2, and 7 mmol/L. Plasma creatinine 1.1mg/dL at 12 hours, and 1.3mg/dL at 24 hours. Blood gas 7.36/36/70 and base excess of -2.7. Ionized calcium 1.23.

Nonoliguric Hyperkalemia

- Infant is anuric, so by definition not nonoliguric hyperkalemia
- The infant has not had urinary K loss as a result of ARF
- Elevate K is likely the result of the ICF to ECF shift of K – infant has also not received any exogenous K (PRBC transfusions should not increase K significantly)

Hyperkalemia

- Prompt treatment indicated for any infant with serum K > 7 mEq/L or infant with electrocardiographic changes

Nonoliguric Hyperkalemia

- Hyperkalemia defined as plasma K > 5 mMol/L or serum K > 5.5 mMol/L
- Shift of K from the ICF to the ECF
- Occurs in 25%-50% of infants < 1000 grams
- Usually occurs in the first 72 hours of life

Case 3 Questions

- Is this baby in acute renal failure?
- How should his renal failure be treated?
- Is this nonoliguric hyperkalemia?
- Should his hyperkalemia be treated, and if so how?
- What adjustments should be made in fluid and electrolyte administration?
Hyperkalemia Treatment

- Ionized calcium is normal – bolus not indicated now
- Insulin + glucose should be administered
- β agonist – use caution with ARF
- Na HCO\(_3\) – not indicated
- Sodium polystyrene sulfonate (Kayexalate)
  - Ion exchange resin (Na for K) may cause or exacerbate expansion of ECF space or hypernatremia
  - Extremely hyperosmolar, associated with development of intragastric masses and intestinal perforation

Case 3 Questions

- Is this baby in acute renal failure?
- How should his renal failure be treated?
- Is this nonoliguric hyperkalemia?
- Should his hyperkalemia be treated, and if so how?
- What adjustments should be made in fluid and Na administration?

Case Study 3 continued

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- Plasma creatinine 1.1mg/dL at 12 hours, and 1.3mg/dL at 24 hours. Blood gas 7.36/39/70 and base excess of -2.7. Ionized calcium 1.23.

Fluid and Electrolyte Adjustments

- Weight loss of 8.4% indicates that intake is less than IWL
- Total fluid should be increased, currently at ~180 mL/kg/day, increase to ~200 mL/kg/day
- No sodium should be administered
- Follow weight, output, and electrolytes every 6 – 8 hours

Case Study 4

- Cody is now 90 days old (~36 wks) with chronic lung disease after a prolonged mechanical ventilation course. He is on 160 mL/kg/day of breast milk 20 kcal/oz. He has been on daily chlorothiazide for 2 weeks. He is on HFNC at 5 Lpm. In the last week he has gained 220 grams. His weekly electrolytes were Na of 135 mmol/L 2 weeks ago and 134 mmol/L 1 week ago.
- Todays labs by heel stick: Na 128, K\(^+\) 3.5, Cl\(^-\) 82, HCO\(_3\) 35, BUN 15, creatinine 0.4, pH 7.46, PCO\(_2\) 54, HCO\(_3\) 39, base excess +12
Case Study 4 Questions
- What is the cause of the hyponatremia?
- How should the hyponatremia be treated?
- Is the metabolic alkalosis compensatory for the respiratory acidosis?
- How should the metabolic alkalosis be treated?

Hyponatremia
- Diuretic therapy is the cause
- Hyponatremia results from low plasma Na or high TBW, or both
- More common to occur with thiazide diuretics than loop diuretics – thiazides decrease sodium reabsorption in the distal tubule

Hyponatremia Treatment
- Restrict free water intake – in this case 140 mL/kg/day of 24 kcal/oz feeds would be appropriate
- Consider decreasing or discontinuing diuretic
- Consider supplementation with Na

Case Study 4
- Todays labs by heel stick: Na+ 128, K+ 3.5, Cl- 82, HCO3- 35, BUN 15, creatinine 0.4, pH 7.46, PO2 54, HCO3- 39, base excess +12
Metabolic Alkalosis
- Partly a compensation for respiratory acidosis, also a primary metabolic alkalosis from K+ depletion.
- Metabolic Alkalosis is caused by a gain of base or loss of acid from the ECF space, resulting in the generation of bicarbonate.
- K depletion can increase renal HCO₃⁻ reabsorption in the distal tubule.
- Metabolic alkalosis can cause renal K secretion in the distal tubule.

Case Study 4 Questions
- What is the cause of the hyponatremia?
- Should the hyponatremia be treated, and how?
- Is the metabolic alkalosis compensatory for the respiratory acidosis?
- Should the metabolic alkalosis be treated, and how?

Todays labs by heel stick: Na⁺ 128, K⁺ 3.5, Cl⁻ 82, HCO₃⁻ 35, BUN 15, creatinine 0.4, pH 7.46, PCO₂ 54, HCO₃⁻ 39, base excess +12

Metabolic Alkalosis Treatment
- Therapy should be directed at the underlying cause, rather than correcting the metabolic alkalosis.
- In this case the cause is in part K depletion – if this becomes severe can cause ventricular ectopy.
- KCl supplementation should correct the alkalemia.

Questions
- tiffanystanley@gmail.com
“The dumbest kidney in the tiniest preterm infant is smarter than the smartest clinician”

-Richard Polin

References