Objectives

- Define Nutrition Support
- Discuss why nutrition support improves outcomes
- Some of the current evidence based ideas used
- How to apply evidence to practice
The Lore of Past to Science of Present

Before
- Fed calories to supporting the stress response
- Prevent “starvation”
- Simply limiting calorie deficit

Now
- Early enteral feeding
- Attenuate disease severity
- Modulate immune responses
- Possibly affect patient outcomes

What is Nutrition Support
- Supporting patient needs in a disease specific fashion as it relate to a current medical illness
- Delivering nutrition via alternative methods if normal route or gut function is altered
- Nutrition support
  - We ARE NOT “feeding patients”
  - We are supporting the immune modulatory functions of the critically ill or hospitalized patient
Nutrition Support Team

- Nutrition Physician
  - Assists with disease specific concerns, helps to determine the safety of applying nutrition therapy to the patient
- Dietitian
  - Work hand in hand with nutrition physician, researched disease specific concerns about nutrients
- Pharmacist
  - Considers drug interactions, absorption issues related to delivery, composition of parenteral formula for safety
- Bedside Provider
  - Delivers information on delivery, tolerance to enteral feeds, input and output information of patient

Starvation and NPO

- Small bowel villus height begin to shorten and blunt
  - This degradation occurs in a time dependent fashion
- Villus blunting increases mucosal permeability (leaky gut)
  - Associated with systemic endotoxemia, infection, organ dysfunction
- Secretory IgA is diminished in respiratory tract then intestines
  - NO protection against respiratory viral infection (mice model)
- Lymphocytes and cytokine pool is altered
  - Balance of pro-inflammatory to anti-inflammatory responses are shifted to more pro-inflammatory cytokines
- T cells and B cell numbers decrease
- Parenteral nutrition does not stop any of these events
Small Bowel Villi

Blunted Villi after npo
Starvation and NPO

- Lack of feeding in animals demonstrates loss of mucosal defenses
  - Decreases bile salts and IgA
- Bacterial translocation
  - Bacteria in lumen becomes a portal to sepsis
  - Mechanisms are unclear in humans as components of animal model are proved in humans but not entire model yet
- Dysregulation of ligands and cytokines
  - Increases E-selectin and ICAM-1 expression
    - Intestinal and pulmonary microvasculature
    - Increase polymorphonuclear neutrophils accumulation
    - This make a subsequent ischemia-reperfusion event more profound leading to organ injury and failure

Enteral Nutrition IS Gut Preservation

- Enteral feeding stimulates gut health
- Mucosal mass and villus height remain intact
- Stimulates cell proliferation and brush border enzymes
- Keep tight junction between cells intact
- Promote normal enzyme function
- Secretory IgA and bile salts continue to be produced
  - They coat bacteria and prevent their adherence and thus reduce their infectivity
EN limits inflammation

- Enteral nutrition vs. Parenteral Nutrition AND EN versus NPO
  - healthy subjects injected with LPS from *E. coli*
  - EN patients had decrease in inflammatory mediators and increase in anti-inflammatory precursors
  - They actually injected healthy volunteers with LPS
- Pancreatitis patients
  - EN patients had lower CRP and faster resolution of SIRS than TPN
  - Faster decrease in APACHE II scores

Enteral Nutrition and Mortality

- Some meta analysis demonstrated no mortality difference between EN and PN
  - However lower infectious complication rate with EN
  - Several trial do demonstrate mortality improvement with EN depending on disease selection
  - Analysis of early EN versus delayed demonstrated a mortality benefit for early EN
What Nutritional Approach Do These Problems Have in Common

- Cholelithiasis
- Cholestasis
- Hyperglycemia
- Electrolyte imbalances
- Hypertriglyceridemia
- Infection
- Impaired immunity
- Pro-inflammatory state

These are Common Problems Associated with TPN
Total Parenteral Nutrition

- If Enteral is not possible
- If patient were well nourished prior to critical illness
  - Can wait 7-14 day prior to starting TPN
- Every effort for Enteral nutrition should be made
- If TPN used selective underfeeding
  - Low calorie and high protein

When to Feed and Where to Feed?

