PREMATURITY: CLINICAL ISSUES
PART TWO

Loy Maslen, MSN, RN, NNP-BC, CPHM
Perinatal Clinical Nurse Specialist
UW/Valley Medical Center
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Birthweight: 2 ½ lbs
1920

The Long Journey Home
Born in 1974, BW: 600 gm
LEARNING OBJECTIVES

• Discuss neurologic factors affecting preterm infants and strategies for minimization
• List causes and interventions for ROP
• Review Infant Blood Protocol and reasons for anemia of prematurity
• Apply knowledge of apnea of prematurity and hyperbilirubinemia to appropriate interventions
• Analyzes the fluid and electrolyte needs of infants
• Discuss nutritional needs and feeding complications of the preterm infant
The third trimester is a time of rapid brain growth and development which continues on through the first year after birth.
• Cortical neurons are generated in the periventricular germinal matrix.
• Almost all neurons have actually been generated by 25 weeks.
Normal anatomy in horizontal section showing location of subependymal germinal matrix
Neurons then migrate out toward the surface of the cortex, branch out, and form synapses.

By 32-34 weeks, focus of development shifts to the cortex.

Germinal matrix tissues themselves undergo involution.
At 24-28 weeks, the supportive structures around the germinal matrix are still very fragile, making the baby vulnerable to hemorrhage into the ventricles.
Figure 32–1. Pathway of cerebrospinal fluid flow from the choroid plexuses in the lateral ventricles to the arachnoidal villi protruding into the dural sinuses.
The autonomic control of cerebral blood flow is poorly developed in the preemie, allowing fluctuations in cerebral blood flow which can lead to bleeding and/or ischemia.
DIAGRAM 2: Site of Frequent Hemorrhage
(Veins involved in hemorrhage labeled on diagram)

Thalamostriate Vein

Choroidal Vein

Internal Cerebral Vein

OTHER VEINS OF THE GALENIC SYSTEM, MID-SAGITTAL VIEW

1. Great vein of Galen
2. Transverse sinus
3. Rectus sinus
4. Inferior sagittal sinus
5. Transverse caudate vein
6. Anterior terminal vein
7. Septal vein
8. Basal vein
INTRAVENTRICULAR HEMORRHAGE (IVH)

• Risk Factors:
  • <34 weeks
  • <1500 grams
  • “unstable”
  • 1st 5 days of life
  • 1st 72 hours most critical

• Incidence:
  • Has decreased over the past 20 years to about 10-20% of VLBW babies in most NICUs. (Down from over 50%)
INTRAVENTRICULAR HEMORRHAGE (IVH)

- **Clinical Presentation:**
  - Ranges from catastrophic (least common) to silent (most common)

- **Diagnosis:**
  - Cranial Ultrasound at 1 week of age detects 95% of all IVH or at time of dramatic condition change
GRADERS I-IV: Periventricular-Intraventricular Hemorrhage

Grade I-Subependymal hemorrhage only

Grade II-Intraventricular hemorrhage without ventricular dilation

Grade III-Intraventricular hemorrhage with ventricular dilation

Grade IV-Intraventricular hemorrhage with parenchymal hemorrhage
INTRAVENTRICULAR HEMORRHAGE (IVH)

- Management
  - Serial ultrasounds
  - CSF taps or drainage for progressive hydrocephalus
  - Ventricular-peritoneal shunt
INTRAVENTRICULAR HEMORRHAGE (IVH)

• Prognosis
  • Neurodevelopmental outcome of preemies with grade I or II hemorrhage similar to comparable babies with no hemorrhage.
  • Some increased risk in visual motor integration skills.
INTRAVENTRICULAR HEMORRHAGE (IVH)

- 30-40% of those with moderate (Grade III) bleeds have major neurologic sequelae (higher with periventricular infarct or PVL)
- “Major” neurologic sequelae
  - Blindness
  - Deafness
  - Cerebral Palsy
  - Severe mental retardation
PERIVENTRICULAR LEUKOMALACIA (PVL)

- Necrosis of white matter in a characteristic distribution dorsal and lateral to the lateral ventricles. It is the primary ischemic lesion of the premature infant.
- Currently the major form of brain injury in preterm infants.
PERIVENTRICULAR LEUKOMALACIA

• Risk Factors
  • Prematurity – 23-32 weeks
  • Postnatal illness – hypoxia, ischemia, inflammation
  • Maternal cocaine and methamphetamine use
  • Maternal smoking
PERIVENTRICULAR LEUKOMALACIA (PVL)

