NUTRITION MANAGEMENT OF INFANTS WITH RENAL DISEASE

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OBJECTIVES

* To learn unique nutrition requirements of premature infants

* To review causes of CKD in newborns

* To apply pediatric renal nutrition goals in the setting of premature infants/term infants with CKD
Meet Rosy!
NICU BASICS

Newborn classifications
Anthropometric goals
Growth charts
Classifications

**Gestational Age**
- Premature: <37 wks
- Term: 37 – 42 wks
- Post-term: >42 wks

**Birth Weight**
- LBW: <2500 g
- VLBW: <1500 g
- ELBW: <1000 g

**Weight for Age**
- SGA: <10^{th} percentile
- AGA: 10^{th} to 90^{th} percentile
- LGA: >90^{th} percentile
Anthropometric Goals

Premature

- 15-20 g/kg/day
- .8-1.1 cm/week (length)
- .5-.6 cm/week (HC)

Term

- 20 – 30 g/day
- .69-.75 cm/week (length)
- .5 cm/week (HC)
Fenton Growth Chart (Girls)
Fenton Growth Chart (Boys)
Plot according to postmenstrual age (PMA) on Fenton chart – PMA is the birth gestational age + the chronological age.

- Can switch over to WHO chart at term or 40 wks
- Plot on WHO chart using corrected age (CA) - chronological age minus the number of weeks/months born before 40 weeks
- Current recommendations are to plot using corrected age until age 3
Growth charts

* Baby born at 28 wks that is now 8 wks old – plot on Fenton at 36 wks PMA.
* Same baby who is now 5 months old: 40 – 28 = 12 wks (or 3 months) premature.
* 5 months – 3 months = corrected age of 2 months.
TYPICAL COMORBIDITIES IN THE NICU

• Respiratory
• Necrotizing Enterocolitis
• Osteopenia of Prematurity
• Patent Ductus Arteriosus
Typical Comorbidities in the NICU

**Respiratory**

* Immature lungs
* Surfactant not produced until 35 wks gestation
* Severe forms: BPD or CLD
* **Nutrition interventions:**
  * High kcals: up to 180 kcals/kg
  * Fluid restriction
  * Electrolyte derangement due to diuretics and bronchodilators

**Necrotizing Enterocolitis**

* Acquired gastrointestinal disease ranging from mild (feeding intolerance) to severe (necrotic bowel with or without perforation)
* **Nutrition interventions:**
  * TPN
  * Semi-elemental or elemental formulas may be needed
## Typical Comorbidities in the NICU

### Osteopenia of Prematurity

- Reduced bone mass
- Maximum accretion of bone minerals occurs during 3\textsuperscript{rd} trimester
- **Nutrition interventions:**
  - Very high Ca and Phos needs
  - Cannot be supplied when on TPN
  - Typical labs: low phos and high alk phos

### Patent Ductus Arteriosus

- Fetal circulatory pathway is called Ductus Arteiosus
- Diverts blood from lungs to aorta in fetus
- If it remains open after birth it is called PDA and leads to left to right shunting, pulmonary distress.
- **Nutrition Interventions:**
  - Fluid restriction
  - Feeding concentration
PREMATURE NUTRIENT REQUIREMENTS
### NORMAL NUTRIENT NEEDS FOR PREMATURE INFANTS

<table>
<thead>
<tr>
<th></th>
<th>PARENTERAL</th>
<th>ENTERAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calories</td>
<td>90-100 kcals/kg</td>
<td>110-150 kcals/kg*</td>
</tr>
<tr>
<td><em>120 kcals/kg is typical initial goal</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>3.2-4.0 g/kg</td>
<td>3.4 – 4.4 g/kg</td>
</tr>
<tr>
<td>Sodium</td>
<td>3-5 meq/kg/d</td>
<td>3-5 meq/kg/d</td>
</tr>
<tr>
<td>Potassium</td>
<td>2-3 meq/kg/d</td>
<td>2-3 meq/kg/d</td>
</tr>
<tr>
<td>Calcium</td>
<td>60-80 mg/kg/d</td>
<td>100 – 220 mg/kg/d</td>
</tr>
<tr>
<td>Term: 210 mg/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phosphorous</td>
<td>45-60 mg/kg/d</td>
<td>60 – 140 mg/kg/d</td>
</tr>
<tr>
<td>Term: 100 mg/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fluid</td>
<td>90-180 mls/kg/d</td>
<td>90-220 mls/kg/d*</td>
</tr>
<tr>
<td><em>150 mls/kg is typical goal</em></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Tsang 2005
Protein Needs by Gestational Age

