Overcoming Barriers and Optimizing Nutrition Support in the Critically Ill Child

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Pediatric ICU
RD, CSP, CNSC, CLE
Learner Objectives:

1. Describe alterations in metabolism in critical illness affecting energy and nutrient needs
2. Evaluate methods for estimating and measuring energy requirements in critically ill children
3. Identify barriers and strategies for delivery of optimal nutrition support in critically ill children
4. Implement best practices for nutrition support of the critically ill child
Who’s Who in the PICU? Typical Diagnoses of PICU patients

- Neurologic alterations: status epilepticus, altered mental status, acute increased ICP, head injury, cerebral edema
- Pulmonary: acute respiratory failure, status asthmaticus, upper airway obstruction, apnea
- Cardiovascular: cardiopulmonary arrest, hypotension, ALTE
- Endocrine: DKA, new onset DM
- General: non accidental trauma, drowning, overdose
- Infectious: sepsis, meningitis, bacterial tracheitis, VPS infection
- Fluid & electrolyte abnormalities: severe dehydration, metabolic, acid-base disturbance
Identifying High Risk Patients:

- Anticipated prolonged PICU LOS >48hrs
- Prolonged duration of mechanical ventilation or escalation of respiratory support
- Surgery anticipated
- High risk diagnosis and/or comorbidities - trauma, burns, cardiac anomalies, IBD, CF
- Malnutrition on admission, moderate or severe based on Zscores, BMI <5\(^{th}\)%
- Obesity >95\(^{th}\)% BMI or BMI >30 in older adolescents, with 1 or more comorbidities

Skillman, Wischmeyer JPEN 2008 32:520; Joosten, Hulst Nutrition Assessment of the Critically Ill Child in Pediatric Critical Care Nutrition 2015
Determining Nutritional Goals:

How are nutrient needs altered in critical illness?
Metabolic Stress Response:

• Metabolic response to stress from sepsis, trauma, burns or surgery is commensurate with the degree and duration of injury and the status of the host

• Characterized by “ebb and flow” phases:
  - initially pt at risk for hypovolemia, shock, tissue hypoxia, decreased cardiac output, hypothermia
  - flow phase includes increased production of cytokines, acute phase proteins, counter regulatory hormones, insulin and growth hormone resistance

• Increased protein catabolism and turnover-skeletal muscle primary source of FAA for gluconeogenesis, acute phase proteins; catabolism exceeds synthesis → neg N2 bal, wt loss
Metabolic Stress Response

- Ketones: Fuel for brain
- Lipolysis:↑ Fatty acids
- Loss of lean body mass
- Acute inflammatory proteins
- Trauma
- Sepsis
- Burn
- Surgery

Critical illness

Muscle breakdown
- Amino acids
- Protein synthesis
- Gluconeogenesis
- Urea
- Fuel for brain, RBC, and kidneys
- Glycolysis:↓ Utilization
- ↑↑ Glucose
- Hyperglycemia

TISSUE REPAIR
WOUND HEALING

Source: Praveen S. Goday, Nilesh M. Mehta: Pediatric Critical Care Nutrition: www.acesspediatrics.com
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Acute Metabolic Response to Tissue Injury

- Cytokines ↑
- Counterregulatory hormones ↑
- GH ↑/IGF-1 ↓
- Constitutive proteins ↓ (Prealbumin ↓)
- Acute-phase reactants ↑
  (C-reactive protein ↑)
- LIVER
- Amino acid pool
- Resting energy expenditure ↑
- Muscle mass ↓ (Urinary 3MH ↑)
- Gluconeogenesis ↑
  (Total urinary nitrogen ↑)
- Fatty acid oxidation ↑ and carbohydrate oxidation ↑

Source: Praveen S. Goday, Nilesh M. Mehta: Pediatric Critical Care Nutrition: www.accesspediatrics.com
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Altered Metabolism in Critical Illness

- Response to injury/stress results in increased counter-regulatory hormones, insulin/growth hormone resistance, catabolism of endogenous protein stores, CHO and fat to provide substrates for the stress response.