Pathophysiology

• Pressure passive cerebral circulation
• Rapidly growing cerebral white matter has high metabolic needs
• Periventricular vascular anatomic factors
• Low blood flow leading to necrosis, hemorrhage, and cysts
• Initial insult plus damage to process of myelinization
FIGURE 1 Cerebral arteries

- Anterior cerebral artery
- Lateral ventricles
- Subependymal area
- Middle cerebral arteries
- Posterior cerebral artery
PERIVENTRICULAR LEUKOMALACIA (PVL)

• Clinical Presentation
  • Generally silent, picked up on ultrasound. Delay before evident on ultrasound.

• Prognosis
  • Major long term sequela is spastic diplegia, especially affecting legs.
  • High risk for developmental problems.
DIFFUSE CELLULAR PVL

• MRI can detect diffuse PVL on cellular level
• May play a role in intellectual deficit
STRATEGIES BEFORE BIRTH

• Prevent/delay premature birth ➔ parental education
• Tocolytics
• Antenatal steroids
• Magnesium Sulfate for neuroprotection
NEUROPROTECTIVE STRATEGIES:

- Delayed umbilical cord clamping
- Stable BP and oxygenation
- Maintain good glucose levels
- Avoid rapid infusion of bicarb or any medication
- Avoid low pCO2 levels
- Keep head in neutral (midline) position for 72 hours of life
- Gentle handling, minimal stimulation, developmental support
TIMING OF UMBILICAL CORD CLAMPING AFTER BIRTH

The optimal timing for clamping the umbilical cord after birth has been a subject of controversy and debate. Although many randomized controlled trials in term and preterm infants have evaluated the benefits of delayed umbilical cord clamping versus immediate umbilical cord clamping, the ideal timing for cord clamping has yet to be established. Several systematic reviews have suggested that clamping the umbilical cord in all births should be delayed for at least 30–60 seconds, with the infant maintained at or below the level of the placenta because of the associated neonatal benefits, including increased blood volume, reduced need for blood transfusion, decreased incidence of intracranial hemorrhage in preterm infants, and lower frequency of iron deficiency anemia in term infants. Evidence exists to support delayed umbilical cord clamping in preterm infants, when feasible. **The single most important clinical benefit for preterm infants is the possibility for a nearly 50% reduction in intraventricular hemorrhage.** However, currently, evidence is insufficient to confirm or refute the potential for benefits from delayed umbilical cord clamping in term infants, especially in settings with rich resources.
MIDLINE HEAD POSITIONING FOR 72 HR

• Measured changes in cerebral blood flow and cerebral blood volume with head in midline and head rotated to the side

• Found that the mean cerebral blood volume was significantly higher when the head was rotated 90 degrees to the side

• “Rotation of the head to one side obliterates the internal jugular vein on the same side”
MIDLINE HEAD POSITIONING

- Measured effects of six different head positions on cerebral blood velocity and intracranial pressure.
- Found that ICP significantly increased when head turned to right compared to midline.
- ICP lower when head elevated 30 degrees.
SYSTEMATIC LITERATURE REVIEW

• Multidisciplinary group from 5 institutions formed to examine evidence for potentially better practices to prevent IVH

• Used evidence-based approach to grade the evidence

• Developed 10 recommendations, including: “Maintain neutral head position when turning and positioning the infant with the head of bed elevated 30 degrees.” This was based on:

  Level IV evidence: Well designed case-control or cohort study
  – Level VII evidence: Expert opinion/committees
HTTPS://WWW.YOUTUBE.COM/WATCH?V=9-N3YDY7RAS
RETINOPATHY OF PREMATURITY (ROP)
RETINOPATHY OF PREMATURITY (ROP)

- **ROP** describes an abnormality of growth and development of the retina of the premature infant.
- Untreated, it may progress to retinal detachment and blindness.
- In the US, approximately **500 to 700** infants lose their vision due to ROP each year.
  - Additionally, **4500** infants will develop complications of ROP including myopia, strabismus, and late-onset retinal detachment/blindness.
NORMAL RETINAL DEVELOPMENT

• The retina begins to develop at the 16th week of gestation.

• Blood vessels grow out of the optic disc and slowly advance outward.

• The retina is not completely vascularized until 36 to 40 weeks gestation.