Adapted from Tsang, et al 2005

For a complete listing of all nutrient requirements recommended see Tsang, et al 2005

<table>
<thead>
<tr>
<th>Gestational Age</th>
<th>Protein (g/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>26 - 30 weeks</td>
<td>3.8 – 4.4</td>
</tr>
<tr>
<td>30 - 36 weeks</td>
<td>3.4 – 4.2</td>
</tr>
<tr>
<td>36 – 40 weeks</td>
<td>2.8 – 3.4</td>
</tr>
</tbody>
</table>
In General, Nutrient Needs are Higher for Premature Infants. Goal is to Provide Nutrient Concentrations that will match Fetal Accretion Rates.
Typical Feeding Choices
FEEDING OPTIONS FOR PREMIES

HUMAN MILK

- Human Milk Fortifiers
  - Similac HMF
  - Enfamil HMF
  - Prolacta
- Meant for NICU stay only
- Adds 1-4 kcals/oz

- Primary purpose is to increase nutrient density of human milk (Protein, Ca, Phos, etc)
FEEDING OPTIONS FOR PREMIES

PRESMATURE FORMULAS

- Similac Special Care
  - Ready to Feed only
  - 20, 24 and 30 kcals/oz
- Enfamil Premature
  - Ready to Feed only
  - 20 and 24 kcals/oz
- Meant for NICU stay only
- Only on rare occasions are babies discharged on these formulas
- Trend currently is to use donor milk
Feeding Options for Premies

Transitional Formulas

* Enfacare and Neosure
* Lower concentration of nutrients than Premature Formulas or Fortified Human Milk

**BUT**

* Higher concentration of nutrients than Term Formulas or Human Milk
* Meant for discharge to home and up to 9 months corrected age
### COMPARISON OF PREMATURE, TRANSITIONAL, AND TERM FORMULAS

<table>
<thead>
<tr>
<th>Per 100 kcals</th>
<th>Sim Spec Care 24 HP</th>
<th>Sim HMF HP + Human Milk</th>
<th>Neosure</th>
<th>Sim Adv</th>
<th>PM 60/40</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein</td>
<td>3.3</td>
<td>3.58</td>
<td>2.8</td>
<td>2.07</td>
<td>2.2</td>
</tr>
<tr>
<td>Na (mEq)</td>
<td>1.9</td>
<td>2</td>
<td>1.4</td>
<td>1.1</td>
<td>1</td>
</tr>
<tr>
<td>K (mEq)</td>
<td>3.3</td>
<td>3.6</td>
<td>3.6</td>
<td>2.8</td>
<td>2.1</td>
</tr>
<tr>
<td>Ca (mg)</td>
<td>180</td>
<td>152</td>
<td>105</td>
<td>82</td>
<td>56</td>
</tr>
<tr>
<td>Phos (mg)</td>
<td>100</td>
<td>85</td>
<td>62</td>
<td>44</td>
<td>28</td>
</tr>
</tbody>
</table>
Nephrogenesis and Prematurity

* Nephrogenesis starts 22 days after conception and start function at 37 days.
* Continues until 36 wks gestation-then it’s over!
* 60% of nephrons are formed during the 3\textsuperscript{rd} trimester
* Autopsy studies have shown a very strong correlation between BW, glomerular number and glomerular size
Brenner Hypothesis

* Barry Brenner expanded on David Barker’s Fetal Origins of Disease concept and applied it to CKD.