- Metabolic response to stress, injury, surgery or inflammation cannot be accurately predicted and metabolic alterations may change through the course of illness- ebb/flow phase, risk of over or under feeding.

- Nutrition support cannot reverse or prevent this response however failure to provide optimal nutrition during this stage may exacerbate nutritional deficiencies and result in malnutrition, which may affect clinical outcomes.

- Goals of nutrition support: augment the stress response, prevent long term harmful consequences.

Winkler, Malone; Krause’s Food & Nutrition Therapy 2008: 1027
Malnutrition and the Risk of Underfeeding:

- Incidence of malnutrition: 30% of hospitalized children
- ASPEN definition of pediatric malnutrition (undernutrition):
  - “An imbalance between nutrient requirement and intake, resulting in cumulative deficits of energy, protein or micronutrients that may negatively affect growth, development and other relevant outcomes”
  - Acute, chronic (>3 months), or mixed - reflects time period; illness/non-illness related or both
- Based on growth chart Z-scores (standard deviations from the median) replacing percentiles and Waterlow Criteria
- Mild (acute event or illness); moderate (longer duration affecting BMI or wt/length); severe (prolonged, decline in linear growth/stunting)

Consensus Statement AND/ASPEN; NCP 2014; 30 (1):147-161
Risk of Underfeeding and Malnutrition in Critically Ill Children:

- Loss of critical lean body mass and subcutaneous fat
- Gastrointestinal dysfunction
- Poor wound healing
- Increased risk of infections with poor immune response
- Increased mechanical ventilation days
- Increased LOS
- Repeat hospital admissions
- Increased risk of developing multi-organ failure
- Increased mortality
Risks of Overfeeding in Critically Ill Children:

- Metabolic response to stress is **variable**-hypermetabolism is unpredictable and unlikely to be sustained over the PICU stay with the exception of specific subsets of patients, ie burn injury, traumatic brain injury
- Critically ill children may not have the usual energy needs of a healthy child for growth, work of breathing and physical activity (mechanical ventilation, sedation, neuromuscular blockade, bedrest, inhibition of anabolism)
- Studies have shown post surgical infants and children to be hypometabolic or normometabolic after injury or illness

Overfeeding defined: *calories or substrates that exceed an individual’s requirements to maintain metabolic homeostasis*

- Potential for harm in critical illness - excess caloric delivery shown to increase morbidity and mortality in both pediatric and adult studies
- Excess CHO contributes to lipogenesis if in excess of max oxidation rates, resulting in excess CO2 production, WOB, prolonged mechanical ventilation
- Potential for increased hepatic work load, dysfunction
- Potential for increasing risk of infection secondary to hyperglycemia, altered immune response
TOO MUCH, TOO LITTLE, OR JUST RIGHT?
Question:

Which is the best method for determining energy needs of critically ill children?

a. DRI  
b. WHO equation  
c. Indirect calorimetry  
d. Penn Stat equation
Indirect Calorimetry (IC) “the gold standard”

Gas exchange measurement of energy expenditure and the respiratory quotient: minute to minute whole body O2 consumption (VO2) and carbon dioxide consumption (VCO2)

Weir equation \( MREE = \text{VO2} \times 3.941 + \text{VCO2} \times 1.11 \times 1440 \)

\( \text{RQ} \) (respiratory quotient) = \( \frac{\text{VCO2}}{\text{VO2}} \) ratio

RQ value constant, specific for each substrate (oxidation of fat 0.7, protein 0.83, carbohydrate 1.0; RQ >1 may represent overfeeding, lipogenesis)
Role of IC in Critical Illness:

- Metabolic demands vary widely in critical illness
- Individuals may respond to similar injury states with a diversity of MEE values
- Studies have shown up to 71% of pediatric critically ill patients meet indications for IC
- Predictive equations frequently overestimate daily energy expenditure, even those derived from pediatric populations and may underestimate in some
- 83% incidence of overfeeding by predictive equations compared to IC with cumulative excess of 8000 kcals/week

Table 2. Suggested Criteria for Selecting Patients for Indirect Calorimetry in the Pediatric Intensive Care Unit.