• Preterm infant has immature formation of antioxidant enzymes and free radical scavengers.
PATHOPHYSIOLOGY OF ROP

• Initial hyperoxic injury
  • Elevated oxygen levels (*oxygen saturations >95*) cause severe 
    vasoconstriction and destruction of immature retinal vessels
  • The vasoconstriction severely inhibits blood flow to the retina (*retinal 
    ischemia*)
  • By about 30-34 weeks gestation the ischemic retina attempts to 
    restore its blood flow by releasing growth factors to stimulate 
    new blood vessel growth (*neovascularization*)
    • This “catch-up” growth is *abnormal and poorly controlled*, and may 
      result in retinal detachment and blindness.
PATHOPHYSIOLOGY OF ROP

**Hyperoxia** (high O2 saturations)
Vessel constriction/destruction

**Neovascularization**
(abnormal vessel growth)
ROP: PREVENTION

• Avoid **hyperoxia** (O2 sat >95%), especially in the first few weeks of life

• Avoid large **fluctuations** in oxygen saturations
RETINOPATHY OF PREMATURITY (ROP): SCREENING GUIDELINES

- 30 weeks or less at birth
- <1500 grams at birth
- Consider screening if premature, 1500-1800 grams at birth, and unstable
- Start 4 to 6 weeks after birth at 31 to 33 weeks corrected gestational age.

Scheme of retina of right eye (RE) and left eye (LE) showing zone borders and clock hours employed to describe location and extent of retinopathy of prematurity.
Stage 1: Demarcation Line

Stage 2: Ridge

Stage 3: Ridge with Extraretinal Vascular Proliferation

Plus Disease: Dilated & Tortuous Vessels
Stage 4:
Subtotal retinal detachment beginning at the ridge.
The retina is pulled anteriorly into the vitreous by the fibrovascular ridge.

http://www.nei.nih.gov/rop/images/ROP10-31-03.mpeg
Laser photocoagulation/retinal ablation has been successfully used to prevent retinal detachment in threshold ROP.
RETINO PathY OF PREMATURITY (ROP) – LASER TREATMENT GUIDELINES

• Zone 1 ROP: any stage with plus disease
• Zone 1 ROP: stage 3 – no plus disease
• Zone II: stage 2 or 3 with plus disease
EMERGING THERAPY: INTRAVITREAL BEVACIZUMAB (AVASTIN)

- 2011 Study showed benefit for vision threatening ROP in Zone 1
- Anti-VEGF agent (vascular endothelial growth factor)

VON presentation Dec. 2011
OUTCOME

Mild ROP (Stage 2 or less) generally has a favorable outcome.

Severe ROP (Stage 3 and above) may progress to partial or total retinal detachment and blindness.

Infants with severe ROP are also at risk for severe myopia (near-sightedness) and strabismus/amblyopia.

Infants with severe ROP may have retinal scarring which can lead to late retinal detachments in adolescence or early adulthood.
HEMATOLOGICAL: ANEMIA

• Acute (pathologic)
  • bruising, hemorrhage, or hemolysis

• Iatrogenic Anemia from lab testing

• Survival of premature infant’s red blood cells about 50% less than adult circulation time. (Adult cells last average of 120 days)
• Bloodworks NW: Infant 0-4 month Protocol uses specimen of infant or cord blood for type and antibody screen.

• This screen is good until infant is 4 months old (when they begin to make significant antibody of their own) or leaves hospital.
TRANSFUSION
ASSIGNED ALIQUOTS

- Pediatric Assigned Unit
- 1 Adult unit divided into 8 aliquots and kept for that baby.
- Reduces exposure to multiple donors
- All units leukoreduced and irradiated
- Good for 42 days from collection
ANEMIA OF PREMATURITY

Physiologic

• At birth, the infant is suddenly transferred from the relative hypoxia of the uterus to the oxygen rich environment of the outside atmosphere.

• Red cell production in the bone marrow virtually ceases and resumes activity when the infant is about 2 months old.

• In the preterm infant, this physiologic process can be prolonged and the hematocrit fall to a lower level.
ANEMIA OF PREMATURITY

• Management
  • Premature infants on breast milk only need supplemental iron once on full oral feedings to ensure they have iron when they begin reticulating.
ANEMIA OF PREMATURITY

• Management
  • Consider transfusion if infant has hematocrit in low 20’s or teens, has apnea or oxygen needs, is not growing well, or has low reticulocyte count.
  • Erythropoetin in limited situations
    • Epo works slowly
    • Needs adequate iron stores
HEMATOLOGICAL:
HYPERBILIRUBINEMIA

• The preterm infant liver is immature and does not clear bilirubin as efficiently.
• Concern that immaturity of the blood/brain barrier makes preemie more vulnerable to bilirubin toxicity - kernicteris.
HYPERBILIRUBINEMIA

- Preemies generally treated for hyperbilirubinemia at lower levels than term infants.
- 2004 AAP guidelines show that even the mildly premature infant is at increased risk.
Figure 4. Guidelines for Intensive Phototherapy in Hospitalized Infants Born at a Gestational Age of 35 Weeks or More.