* Terminology uniquely describing idea of reduced nephron mass include:
  * Oligomeganephronia
  * Oligonephropathy
  * Renal hypoplasia
  * Low nephron number
Brenner Hypothesis

Diagram of the Brenner Hypothesis showing the progression from prematurity and low birth weight (LBW) to reduced nephron number (Oligonephropathy) and eventually to chronic kidney disease (CKD). The diagram includes steps such as hyperfiltration, glomerular hypertension, systemic hypertension, proteinuria, and nephron loss due to glomerulosclerosis.
Rodriquez, 2004

- Found in a series of 56 very preterm babies that nephrogenesis stops 40 days after birth
- Critical window of time after birth for final nephrogenesis spans between 32-35 wks gestation
- If any kidney insult occurs during this time it will affect the potential for further nephron numbers
- Incidence of AKI during this period is high (8-24%)
Incidence of AKI

- Carmody JB et al. 2014. Recognition and reporting of AKI in very low birth weight infants.
  - Evaluated incidence of AKI from 2008-2011
  - 455 infants; Gestational age range: 22-37 wks
  - BW ranged from 370-1495 g
  - Found **39.8%** experienced AKI
    - 16.5% with multiple episodes
  - Inclusion of AKI in discharge summary infrequent
  - No referrals to pediatric nephrologist
Consequences

Franke D. et al, 2010
* 435 children with CKD
* Prevalence of SGA was 3 X higher in children with CKD compared to normal controls
* 1/3 of children with CKD born premature

Greenbaum et al, 2011
* Analysis of CkiD study
* 400 children with CKD
* 17% were LBW
* 14% were SGA
* 40% were in a NICU
* LBW and SGA higher incidence of short stature
Rosy goes home
RENAL DISEASE IN NEONATES

Acute Kidney Injury (AKI)
<table>
<thead>
<tr>
<th>Study</th>
<th>Study Population</th>
<th>AKI Definition</th>
<th>Number of Infants</th>
<th>AKI Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viswanathan et al&lt;sup&gt;141&lt;/sup&gt;</td>
<td>Extremely LBW (&lt;1000 g)</td>
<td>Serum creatinine ≥1.5 mg/dL or urine output &lt;1 mL/kg/h</td>
<td>472</td>
<td>12.5%</td>
</tr>
<tr>
<td>Koralkar et al&lt;sup&gt;142&lt;/sup&gt;</td>
<td>Very LBW (&lt;1500 g)</td>
<td>AKIN&lt;sup&gt;88&lt;/sup&gt;</td>
<td>229</td>
<td>18%</td>
</tr>
<tr>
<td>Selewski et al&lt;sup&gt;143&lt;/sup&gt;</td>
<td>Asphyxiated newborns undergoing therapeutic hypothermia</td>
<td>AKIN</td>
<td>96</td>
<td>38%</td>
</tr>
<tr>
<td>Kaur et al&lt;sup&gt;144&lt;/sup&gt;</td>
<td>Infants ≥34 wk gestation with asphyxia (Apgar &lt;7 at 1 min after birth)</td>
<td>AKIN</td>
<td>36</td>
<td>41.7%</td>
</tr>
<tr>
<td>Blinder et al&lt;sup&gt;145&lt;/sup&gt;</td>
<td>Infants &lt;90 d old with congenital heart disease undergoing surgery</td>
<td>AKIN</td>
<td>430</td>
<td>52%</td>
</tr>
<tr>
<td>Gadepalli et al&lt;sup&gt;146&lt;/sup&gt;</td>
<td>Infants with congenital diaphragmatic hernia requiring extracorporeal membrane oxygenation</td>
<td>RIFLE&lt;sup&gt;47&lt;/sup&gt;</td>
<td>68</td>
<td>71%</td>
</tr>
</tbody>
</table>

Box 1.
Proposed neonatal AKI classification


<table>
<thead>
<tr>
<th>Stage</th>
<th>SCr</th>
<th>Urine Output</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No change in SCr or increase &lt;0.3 mg/dL</td>
<td>≥0.5 mL/kg/h</td>
</tr>
<tr>
<td>1</td>
<td>SCr increase ≥0.3 mg/dL within 48 h or SCr increase ≥1.5–1.9 × reference SCr&lt;sup&gt;a&lt;/sup&gt; within 7 d</td>
<td>&lt;0.5 mL/kg/h for 6–12 h</td>
</tr>
<tr>
<td>2</td>
<td>SCr increase ≥2 to 2.9 × reference SCr&lt;sup&gt;a&lt;/sup&gt;</td>
<td>&lt;0.5 mL/kg/h for ≥12 h</td>
</tr>
<tr>
<td>3</td>
<td>SCr increase ≥3 × reference SCr&lt;sup&gt;a&lt;/sup&gt; or SCr ≥2.5 mg/dL or Receipt of dialysis</td>
<td>&lt;0.3 mL/kg/h for ≥24 h or anuria for ≥12 h</td>
</tr>
</tbody>
</table>

<sup>a</sup> Baseline SCr is defined as the lowest previous SCr value.