<table>
<thead>
<tr>
<th></th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Underweight (BMI &lt;5th percentile for age), at risk of overweight (BMI &gt;85th percentile for age), or overweight (BMI &gt;95th percentile for age)</td>
</tr>
<tr>
<td>2</td>
<td>&gt;10% weight gain or loss during medical-surgical intensive care unit stay</td>
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<tr>
<td>3</td>
<td>Failure to consistently meet prescribed caloric goals</td>
</tr>
<tr>
<td>4</td>
<td>Failure to wean or escalation in respiratory support</td>
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<tr>
<td>5</td>
<td>Need for muscle relaxants for &gt;7 days</td>
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<tr>
<td>6</td>
<td>Neurologic trauma (traumatic, hypoxic, and/or ischemic) with evidence of dysautonomia</td>
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<tr>
<td>7</td>
<td>Oncologic diagnoses (including stem cell or bone marrow transplantation)</td>
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<tr>
<td>8</td>
<td>Need for mechanical ventilatory support &gt;7 days</td>
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<tr>
<td>9</td>
<td>Suspicion of severe hypermetabolism (status epilepticus, hyperthermia, systemic inflammatory response syndrome, dysautonomic storms) or hypometabolism (hypothermia, hypothyroidism, pentobarbital or midazolam coma)</td>
</tr>
<tr>
<td>10</td>
<td>Intensive care unit length of stay &gt;4 weeks</td>
</tr>
</tbody>
</table>

BMI, body mass index. Adapted with permission from Mehta et al.³
### Table 1. The Correlation Between Predictive Equations to Indirect Calorimetry.

<table>
<thead>
<tr>
<th>Study</th>
<th>Trial</th>
<th>Population</th>
<th>Age</th>
<th>n</th>
<th>Comparing Predictive Equations to Indirect Calorimetry</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gebara et al, 1992²⁵</td>
<td>Prospective cohort</td>
<td>PICU, cardiac</td>
<td>19.4 (2-146) moᵃ</td>
<td>26</td>
<td><strong>Predictive Equations</strong></td>
</tr>
<tr>
<td>Coss-Bu et al, 1998²⁴</td>
<td>Prospective cohort</td>
<td>PICU, centilated</td>
<td>5.7 ± 5.9 yᵃ</td>
<td>55</td>
<td>Talbot</td>
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<td></td>
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<td>HBE (SF: 1.5)</td>
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<td>HBE (SF: 1.3)</td>
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<td></td>
<td></td>
<td>Talbot (SF: 1.5)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Talbot (SF: 1.3)</td>
</tr>
<tr>
<td>Avitzur et al, 2003²²</td>
<td>Prospective cohort</td>
<td>PICU, cardiac</td>
<td>Cyanotic: 3.2 mo (0.2-13)ᵃ</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>Noncyanotic: 12.3 mo (0.1-30)ᵃ</td>
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<td></td>
<td></td>
<td>29</td>
<td>WHO</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Schofield</td>
</tr>
<tr>
<td>Mehta et al, 2009⁴</td>
<td>Retrospective cohort</td>
<td>PICU</td>
<td>11.2 y (1.6 mo-32 yᵃ)</td>
<td>14</td>
<td>Bland–Altman</td>
</tr>
<tr>
<td>de Wit et al, 2010²⁶</td>
<td>Prospective cohort</td>
<td>PICU, cardiac</td>
<td>7.3 ± 10.27 moᵃ</td>
<td>21</td>
<td>Schofield</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Schofield w/ SF</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>WHO</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>White equation</td>
</tr>
<tr>
<td>Mehta et al, 2011²⁰</td>
<td>Prospective cohort</td>
<td>PICU</td>
<td>2 y (0.1-25.8 yᵇ)</td>
<td>33</td>
<td>Bland–Altman</td>
</tr>
</tbody>
</table>

HBE, Harris–Benedict equation; PICU, pediatric intensive care unit; SF, stress factor; WHO, World Health Organization equation.