- Use total bilirubin. Do not subtract direct reacting or conjugated bilirubin.
- Risk factors = isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis, or albumin < 3.0g/dL (if measured).
- For well infants 35-37 6/7 wk can adjust TSB levels for intervention around the medium risk line. It is an option to intervene at lower TSB levels for infants closer to 35 wks and at higher TSB levels for those closer to 37 6/7 wk.
- It is an option to provide conventional phototherapy in hospital or at home at TSB levels 2-3 mg/dL (35-50mmol/L) below those shown but home phototherapy should not be used in any infant with risk factors.
HYPERBILIRUBINEMIA IN THE PRETERM INFANT

<table>
<thead>
<tr>
<th>Gestational Age (week)</th>
<th>Phototherapy</th>
<th>Exchange Transfusion</th>
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<tbody>
<tr>
<td></td>
<td>Initiate Phototherapy total serum bili (mg/dl⁻¹)</td>
<td>Total serum bili (mg/dl⁻¹)</td>
</tr>
<tr>
<td>&lt;28 0/7</td>
<td>5-6</td>
<td>11-14</td>
</tr>
<tr>
<td>28 0/7 – 29 6/7</td>
<td>6-8</td>
<td>12-14</td>
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<tr>
<td>30 0/7 – 31 6/7</td>
<td>8-10</td>
<td>13-16</td>
</tr>
<tr>
<td>32 0/7 – 33 6/7</td>
<td>10-12</td>
<td>15-18</td>
</tr>
<tr>
<td>34 0/7 – 34 6/7</td>
<td>12-14</td>
<td>17-19</td>
</tr>
</tbody>
</table>

“Not based on good evidence” Use lower range of listed TSB levels for lower gestational age, serum albumin levels < 2.5 g/dl⁻¹, rapidly rising TSB levels, or clinically unstable infants.

HYPERBILIRUBINEMIA

- Late preterm infant at risk due to
  - immaturity of liver
  - immaturity of feeding skills
  - early discharge home without adequate understanding and supervision.
Newborn babies have high total body water content.

Neonates have higher basic metabolic rate.

Newborn water requirements are 4-5 times greater.
FLUIDS & ELECTROLYTES

- All newborns lose fluid after birth
- Loss of fluid from extracellular compartment followed by diuresis, and weight loss
- NNB regains weight as muscle and fat
- This process complicated in preterm infants
  - More dilute urine, greater sodium and water losses
  - Fluid overload may contribute to respiratory distress, congestive heart failure, necrotizing enterocolitis, PDA
IMMATURE KIDNEYS

• Newborns have a lower renal blood flow than adults
  • increases rapidly after birth in the term and older premature with postnatal shifts in blood flow
  • more slowly in the preterm less than 34-35 weeks. (Remember: mom was doing this!)
• Newborn has lower glomerular filtration rate; again, it doubles by two weeks of age, but increases more slowly in the younger preemie
RENAL

• Newborns have limited ability to concentrate urine, and prematures are even more limited in this regard. Lose salt and water in urine

• Full complement of nephrons develops at around 35 weeks

• Postnatal renal function is more related to postbirth age than gestational age; birth stimulates some maturation
RENAL: PRETERM PROBLEMS

• Narrow margin of safety in calculating and administering fluids and electrolytes

• Fluid and Electrolyte Problems: Vulnerable to overhydration, dehydration, hyper/hyponatremia, hypo/hyperkalemia

• Buffering Capacity: The preterm infant is vulnerable to any event that causes acidosis or alkalosis. They do not retain bicarb well
RENAL: PRETERM PROBLEMS

• Drug Clearance:
  • Prematurity and illness restrict a neonate’s ability to excrete certain drugs and increase risk of toxicity
    • Aminoglycosides in particular are timed with regard to gestational age and levels are followed closely
    • Asphyxia can worsen this problem
# GENTAMICIN