AKI

- Prerenal
- Intrinsic
- Postrenal

Remember that AKI can occur in both non-CKD babies as well as CKD babies
* Usually due to inadequate renal perfusion
  * Dehydration
* Kidney is intrinsically normal
  * But remember nephrogenesis not complete if premature
* Most common reason for AKI in NICU
* Can lead to CKD if not corrected: ATN and/or acute cortical necrosis with scarring
AKI INTRINSIC

- ATN from ischemic/hypoxic events
- Drug Induced - Gentamicin, Tobramycin Ibuprofen, Indomethacin
- Vascular Insults – Renal (artery or vein) Thrombosis
- Infectious – Sepsis, Pyelonephritis
<table>
<thead>
<tr>
<th>Parenchymal (intrinsic) kidney injury</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ischemic injury/ATN</strong></td>
</tr>
<tr>
<td>Any of the prerenal causes if prolonged</td>
</tr>
<tr>
<td>Patient is at risk for further kidney injury throughout the injury and recovery phases, so avoid additional insults as much as possible</td>
</tr>
<tr>
<td>Perinatal asphyxia/hypoxic-ischemic injury</td>
</tr>
<tr>
<td>Endothelial and tubular cell damage may trigger a systemic inflammatory response that causes distant organ dysfunction14, 48 and 49</td>
</tr>
<tr>
<td><strong>Nephrotoxic medications</strong></td>
</tr>
<tr>
<td>Aminoglycosides, amphotericin, intravenous contrast</td>
</tr>
<tr>
<td>Aminoglycosides: primarily proximal tubular cell damage, use with caution in any patient with preexisting AKI, concomitant nephrotoxic medication use, or poor renal perfusion. Usually nonoliguric AKI</td>
</tr>
<tr>
<td>Amphotericin B: causes renal tubular acidosis and increased urinary potassium excretion. Reported levels of toxicity vary widely51 and 52</td>
</tr>
<tr>
<td><strong>Decreased renal perfusion</strong></td>
</tr>
<tr>
<td>ACE inhibitors, NSAIDs (indomethacin), diuretics</td>
</tr>
<tr>
<td>Indomethacin: commonly associated with increased SCr concentrations, decreased urine output, hyponatremia. Usually reversible47 and 53</td>
</tr>
<tr>
<td><strong>Tubular obstruction</strong></td>
</tr>
<tr>
<td>Acyclovir</td>
</tr>
<tr>
<td><strong>Sepsis and other infections</strong></td>
</tr>
<tr>
<td>Decreased renal blood flow and subsequent ATN from shock/hypotension</td>
</tr>
<tr>
<td>Sepsis-associated AKI</td>
</tr>
<tr>
<td>Microvascular dysfunction associated with normal or increased renal blood flow that manifests with decreased GFR and tubular dysfunction; histologically distinct from ATN54</td>
</tr>
<tr>
<td>Pyelonephritis</td>
</tr>
<tr>
<td>Congenital infections</td>
</tr>
<tr>
<td><strong>Vascular lesions</strong></td>
</tr>
<tr>
<td>Renal vein and artery thrombosis</td>
</tr>
<tr>
<td>Perinatal event; risk factors include perinatal asphyxia, dehydration, infection, prematurity, maternal diabetes, and underlying hypercoagulable state55</td>
</tr>
</tbody>
</table>

- Retrospectively looked at 107 VLBW infants.
- 87% were exposed to nephrotoxic medications at least once.
- Lower GA and lower BW infants were more frequently exposed.
AKI POSTRENAL

- Obstructive Uropathy
- Post Urethral Valves
- Obstruction
RENAL DISEASE IN NEONATES

CHRONIC KIDNEY DISEASE
Chronic Kidney Disease

* Anytime AKI leads to scarring or damage to the kidney (as a result of perinatal asphyxia, hypoxia, sepsis or hypovolemia)