ᵃMean (range) ± SD.
ᵇMedian (range) ± SD.

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Assessment of Energy Needs: *Know What You Are Estimating*

\[ TEE = \text{BMR} + \text{SDA} + E \text{ (activity)} + E \text{ (growth)} + E \text{ (losses)} \]

- **REE** = BMR + 10%
- **BMR** = 60-70% of TEE
- **SDA** = 8-10% specific dynamic action of food (energy required to absorb, process, store nutrients)
- **E** = growth (30-35% of TEE in infants, decreasing to 10% in later adolescence)
- **E** = physical activity
- **E** = losses (energy lost in stool, urine, fistulae, ostomy or CT losses)
Estimating Energy Needs – Predictive Equations:

- Equations derived from measurements of BMR in healthy children, +/- stress factors based on adult research
- Harris-Benedict: (1919 based on measurement of 97 infants <8days old and 239 individuals >16yrs; equation never validated in children)
- FAO/WHO/UNU: (based on 6100 individuals, 1985)
- Schofield: (based on FAO/WHO/UNU report with additional data, 1985)
- Talbot: (BMR measurements in children, 1938)
- White: (measurements on 100 ventilated critically ill children, includes body temp factor, 2000)
Assessment of Energy Needs in Absence of IC:

- Intubated Infants 0-12 months-may require more than REE; consensus to provide 75-80% DRI/age
- Intubated >12 months-may use equations to predict REE (WHO, Schofield, White) usually without activity or injury factors
- Post extubation-may increase stepwise to DRI for actual weight or IBW if catch up growth is needed
- If >85th% BMI for older children, consider DRI for IBW or BMI/age at 50th%
  
  \[(\text{BMI } @50^{\text{th}}\% \times \text{actual wt}) / \text{actual BMI}\]

Mehta, et al  JPEN 2009 33: 260
## Nutrient Requirements: EER

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Gender</th>
<th>WHO</th>
<th>Schofield</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3</td>
<td>Male</td>
<td>60.7W – 54</td>
<td>0.17W + 15.7H – 617.6</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>61W – 51</td>
<td>16.25W + 10.232H – 413.5</td>
</tr>
<tr>
<td>3-10</td>
<td>Male</td>
<td>22.7W + 495</td>
<td>19.6W + 1.303H + 414.9</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>22.5W + 499</td>
<td>16.97W + 1.618H + 371.2</td>
</tr>
<tr>
<td>10-18</td>
<td>Male</td>
<td>17.5W + 651</td>
<td>16.25W + 1.372H + 515.5</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>12.2W + 746</td>
<td>8.365W + 4.65H + 200</td>
</tr>
</tbody>
</table>

### Activity Factors

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th>Stress Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paralyzed</td>
<td>1.0</td>
<td>Surgery</td>
<td>1.2-1.5</td>
</tr>
<tr>
<td>Confined to Bed</td>
<td>1.1</td>
<td>Infection</td>
<td>1.2-1.6</td>
</tr>
<tr>
<td>Ambulatory</td>
<td>1.2-1.3</td>
<td>Trauma</td>
<td>1.1-1.8</td>
</tr>
</tbody>
</table>

### Stress Factors

- Burns: 1.5-2.5
- Starvation: 0.7
- Growth Failure: 1.5-2.0

The ASPEN Pediatric Core Curriculum, ASPEN, 2010*
Alterations in Fluid Needs:

- Intubated patients receive humidified air, decreased insensible losses from respiration (75-80% of maintenance)

- Need for fluid restriction in SIADH-increased risk in CNS injury characterized by hyponatremia, low serum osmolality, increased urine Na

- Renal failure-anuria or oliguria-fluids limited to insensible losses (1/3 maintenance) + urine output if any

- Increased fluid needs for resuscitation/hypotension, excessive GI output, hyperthermia, certain medications for renal clearance
Assessment of Fluid Needs in Critical Illness:

- Holliday-Segar formula – estimate of basal/maintenance fluid needs only in healthy children

- Cumulative positive fluid balance associated with increased duration of mechanical ventilation, worse outcomes in children with ALI

- Fluid needs are dynamic - nutrition support interventions are dependent on medical team fluid management

Question:

There is a 25-100% increase in the need for which nutrient in critical illness?

a. Glucose
b. Zinc
c. Protein
d. Glutamine
Assessment of Protein Needs in Critical Illness:

- Significant increase in muscle protein catabolism and turnover during inflammatory stress response and recovery from surgery or trauma
- Critically ill children at risk for net negative N2 balance, loss of lean tissue weight, immune dysfunction
- Protein requirements potentially 25-100% greater in critically ill infants and children
- Delivery of >60% of prescribed protein intake associated with lower odds of mortality in mechanically ventilated patients
- Goals to optimize protein delivery to modulate protein catabolism

### Protein Requirements:

<table>
<thead>
<tr>
<th>Age</th>
<th>DRI</th>
<th>Critical Illness</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-6mo</td>
<td>1.5g/kg/day</td>
<td>2-3g/kg/day</td>
</tr>
<tr>
<td>7-12mo</td>
<td>1.2g/kg/day</td>
<td>2-3g/kg/day</td>
</tr>
<tr>
<td>13-23mo</td>
<td>1.05g/kg/day</td>
<td>2-3g/kg/day</td>
</tr>
<tr>
<td>2-3yrs</td>
<td>1.05g/kg/day</td>
<td>1.5-2g/kg/day</td>
</tr>
<tr>
<td>4-13yrs</td>
<td>0.95g/kg/day</td>
<td>1.5-2g/kg/day</td>
</tr>
<tr>
<td>14-18yrs</td>
<td>0.85g/kg/day</td>
<td>1.5g/kg/day</td>
</tr>
</tbody>
</table>

Question:
What is the best route for providing early nutrition in critical illness?

a. Parenteral solution via peripheral line
b. Enteral formula via gastric feeding tube
c. Enteral formula via post-pyloric feeding tube
d. Parenteral solution via central line
Adequate Enteral Protein Intake Inversely Associated with 60 day Mortality in Critically Ill Children

- 1245 patients from 59 PICU’s from 15 countries
- 1mo to 18 years (median age 1.7 years)
- Mechanically ventilated for >48hrs
- 985 received EN, 354 (36%) received EN via post pyloric route
- Mean prescribed energy and protein was 69 kcals/kg and 1.9g pro/kg
- Mean delivery of enteral energy and protein goals were 36 % and 38%

Adequate Enteral Protein Inversely Associated with 60 day Mortality in Critically Ill Children

• “Delivery of >60% of prescribed protein intake associated with lower odds of mortality in mechanically ventilated children. Optimal prescription and modifiable practices at the bedside might enhance enteral protein delivery in the PICU with a potential for improved outcomes.”

• “Early initiation, post-pylorics route, shorter interruptions, larger PICU size, a dedicated dietitian in the PICU, were associated with higher enteral protein delivery”

Nutrition Support Considerations:

APPROPRIATE TIME?

APPROPRIATE ROUTE?