## Dosing Chart

<table>
<thead>
<tr>
<th>PMA (weeks)</th>
<th>Postnatal (days)</th>
<th>Dose (mg/kg)</th>
<th>Interval (hours)</th>
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<tbody>
<tr>
<td>≤29*</td>
<td>0 to 7</td>
<td>5</td>
<td>48</td>
</tr>
<tr>
<td></td>
<td>8 to 28</td>
<td>4</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>≥29</td>
<td>4</td>
<td>24</td>
</tr>
<tr>
<td>30 to 34</td>
<td>0 to 7</td>
<td>4.5</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>≥8</td>
<td>4</td>
<td>24</td>
</tr>
<tr>
<td>≥35</td>
<td>ALL</td>
<td>4</td>
<td>24</td>
</tr>
</tbody>
</table>

*or significant asphyxia, PDA, or treatment with indomethacin

Neofax 2011
FLUID & ELECTROLYTE MANAGEMENT

• Fluid requirements
  • (May be much higher for the extremely low birthweight without humidity)
    Day 1       80 cc/Kg/day
    Day 2      80-100 cc/Kg/day
    Day 3      100-120 cc/Kg/day
    Day 4     120-150 cc/Kg/day

• Adjust baseline for insensible water loss such as phototherapy, radiant warmer, surgical conditions, immature skin
FLUID & ELECTROLYTE MANAGEMENT

• Electrolytes added once the baby voids
• Strict monitoring of intake and output until stable
• Monitor electrolytes, pH, and blood gases.
Fluid status is assessed based on I&O, daily weight, appearance, and electrolytes (mainly Na$^+$)

- Normal electrolyte values:
  - Na$^+$: 135-145
  - K: 3.5-5.5
  - Cl: 90-110
Only one way out of NICU:

Grow, Baby, Grow!
• Immature GI tract function until 36-40 weeks limits digestion and absorption
• GI motility is decreased in preterm infants
• Lower production of digestive and hepatic enzymes
GASTROINTESTINAL/NUTRITIONAL

• Maintaining Adequate Nutrition
  • Preterm infants are born without the reserves of fat, carbohydrate, vitamins and minerals which would have been transferred from their mothers during the latter part of pregnancy
GASTROINTESTINAL/NUTRITIONAL

- Maintaining Adequate Nutrition
  - Higher energy expenditure than in the womb
  - Caloric needs related to the disease processes
  - Developmentally, high caloric needs for growth
GROWTH & NUTRITION

• Many premature infants born on lower end of growth curve
• With inadequate nutrition, they tend to fall even lower on the curve
• Inadequate nutrition leads to growth failure and impaired neurocognitive development
Olsen Growth Curve
The Challenge of Catch-up Growth

Most VLBW infants regain birthweight by 2\textsuperscript{nd} or 3\textsuperscript{rd} week of life. The longer the time it takes to regain, the slower the catchup growth will be.
GASTROINTESTINAL/NUTRITIONAL

• Goals for Growth:
  • To approximate in utero growth of a normal fetus of the same postconceptual age
    • Weight gain of 18 grams/kg/day
    • Length: about .75 cm per week
    • Head circumference: about .5 cm per week
<table>
<thead>
<tr>
<th>Factor</th>
<th>American Academy of Pediatrics</th>
<th>European Society of Gastroenterology and Nutrition</th>
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</thead>
<tbody>
<tr>
<td>Energy expenditure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Resting metabolic rate</td>
<td>50</td>
<td>52.5</td>
</tr>
<tr>
<td>Activity</td>
<td>15</td>
<td>7.5</td>
</tr>
<tr>
<td>Cold stress</td>
<td>10</td>
<td>7.5</td>
</tr>
<tr>
<td>Energy cost of digestion</td>
<td>8</td>
<td>17.5</td>
</tr>
<tr>
<td>Energy stored</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>Energy excreted</td>
<td>12</td>
<td>20</td>
</tr>
<tr>
<td><strong>Total requirements</strong></td>
<td><strong>120</strong></td>
<td><strong>130</strong></td>
</tr>
</tbody>
</table>

PROTEIN NEEDS

• Parenteral nutrition now started within 24 hours of birth

• Trend is to start enteral nutrition earlier than in the past and be more aggressive in enteral feeding advancement
## RECOMMENDED PROTEIN INTAKE