* Since nephrogenesis proceeds through 36 weeks gestation, any events can not only cause AKI but can also lead to CKD
Disorders resulting in neonatal CKD


- Aplastic/hypoplastic/dysplastic kidneys
- Autosomal dominant polycystic kidney disease
- Autosomal recessive polycystic kidney disease
- Obstructive uropathy (posterior urethral valves)
- Pyelonephritis
- Reflux nephropathy
- Renal infarct
- Syndrome of agenesis of abdominal musculature

* May result in need for dialysis in neonatal period.
Diagnosis of neonates with end-stage renal disease

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>n</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal dysplasia</td>
<td>72</td>
<td>(37.3)</td>
</tr>
<tr>
<td>Obstructive uropathy</td>
<td>39</td>
<td>(20.2)</td>
</tr>
<tr>
<td>ARPKD</td>
<td>23</td>
<td>(11.9)</td>
</tr>
<tr>
<td>Congenital nephrotic syndrome</td>
<td>3</td>
<td>(1.5)</td>
</tr>
<tr>
<td>Other</td>
<td>56</td>
<td>(29)</td>
</tr>
</tbody>
</table>

Abbreviation: ARPKD, autosomal recessive polycystic kidney disease.

The typical diagnostic criteria of GFR < 60 (KDOQI) does not apply until > 2 years old.

The updated Schwartz formula: eGFR = 0.413 * height/Scr does not apply in children 0 – 2 yrs old

Normal GFR in newborn period is significantly < 60
**Glomerular filtration rate (GFR) in healthy infants as assessed by inulin clearance**

<table>
<thead>
<tr>
<th>Age</th>
<th>Mean GFR ± SD (mL/min/1.73 m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Preterm babies</strong></td>
<td></td>
</tr>
<tr>
<td>1–3 d</td>
<td>14.0 ± 5</td>
</tr>
<tr>
<td>1–7 d</td>
<td>18.7 ± 5.5</td>
</tr>
<tr>
<td>4–8 d</td>
<td>44.3 ± 9.3</td>
</tr>
<tr>
<td>3–13 d</td>
<td>47.8 ± 10.7</td>
</tr>
<tr>
<td>8–14 d</td>
<td>35.4 ± 13.4</td>
</tr>
<tr>
<td>1.5–4 mo</td>
<td>67.4 ± 16.6</td>
</tr>
<tr>
<td><strong>Term babies</strong></td>
<td></td>
</tr>
<tr>
<td>1–3 d</td>
<td>20.8 ± 5.0</td>
</tr>
<tr>
<td>3–4 d</td>
<td>39.0 ± 15.1</td>
</tr>
<tr>
<td>4–14 d</td>
<td>36.8 ± 7.2</td>
</tr>
<tr>
<td>6–14 d</td>
<td>54.6 ± 7.6</td>
</tr>
<tr>
<td>15–19 d</td>
<td>46.9 ± 12.5</td>
</tr>
<tr>
<td>1–3 mo</td>
<td>85.3 ± 35.1</td>
</tr>
</tbody>
</table>

*Abbreviation:* SD, standard deviation.

### KDIGO classification schemata for CKD for ages less than 2 years

<table>
<thead>
<tr>
<th>Neonatal CKD Classification</th>
<th>GFR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal GFR</td>
<td>GFR ≤1 SD below the mean</td>
</tr>
<tr>
<td>Moderately reduced GFR</td>
<td>GFR &gt;1 SD to ≤2 SD below the mean</td>
</tr>
<tr>
<td>Severely reduced GFR</td>
<td>GFR &gt;2 SD below the mean</td>
</tr>
</tbody>
</table>

**Abbreviations:** KDIGO, Kidney Disease: Improving Global Outcomes; SD, standard deviation.

Factors Influencing Lab Assessment
Normal BUN levels are higher with lower GA and BW

Elevated BUN levels are often cited as reason for limiting AA or protein intake
Ridout, E. et al, J of Perinatology (2005) did retrospective review of BUN levels and AA intake of 121 infants with BW <1250g.