APPROPRIATE FORMULATION?
Benefits of EEN (Early Enteral Nutrition):

- EEN - minimal or early enteral nutrition defined as 25% of caloric goals within 48 hours of admission to PICU correlated with decreased mortality
- Correlated with decreased time to achieving goal feeds
- Preserves gut mucosal integrity
- Decreased nosocomial infection risk compared with PN
- Promotes anti-inflammatory effects by decreasing expression of cytokines (IL-6)
- Cost effective and physiologic

Relative Indications for PN

- **Vasoactive, inotropic support:**
  Potential for decreased splanchnic blood flow with escalating pressor support? may consider trophic EN if post adequate fluid resuscitation, steady, low dose or decreasing pressor support

- **ECMO:**
  Concern for NEC and effect of hypoxia on the gut-studies in infants on ECMO receiving EN tolerated feedings without developing NEC or intestinal perforation

- **Severe sepsis, pulmonary disease, CHD, peritoneal dialysis**

- **Supplemental nutrition support:** for malnourished child unable to achieve or tolerate goal EN after 3-5 days
Prudent Considerations for Feeding the Septic/Critically Ill Child

- Impact of sepsis on gut mucosal perfusion?
  - hypovolemia - role in decreasing splanchnic blood flow
  - mechanical ventilation - reduced splanchnic blood flow with increased intra-thoracic pressure
  - inotropes/pressors have variable effect - concern for small bowel necrosis with preferential shunting of blood to heart, brain

- EN the preferred route, however consider initiate after adequate fluid resuscitation and HD stability, steady or decreasing vasoactive/inotrophic infusion, no fluid boluses for past 24-48hrs
It’s Time to Feed!

- Which route? NGT, OGT, NJT, GTT
- Risk of aspiration or reflux?
- Continuous vs bolus?
- What are fluid goals/limits?
- H/o allergies, intolerance or formula feeds PTA?
- Age appropriate formula? consider “wt age” of patient
- Formula type? isotonic preferred initially, establish gut tolerance-none specific for critical illness in children
- Medications? decreased gut motility with sedation, muscle relaxants-benefit of whey based formulations
# Gastric or Postpyloric Feeding

<table>
<thead>
<tr>
<th></th>
<th><strong>Advantages</strong></th>
<th><strong>Disadvantages</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gastric Feeding</strong></td>
<td>Faster initiation</td>
<td>Risk of aspiration</td>
</tr>
<tr>
<td></td>
<td>Easier tube placement</td>
<td>in pts with severe</td>
</tr>
<tr>
<td></td>
<td>Physiologic, tolerated</td>
<td>GER, delayed</td>
</tr>
<tr>
<td></td>
<td>Able to meet goals</td>
<td>emptying, un-protected airway</td>
</tr>
<tr>
<td><strong>Postpyloric Feeding</strong></td>
<td>Shorter time to goal</td>
<td>Delayed initiation</td>
</tr>
<tr>
<td></td>
<td>Reduced fasting</td>
<td>Increased radiographs</td>
</tr>
<tr>
<td></td>
<td>Decreased PN use</td>
<td>May not prevent aspiration</td>
</tr>
<tr>
<td></td>
<td>May decrease PNA</td>
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</tr>
</tbody>
</table>
Special Considerations:

ECMO

OBESITY
Nutrition Implications of Respiratory Support Modes:

• Definitions of ALI and ARDS:
  Acute onset of hypoxia; Bilateral infiltrates on CXR; Lack of evidence of heart failure as cause of lung disease; PaO2/FiO2 <300 (ALI) and <200 (ARDS)

• Direct or indirect lung injury: direct (pneumonia, aspiration pneumonitis, inhalation injury, traumatic pulmonary contusions); indirect (sepsis, shock, cardiopulmonary bypass)

• Modes of ventilation:
  - non-invasive (HFNC, CPAP, BIPAP)

  Concern for seal of device-not an absolute contraindication to NG or OGT feeds; air delivered inadvertently to the stomach
  - Invasive (CMV, NAVA, HFOV) positive pressure-fluid, Na retention, need for fluid restriction, diuretics; NGT w/ NAVA; need for sedation with HFOV