<table>
<thead>
<tr>
<th>Weight</th>
<th>g/Kg/day</th>
<th>g/100 kcal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight &lt;1000 gm</td>
<td>4.0-4.5</td>
<td>3.6-4.1</td>
</tr>
<tr>
<td>Weight 1000-1800 gm</td>
<td>3.5-4.0</td>
<td>3.2-3.6</td>
</tr>
</tbody>
</table>

European Society for Pediatric Gastroenterology, Hepatology and Nutrition

2010

Also endorsed by US experts
OPTIONS FOR FEEDING

1. Preterm human milk
2. Donor milk
3. Premature formula or fortified breastmilk
4. Fortification beyond the 24 calories
5. Post-premature formula
6. ELBW
SELECTING APPROPRIATE FEEDING:

• Preterm Human Milk is specially suited to the preterm infant’s higher protein and calorie needs for the first two to four weeks.
“The potent benefits of human milk are such that all preterm infants should receive human milk. Mother’s own milk, fresh or frozen, should be the primary diet, and it should be fortified appropriately for the infant born weighing less than 1.5 kg. If mother’s own milk is unavailable despite significant lactation support, pasteurized donor milk should be used”

AAP Recommendation 2012
ADVANTAGES OF HUMAN MILK

• Establishes healthy intestinal flora
• Contains factors that stimulate intestinal growth, motility and maturation
• Improves transition to enteral feedings (off IVs sooner)
• Reduces incidence of necrotizing enterocolitis and sepsis
  • 6-10x less likely with Maternal Breast Milk
  • 4x less likely with Donor Breast Milk
• Lactoferrin- binds to iron making it less available
SELECTING APPROPRIATE FEEDING:

• Preterm infant born less than 1500 grams get fortified breast milk or premature formula once they are on full feeding volume

• Fortifier (HMF) designed to be used until the infant reaches 2000-2500 grams body weight. (Use past that weight puts the infant at risk for toxicity of fat soluble vitamins)

• Fortifier made from human milk now available
FORTIFICATION:

• Many preemies need fortification beyond the 24 calories in premature formulas in order to achieve adequate weight gain

• Various ways to do this depend on:
  • Baby’s clinical situation
  • Hospital formula contract
  • Local availability of products after DC

• Call nutritionist at Level III center for questions/recipes.
POST-PREMATURE FORMULA

• If the infant is formula feeding, both formula companies make 22-calorie formulas that are higher in protein, vitamins and minerals than standard formula, but not as rich as premature formulas.
ELBW INFANTS

• The extremely low birthweight infant (ELBW, < 1000 grams) poses a challenge for bone mineralization

• Some of these babies have been found to have fractures later if exclusively breastfed

• Nutritionists recommend some fortification for them through the first year of life.
SOY-BASED FORMULA (LACTOSE-FREE)

• Not appropriate for preterm infants born weighing less than 1800 grams

• Substantial cross allergy to soy among people with lactose allergy
ELEMENTAL FORMULAS

- Neonates with fat malabsorption or intolerance to milk/soy based formulas
- Type of hypoallergenic infant formula made from individual amino acids.
- Examples:
  - Post necrotizing enterocolitis
  - Short bowel syndrome
GASTROINTESTINAL/NUTRITIONAL: FEEDING INTOXICANCE

• May be mild and respond to nursing measures or serious and require medical intervention
FEEDING INTOLERANCE

• Signs and Symptoms:
  • Residuals: Current thinking is to tolerate up to 50% of previous feed if no associated GI symptoms
  • Emesis
  • Abdominal distension
  • Diarrhea
  • Increased apnea and/or bradycardia
  • Reflux
THE FEAR: A PERFECT STORM
NECROTIZING ENTEROCOLITIS
GASTROINTESTINAL/NUTRITIONAL: NECROTIZING ENTEROCOLITIS (NEC)

• NEC is an idiopathic disorder characterized by inflammation and necrosis of the mucosal layers of the gastrointestinal tract.

• Any part of the bowel can be affected, but most often the distal ileum and proximal colon are involved.
NECROTIZING ENTEROCOLITIS (NEC)

• What causes NEC?
  • We still do not know for sure, the etiology has been debated and researched for many years.
  • Some ischemic or hypoxic insult to gut?
  • Bacteria involved
  • Indomethacin?
NECROTIZING ENTEROCOLITIS (NEC)

- Who is at risk for NEC?
  - Prematurity is the greatest risk factor
  - Smaller babies born weighing less than 1500 grams make up 70% of cases, but it can happen to a term infant
  - 90-95% have been fed
NECROTIZING ENTEROCOLITIS (NEC)