- 4 groups: <1gAA/kg, 1-1.9gAA/kg, 2-2.9gAA/kg, >3gAA/kg
- NO Correlation Found Between AA Intake and BUN levels
- BUN is a complex outcome of hydration status, AA oxidation, renal function, energy intake and degree of illness.
- Evaluating BUN as a single marker of protein intolerance is not justified.
Roggero, 2010: Prospective, longitudinal study of 92 infants with mean GA and BW of 29.7 weeks and 1125 g

- Progressive AA intake of 1.5-3.5 g/kg over 1st 5 days of life. Maintenance of higher enteral protein intake when transitioned to feedings.
- No correlation between AA intake and BUN levels
- GA was inversely correlated with BUN levels
- After transition to full enteral feeds, BUN levels more closely correlated with enteral protein intake.

- 249 infants, all <30 wks gestation at birth
- Provided recommended protein/AA intakes over the 1st 3 weeks of life (range: 2.1 – 3.9 g/kg)
- Measured BUN, Cr and protein/AA intake for each week
Mean BUN, mg/dl

Weintraub AS et al 2015
Weintraub AS et al 2015
Mean Protein Intake, g/kg/day

Week 1  Week 2  Week 3

Weintraub AS et al 2015
Creatinine

- High at birth; reflects mother’s level.
- Transient increase (2 -5 days) initially reflecting diuresis. Can take up to 3-4 wks to normalize in a premature infant.
- These higher levels the 1st month also reflect incomplete nephrogenesis.
- Harriet Lane:
  - Newborn: .3-1.0
  - Infant: .2-.4
<table>
<thead>
<tr>
<th>Factors Influencing Lab Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Phosphorus</strong></td>
</tr>
<tr>
<td>* Normal reference range 4.2 – 8.5mg/dl</td>
</tr>
<tr>
<td>* Premies have very high phosphorous requirements.</td>
</tr>
<tr>
<td><strong>Potassium</strong></td>
</tr>
<tr>
<td>* Hyperkalemia is common but may not be real.</td>
</tr>
<tr>
<td>* Beware of false elevation due to frequent heel sticks and hemolysis.</td>
</tr>
<tr>
<td>* Potassium will be elevated if baby is acidotic.</td>
</tr>
</tbody>
</table>
Sodium and Water

- Term and premies undergo 10 – 20% loss of extracellular fluid immediately after birth.

- Term: regains BW in 7-10 days
  Preterm: regains BW in 14-21 days

- Fluid loss accompanied by Sodium loss.

- In Premies: Renal sodium losing state is greater and more prolonged. Most normal premies may temporarily require sodium supplementation
Fractional Excretion of Sodium (FENa) is inversely related to gestational age. The lower the GA, the higher the loss of sodium.

Many premature infants will require Na supplementation (even without dx of CKD)

If there is AKI/CKD, with normal or high urine output, the Na supplementation needed may be higher.

Examples are recovering ATN in AKI and obstructive uropathy with tubular damage in CKD. (Exception: anuric/oliguric and not on dialysis)
Sodium and Water

- FENa: \[
\frac{(UNa/PNa)}{(UCr/PCr)} \times 100\%
\]

- FENa can be as high as 5% in term babies immediately after birth. Falls to normal in a few days in term babies. This process is delayed premature infants.

- Normal FENa is <1%. 
Medical and Nutrition
INTERVENTION
INTERVENTION:

* Management of fluid balance
* Management of electrolytes
* Acid/Base Balance
* Renal Replacement Therapy (RRT)
* Nutrition
MANAGEMENT OF FLUID BALANCE

- Maintenance fluid = 100 ml/kg/d
- Typical fluids to meet kcal needs with 24/oz feedings = 150 mls/kg
- If Anuric/Oliguric may need severe fluid restrictions (ie) 60 – 80 ml/kg/d
- Requires concentration of formulas (up to 60 kcals/oz) and/or concentration of TPN
- If Polyuric, fluid needs may increase up to 200 ml -250 mls/kg/d (examples would be in babies with concentrating defects)
MANAGEMENT of ELECTROLYTES
HYponatremia

- May require fluid restriction
- May require Sodium supplementation
  - If very premature
  - If there is a sodium losing component with renal failure
    - Calculate FENa
  - If baby is on peritoneal dialysis
- Sodium can be increased in TPN or NaCl added to formula
- Phos and bicarb supplementation will also increase the Na supplementation
Hyperkalemia is common