Nutrition Support with ECMO

- Extracorporeal membrane oxygenation –therapy for profound cardiopulmonary failure using membrane oxygenator and modified heart –lung bypass; metabolic burden during and after substantial and long lasting
- Variability in EE on ECMO however protein turnover is doubled; neonates may lose up to 15% of lean body mass during 7 day course
- IC and N2 balance inaccurate on ECMO
- Guidelines recommend caloric provision be based on age-matched healthy neonates, 100-120 kcals/kg/day
- Adequate provision of protein is most effective strategy in protecting lean body mass, up to 3g/kg/day in neonates

Jaksic, T et al. JPEN 2010;34:247-253
Challenges of the Obese Critically Ill Child:

• **Definitions**: BMI >95\textsuperscript{th}% 2-19 years or BMI of >30, whichever is lower; wt/length >95\textsuperscript{th}% in <2 years of age (overweight)

• **Prevalence** in U.S children and adolescents at 17%; 10% <2 years of age; 11-20% in hospitalized children, 13% who require mechanical ventilation

• **Comorbidities**: cardiovascular, respiratory, endocrine, hematologic, GI, orthopedic, renal, psychosocial

• **Medical Care**: difficulties with airway management, vascular access, medication dosing, fluid management, skin care, need for weight appropriate equipment
Challenges of the Obese Critically Ill Child:

• “Obesity - state of inflammation at baseline, which may lead to weak immune defense resulting in higher rates of acquired infections, impaired wound healing, and decubital ulcers”

• Evidence based guidelines established for critically ill adults but not for critically ill obese children

• Suggested protein intake 2-2.5g pro/kg/ IBW for critically ill obese adults -may be appropriate approach in children but further studies needed

• Existing equations may over or underestimate caloric requirements; when feasible, IC should be conducted

• Hypocaloric nutrition therapy and weight reduction during critical illness is not recommended

Question:

What are the most common barriers to meeting nutrition support goals in critically ill children?

a. Holding of enteral feeds for perceived GI Intolerance
b. Fluid restriction
c. Interruptions for extubation/intubation or procedures
d. Undocumented, unspecified
Barriers to Optimal Enteral Nutrition:

• Symptoms of potential intolerance:
  emesis, diarrhea, gastric residuals, abd girth or discomfort

• Feeding tube access:
  dislodgement or displacement, blocked or leaking feeding tubes

• Pre-procedural fasting times (risk of aspiration):
  extubation, intubation, radiology, OR, bedside procedures w/ sedation

• Fluid restriction:
  limited volume for nutrition support if fluid overload, high volumes required for medications and drips

• Hemodynamic instability:
  concern for splanchnic blood flow with hypotension and escalating pressor support
Barriers to Optimal Enteral Nutrition:

• **Unavoidable interruptions**: intubation/extubation, anesthesia, hemodynamic instability, pneumatosiss intestinalis, significant GI bleeding, vomiting, aspiration

• **Avoidable interruptions**: feeding orders not resumed post procedure, unclear guidelines for holding feeds and intolerance, holding for neuromuscular blocking agents
Figure 1. Frequency of causes of interruptions to nutrition therapy.
Overcoming Barriers to Optimal Nutrition:

- Unit specific feeding protocols or algorithms
- Checklists
- Dietitian participation in medical team rounding
- Early documentation and communication of feeding goals
- Clarification of feeding orders
- Education to medical and nursing staff
- Nursing advocacy
- Family/patient advocacy
- Data collection-participation in collaborative research studies - sharing the “evidence”

Abad-Jorge ICAN 2013; 5: 221
Monitoring

- Prealbumin and CRP measurements-serial measurements may help determine the shift from catabolic to anabolic state and subsequent changes in energy needs-inverse relationship with CRP

- Prealbumin *NOT* a sensitive marker of nutrition adequacy in inflammation; levels increased with renal disease, corticosteroids, oral contraceptives; levels decreased with liver disease, dialysis, pregnancy, hyperthyroidism, hyperglycemia