• Signs and Symptoms
  • Feeding Intolerance
    • Abdominal Distension
    • Visible Loops of Bowel
    • Blood in Stool
    • Gastric Residuals
    • Vomiting
NECROTIZING ENTEROCOLITIS (NEC)

• Signs and Symptoms
  • General systemic signs
    • Lethargy
    • Apnea/bradycardia
    • Temperature instability
    • Hypotension/hypoperfusion
NECROTIZING ENTEROCOLITIS (NEC)

• Signs and Symptoms
  • Evidence of peritonitis
    • Erythema or discoloration
    • Abdominal tenderness

• Can move very fast! Any of these signs warrant a careful look for others!
NECROTIZING ENTEROCOLITIS (NEC)

• Diagnostic Tests
  • X-Ray
    • Dilated bowel loops with thickened walls due to local edema
    • Pneumatosis intestinalis occurs when gas-forming bacteria invade the intestinal wall (diagnostic of NEC)
    • Pneumoperitoneum occurs with bowel perforation
pneumatosis
Pneumoperitoneum – free air in abdomen
NECROTIZING ENTEROCOLITIS (NEC)

- Diagnostic Tests
  - Blood culture
  - CBC
  - Electrolytes
NECROTIZING ENTEROCOLITIS (NEC)

- **Medical Management**
  - NPO for bowel rest (often for 7-10 days)
  - Gastric decompression
  - Antibiotics
  - Serial X-Rays
  - Careful monitoring of blood glucose levels
NECROTIZING ENTEROCOLITIS (NEC)

• Medical Management
  • Respiratory support and correction of acidosis
  • Circulatory support for hypotension: hydration and inotropes
  • Blood components to correct thrombocytopenia and DIC
  • Pain management
NECROTIZING ENTEROCOLITIS (NEC)

- **Surgical Management**
  - Resection of necrotic bowel
  - Creation of a stoma if necessary
  - Drain
NECROTIZING ENTEROCOLITIS (NEC)

• Prevention
  • Prevent prematurity
  • Encourage pumping and breastfeeding, as antibodies in breast milk provide some protection
  • Donor milk if mother’s milk is inadequate or unavailable
  • Careful use of oral medications
  • Careful infection control, as NEC does occur in clusters
NECROTIZING ENTEROCOLITIS (NEC)

• Prevention
  • Various philosophies exist around feeding practices
    • Delaying feedings delays NEC, but does not prevent it
    • Excessive volume or rapid advancement of feeding may be the critical factors rather than early feeding
NECROTIZING ENTEROCOLITIS (NEC)

• Prevention
  • Slow advancement of feeding volumes is probably the best approach, although there is no uniform standard
  • One cautious approach recommended in the literature is to initiate small volume feedings and advance in increments of not > 20 ml/Kg/day
  • Any advancement protocol better than none
GAVAGE: INDWELLING CATHETER
GAVAGE FEEDING

- Soft indwelling 5 or 6 Fr. Silicone or polyurethane catheter – smallest size that works

- Orogastric vs. nasogastric tubes

- For safety, enteral tubings must not be physically compatible with IV connections (JCAHO Sentinel Event Alert)

- Can be left in to complete feeds of infants who are learning to nipple
GAVAGE FEEDING

• Deliver feeding slowly over 20-30 minutes
• Social contact important during feeding as directed by infant
  • Kangaroo care
  • Holding or hand swaddling
  • Offer tastes and smells of human milk
PROGRESSION TO ORAL FEEDING

• Bottle feeding usually plays a role in getting a preemie to full oral feeding so they can go home, especially if mother cannot be in the hospital round the clock

• Also important because of need for continued fortification

Courtesy Children’s Medical Ventures
NIPPLE FEEDING:
WHAT MAKES IT HARD FOR A PREMIE?

• Weaker muscle tone
• Suck, swallow immature
• Lack flexion
• Absent or reduced sucking pads
• Few opportunities for positive oral sensation
• Absent or immature reflexes
• Limited energy stores
PROGRESSION TO ORAL FEEDING

• Feeding should be Infant-Directed based on feeding cues
• Parents are primary feeders of infants – involve them from the beginning
• Provide early positive sensory experiences
• Recognition of engagement/disengagement cues is critical
FEEDING CUES

1. EARLY CUES: "I'm hungry"
   - Crying
   - Mouth opening
   - Turning head
   -Seeking/ rooting
   - Calm crying baby before feeding
   - Cuddling, skin-to-skin on chest
   - Soothing