- Beware of false elevations – ie) heel sticks
- Change feedings to lower K content
- Kayexalate can be added to formula followed by decanting
  - 1 g kayexalate for each meq of K removed
  - Range of 0.5-1.5g Kayexalate per 100 mls EMM or formula
- Remove K from TPN or titrate down
- Dialysis may be necessary
MANAGEMENT of ELECTROLYTES
HYPERPHOSPHATEMIA

- Remember... the normal reference range for phosphorous
  - For premies: 4.2 – 8.5mg/dl
  - For term: 4.2 – 7.0mg/dl

- If phosphorous is above those ranges
  - Use Low Phos formula (PM 60/40) and/or Breast Milk
  - Add liquid Calcium Carbonate to formula to bind

- Phos is likely to go too low once dialysis started
  - Titrate down or stop dose of calcium carbonate
  - May need phos supplementation (sodium phosphate: 93 mg/ml)
MANAGEMENT of ELECTROLYTES
ACID/BASE BALANCE

- Metabolic Acidosis
  - Very common in AKI
  - Ongoing issue in CKD
- Treated with Sodium Bicarbonate, Sodium Citrate or Dialysis
- Remember...
  - Acidotic babies will not gain weight or grow
  - Treatment will add to sodium intake so it needs to be accounted for if you are also using NaCl
  - Acidosis can cause the hyperkalemia!
Peritoneal Dialysis is predominant choice

Literature cites premies as low as 930 g getting long term PD and smaller for short term PD

Hemodialysis: depends on expertise available

CRRT: depends on expertise

As low as 1500 g at OHSU

HD and CRRT not good long term options due to frequent clotting and infection.
Nutritional Challenges In Infants with CKD

- anorexia
dysgeusia
chewing/swallowing problems
delayed stomach emptying
vomiting, GER
psychogenic

↓ intake

- dialysis
vomiting
peritonitis

↑ losses

- metabolic acidosis
medications
catch-up growth

↑ needs

energy &/or protein deficit

poor growth/FTT
## Term Infants

### Table 2. Equations to Estimate Energy Requirements for Children at Healthy Weights

<table>
<thead>
<tr>
<th>Age</th>
<th>Estimated Energy Requirement (EER) (kcal/d) = Total Energy Expenditure + Energy Deposition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3 mo</td>
<td>(EER = [89 \times \text{weight (kg)} - 100] + 175)</td>
</tr>
<tr>
<td>4-6 mo</td>
<td>(EER = [89 \times \text{weight (kg)} - 100] + 56)</td>
</tr>
<tr>
<td>7-12 mo</td>
<td>(EER = [89 \times \text{weight (kg)} - 100] + 22)</td>
</tr>
</tbody>
</table>
### Table 12. Recommended Dietary Protein Intake in Children with CKD Stages 3 to 5 and 5D

<table>
<thead>
<tr>
<th>Age</th>
<th>DRI (g/kg/d)</th>
<th>Recommended for CKD Stage 3 (g/kg/d)</th>
<th>Recommended for CKD Stages 4-5 (g/kg/d)</th>
<th>Recommended for HD (g/kg/d)*</th>
<th>Recommended for PD (g/kg/d)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-6 mo</td>
<td>1.5</td>
<td>1.5-2.1</td>
<td>1.5-1.8</td>
<td>1.6</td>
<td>1.8</td>
</tr>
<tr>
<td>7-12 mo</td>
<td>1.2</td>
<td>1.2-1.7</td>
<td>1.2-1.5</td>
<td>1.3</td>
<td>1.5</td>
</tr>
</tbody>
</table>
CALORIE AND PROTEIN RECOMMENDATIONS

Premature

<table>
<thead>
<tr>
<th></th>
<th>KCALS</th>
<th>PROTEIN</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONSERVATIVE</td>
<td>110 – 150 kcals/kg</td>
<td>3.4 – 4.4 g/kg</td>
</tr>
<tr>
<td>PERITONEAL DIALYSIS</td>
<td>110 – 150 kcals/kg</td>
<td>?</td>
</tr>
<tr>
<td>HEMODIALYSIS</td>
<td>110 – 150 kcals/kg</td>
<td>?</td>
</tr>
</tbody>
</table>