- N2 balance study-24hr urine collection for UUN

Davis, et al  JPEN 2012; 36: 197
Monitoring of Nutrition Support:

- I/O: fluid balance, calories and protein delivered vs prescribed? assess for cumulative deficits, stool pattern
- Anthropometrics: weights variable with fluid shifts, edema, ascites; mid upper arm circumference or TSF in selected patients
- Nutrition support orders: are feeding orders accurate, complete? Monitor for unnecessary interruptions, prolonged restart of feeds
- Labs: electrolytes, prealbumin and CRP trends, serial PN labs, N2 balance
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| 1  | 1A) Children admitted with critical illnesses should undergo nutrition screening to identify those with existing malnutrition and those who are nutritionally-at-risk.  
    1B) A formal nutrition assessment with the development of a nutrition care plan should be required, especially in those children with premorbid malnutrition.                                      | D     |
| 2  | 2A) Energy expenditure should be assessed throughout the course of illness to determine the energy needs of critically ill children. Estimates of energy expenditure using available standard equations are often unreliable.  
    2B) In a subgroup of patients with suspected metabolic alterations or malnutrition, accurate measurement of energy expenditure using indirect calorimetry (IC) is desirable. If IC is not feasible or available, initial energy provision may be based on published formulas or nomograms. Attention to imbalance between energy intake and expenditure will help to prevent overfeeding and underfeeding in this population. | D     |
| 3  | There are insufficient data to make evidence-based recommendations for macronutrient intake in critically ill children. After determination of energy needs for the critically ill child, the rational partitioning of the major substrates should be based upon understanding of protein metabolism and carbohydrate- and lipid-handling during critical illness. | E     |
| 4  | 4A) In critically ill children with a functioning gastrointestinal tract, enteral nutrition (EN) should be the preferred mode of nutrient provision, if tolerated.  
    4B) A variety of barriers to EN exist in the pediatric intensive care unit (PICU) Clinicians must identify and prevent avoidable interruptions to EN in critically ill children.  
    4C) There are insufficient data to recommend the appropriate site (gastric vs post-pyloric/transpyloric) for enteral feeding in critically ill children. Post-pyloric or transpyloric feeding may improve caloric intake when compared to gastric feeds. Post-pyloric feeding may be considered in children at high risk of aspiration or those who have failed a trial of gastric feeding. | C     |
| 5  | Based on the available pediatric data, the routine use of immunonutrition or immune-enhancing diets/nutrients in critically ill children is not recommended.                                                                 | D     |
| 6  | A specialized nutrition support team in the PICU and aggressive feeding protocols may enhance the overall delivery of nutrition, with shorter time to goal nutrition, increased delivery of EN, and decreased use of parenteral nutrition. The affect of these strategies on patient outcomes has not been demonstrated. | E     |
Why Does How We Provide Nutrition Matter?

“Intake of a higher percentage of prescribed dietary energy goal via enteral route was associated with improved 60-day survival, conversely parenteral nutrition use was associated with higher mortality.”

“Pediatric intensive care units that utilized protocols for the initiation and advancement of enteral nutrient intake had a lower prevalence of acquired infections. Optimizing nutrition therapy is a potential avenue for improving clinical outcomes in critically ill children.”

Goals of Nutrition Therapy in the Critically Ill Child

• Minimize the Losses
  Promote *early* implementation of enteral nutrition support to attenuate the obligatory protein and fat catabolism in the stress response to injury

• Prevent “Over” or “Under” feeding
  *Measure* energy expenditure ideally, in absence of IC, give careful consideration for alterations in metabolism during the metabolic stress response phase

• Follow the Moving Target
  Promote improved *adequacy* of calorie and protein delivery by close monitoring of clinical parameters to minimize the complications and promote anabolism with recovery
Thank you!