2. MID-CUES: "I'm really hungry"
   - Stretching
   - Increasing movement
   - Hand to mouth

3. LATE CUES: "Calm me, then feed me"
   - Crying
   - Lots of movement
   - Color turning red

LOOK FOR EARLY FEEDING CUES
PROGRESSION TO ORAL FEEDING

• Feeding readiness
  • 32-34 weeks corrected age
  • Physiologic stability during cares and during non-nutritive sucking
  • Alertness at feeding time
  • Feeding cues may be subtle
PROGRESSION TO ORAL FEEDING

• Cues to STOP nipple feeding
  • Falling asleep
  • Apnea/Bradycardia
  • Color change
  • Loss of tone
  • Lack of response to stimulation of rooting response
  • Time out signal from baby
• Goal is good quality feeding not necessarily volume
NIPPLE FEEDING: ENHANCING SUCCESS

- Consistent team approach
- Quiet environment
- Watch for appropriate infant state
- Time
- Frequent burping/pacing
- Flow/nipple choice – start with low-flow
- Positioning – swaddle to support flexion
- Good breastfeeding support and a plan for the transition home
- Lactation consult
TECHNIQUES THAT TAKE THE INITIATIVE AWAY FROM THE BABY

• Twisting/turning the bottle
• Moving nipple up/down
• Moving nipple in/out of baby’s mouth
• Massaging chin or stimulating the face
DISCHARGE/FOLLOWUP ISSUES

Remember: Day of discharge almost as stressful as admission. Don’t leave teaching til last day!!
DISCHARGE/FOLLOWUP ISSUES

- Do you have a followup pediatrician?! 
- Infant safety & CPR class 
- Immunizations, on same schedule as term infants 
- Post discharge follow-up appts with Lacation
DISCHARGE/FOLLOWUP ISSUES

ROP Exams

• Communication about follow-up extremely important
DISCHARGE/FOLLOWUP ISSUES

• Car Seat Test recommended by AAP for infants born at less than 37 weeks gestational age or 2500 grams
CAR SEAT TEST – 2009 AAP REVISED GUIDELINES

• 90 – 120 min or duration of travel time if greater
• Advise parents to use car seat for travel only
• Advises monitoring in car bed if that is used
• Crash protection of car seats better documented than that of car beds
• Advises considering retesting before transition to car seat from bed
• Avoid similar upright equipment like swings, infant seat, carriers until baby is larger

Pediatrics Vol 123, #5, May 2009
DISCHARGE/FOLLOWUP ISSUES

• Bed flat before baby goes home
• No sheepskins or fluffy bedding
• BUT, they need play time on their tummy while they are awake!

“Back to Sleep” unless otherwise ordered
DISCHARGE/FOLLOWUP ISSUES

• Susceptibility to RSV
  • Teach parents to wash hands and protect baby from smoke and people with colds
DISCHARGE/FOLLOWUP ISSUES

Synagis Criteria – 2014 revisions

• Less than 1 year old at start of RSV season:
  • Born before 29 weeks gestation
  • Born 29 weeks, 0 days to 31 6/7 weeks with supplemental oxygen requirement for 28 days
  • Some hemodynamically significant CHD
  • Consider for anatomic pulmonary abnormality or neuromuscular disorder

• Others
  • Depending on AK RSV epidemiology, possibly treat more Alaska Natives
  • Same for some American Indian populations – little data

• Not recommended in second year of life except for some patients with CLD on continuing oxygen, steroids, or diuretics

Pediatrics, August 2014
HEARING SCREEN:
ALL BABIES WHO HAVE BEEN IN INTENSIVE CARE ARE AT INCREASED RISK

- Make sure they get their hearing screen before discharge
- Increased incidence of both sensorineural and conductive hearing loss
- Hearing loss occurs both in the newborn period and during the first year
DISCHARGE/FOLLOWUP ISSUES

- High Risk Followup Clinic
  - VLBW (<1250 grams)
  - 30 weeks gestational age or less
  - Bronchopulmonary dysplasia/significant mechanical ventilation
  - CNS complications: IVH, PVL, seizures, meningitis
  - Prenatal drug exposure
DISCHARGE/FOLLOWUP ISSUES

- Feeding Plan / Growth Monitoring
- Physical and Occupational Therapy
- Developmental milestones assessment
THE LONG JOURNEY HOME
REFERENCES


REFERENCES CONT

• Perinatology. 36(4):737-62.