• No published references on Protein needs for Premies on dialysis

• If term baby recs. are to increase Protein intake by .1-.35 g/kg/day (K/DOQI 2009) then a conservative approach would be to increase premies protein intake above normal needs by .1g/kg (for HD) and .35 g/kg (for PD)

• With HD protein needs increase: 3.5-4.5 g/kg/day

• With PD protein needs increase: 3.75-4.75g/kg/day
**Protein Recommendations by Gestational Age**

<table>
<thead>
<tr>
<th>Gestational Age</th>
<th>Conservative* g/kg</th>
<th>PD g/kg</th>
<th>HD g/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>26 – 30 wks</td>
<td>3.8 – 4.4</td>
<td>4.15 – 4.75</td>
<td>3.9 – 4.5</td>
</tr>
<tr>
<td>30 – 36 wks</td>
<td>3.4 – 4.2</td>
<td>3.75 – 4.55</td>
<td>3.5 – 4.3</td>
</tr>
<tr>
<td>36 – 40 wks</td>
<td>2.8 – 3.4</td>
<td>3.15 – 3.75</td>
<td>2.9 – 3.5</td>
</tr>
</tbody>
</table>

*Adapted from Tsang, 2005*
INTERVENTION
Nutrition

* Monitor growth parameters closely
* **Start by determining fluid needs**-anuric, oliguric, normal, polyuric? - with or without dialysis?
* Make modifications to calorie or protein intake based on your assessment of current calorie intake, protein intake, growth.
* Monitor BUN:Cr Ratio
  * Normal ratio is 10-20: 1
  * If ratio is high: perhaps not enough calories, too much protein or breakdown of lean body mass, dehydration, needs more dialysis?
  * If ratio is low: not enough protein, fluid overload
INTERVENTION
NUTRITION
Vitamins and Minerals

* Need to meet the requirements of the Term or Premature Infant
* If K or Phos restriction warrants a change from normal premature feedings, then MVI supplements will be needed
* Use single Mineral supplements to provide mineral requirements (ie) CaCO₃, Ferinsol, NaPhosphate, KCl
* Watch Calcium and Phosphorous levels closely
  * Remember the increased needs of premies
  * May need both Calcium and Phos. Supplementation
* Watch Sodium levels closely.
  * Premies, Polyuric, and PD babies tend to be salt wasters.
* May need additional B vitamins and Vit C if on dialysis.
* Nutrient needs are high for premies. But because of high serum K and Phos with renal failure…. Premature formulas are discontinued and Fortified Breast Milk is stopped.

* Usual practice is to start PM 60/40 or unfortified Breast Milk: then concentrate as needed

* Supplementation of individual nutrients may be necessary: MVI, iron, Na, Ca and Phos

* Pay close attention to serum phos levels. Keep phos levels above 5 and below high end of reference range.
FORMULA DECISIONS
GENERAL CONSIDERATIONS

- Consider slowly changing back to higher nutrient density formulas or fortified breast milk once on dialysis or if potassium and phosphorous levels drop to lower ends of reference ranges.

- Individual nutrient supplementation will likely increase once dialysis is started.
* Determine urine output status of infant: anuric, oliguric, normal, polyuric
* Determine fluid status of infant: dehydrated, normal hydration, edematous
* If fluids restricted to maintenance fluid (100 ml/kg) Requires 36 kcals/oz to meet basic premie needs and 32 kcals/oz to meet term requirements.
* If more fluid restricted (ie 60-80 ml/kg) then greater concentration required.
FORMULA DECISIONS

CONCENTRATING

- Increase formula powder to fluid (water or breast milk) ratio until protein needs are met
- Use carbohydrate/fat modulars for additional concentration if necessary to meet calorie goals
  
  OR

- Start with 20/oz formula or breast milk + protein/carbohydrate/fat modulars to meet calorie per oz desired (see Yiu 1996 reference)
If highly polyuric, formula may need to be prepared to less than 20 kcals/oz

There are 2 ways to do this:
- Prepare recipes for less than 20/oz – ie) 14, 16, 18 etc
- Prepare 20/oz feedings but instruct on giving additional water