- Don’t feed during shock and hypoperfusion, goals should be resuscitation so...ensure no ongoing ischemia
- Use of vasopressors is not a contraindication to feeds so long as vasopressor dose is stable or decreasing
- A clinical gut not working (presence of bowel sounds) is misleading because bowels sounds take days to return...bowel sounds are not the hallmark for function
- SO FEED AS SOON AS POSSIBLE AFTER ADMISSION
  - Make a four hour goal from admission for critically ill patients.
Ileus

- Post-operative small bowel can recover function in hours
- Absorption of nutrients into the small bowel does not require intestinal motility
- Nutrients given to small bowel can stimulate motility by causing release of
  - Gastrin, bombesin, motilin, and other promotility hormones
- Ileus does not necessarily need to be NPO
  - Not full rate feeds
  - Find underlying cause (electrolytes, edema, opiates, benzodiazepines, chloride)

Access Devices

- NGT
  - Most common, usually safe to use. Gastric feeding rarely contraindicated
- Nasojujenal Tubes
  - Post-pyloric placement does not prevent or decrease aspiration
  - Best to use is patient fail gastric feeds with motility agents
  - Possibly safer in patient that must be kept supine
  - Possibly improved delivery of nutrient to pancreatitis
- PEG tubes
  - No decrease in aspiration risk
  - Long term access device into stomach
- PEG-J or J-tubes
  - PEG-J may have benefit as there is no direct puncture into jejunum
  - J-tube – common to develop leaks
Gastric Residual

- Newer Literature says don’t bother in that the impact of residual check has not made feeding safer and may have increased aspiration risk
- If checked recommendation are to continue feeding with anything under 500cc
  - For residuals 200-500cc recommend motility agents
- Trophic feeding usually not necessary
  - May be some disease specific conditions that are helpful

How Much to Feed

- Formerly hyperalimentation
  - Formerly tried to give aggressive caloric replacement
  - This worsened outcomes
- Permissive Underfeeding
  - Has improved outcomes
    - Decreased vent days and length of stay
    - Now debate is how long to under feed and what is real caloric goal
- Suggested 20kcal/kg/day of adjusted body weight
  - Probably lower in morbid obesity and perhaps lower for everyone while acutely ill
  - Maybe 8-10kcal/kg/day of actual for obese
- Once improved or after first 5-8 days, can increase to 30-35 kcal/kg/day
Don’t Skimp on Protein

- Meet protein requirements day one
  - At 1 gram aa/kg
  - Acute illness 1.5-2.0 grams aa/kg
  - Possible benefit critical illness 2.5 grams aa/kg
- Short change significantly on lipids
  - In parenteral patient lipids just once weekly avoids essential fatty acid deficiency
- Remainder is carbohydrate

Consideration of Formulation

- Intact versus peptide formulas
  - Stress states can limit ability to handle and process intact protein
  - Peptide based formulas have demonstrated improved tolerances
    - Lower stool volume and higher absolute weight gains
- Medium chain Triglycerides versus Long Chain Triglycerides
  - MCT are absorbed by small bowel more easily
Arginine

- Best studied amino acid
- Theorized to cause hypotension in septic patient via nitric oxide release
  - never demonstrated clinically.
- Outside of sepsis (trauma, wound) supraphysiologic doses (greater than 5 grams a day) confer benefit
- Possible benefit in medical septic patient
  - No idea how high
  - Probably works in concert with other immune enhancing agent and not alone.

Omega-3 Fatty Acids

- Omega-3 and Omega-6 fatty acids are substrates in the arachidonic acid pathway
  - Omega-3 leads to more anti-inflammatory mediators
  - Omega-6 leads to more pro-inflammatory mediators
- Initially benefits seen in several studies with higher omega-3 ratios
- Newer evidence where omega-3 supplemented alone demonstrated no benefit
  - So is there no benefit?
  - Does it need arginine???
L-Glutamine

- Important amino for nitrogen transport and fuel source for gut epithelial cells
- Formerly regarded as conditionally essential in critical illness because its amounts drop significantly in SIRS
- Only small studies demonstrate benefit in reduction of infection for EN supplementation
- Most benefit seen in several Parenteral studies
- However . . . .

Randomized trial of glutamine, selenium, or both, to supplement parenteral nutrition for critically ill patients. BMJ 2011

- Randomized double blinded
- Parenteral glutamine, selenium or both
- No evidence of improvement with glutamine
- Possible improvement in no new infections in parenteral selenium group
REDOX Study

- 40 ICUs, 1223 patients
- Canada, USA, and Europe
- Supplementation provided both IV and enterally
- Glutamine and antioxidants did not improve outcomes
- Glutamine was associated with increase in mortality in critically ill patients with MOF
- New England Journal 2013

Parenteral glutamine supplementation in critical illness: a systematic review.
Wischmeyer et al. Critical Care 2014, 18R76.

- Meta analysis of IV glutamine only; excluded enteral and combined IV and enteral studies
- IV glutamine had trends
  - Hospital mortality reduction
  - Length of stay
    - Hospital and ICU
  - Infectious complication
A Team of One

- Common problem, especially at the community level
- Dietitian by themselves seeing multiple patients
- Dietitian recommendation are often overlooked and ignored
- How do you win?
  - Firm knowledge based on clinical outcome literature
  - Use ASPEN clinical guidelines as a means that is easily referenced
  - Find physician champion in the hospital who is passionate about patient outcome and move forward
Bibliography

- Marik, Paul, MD. Enteral Nutrition in the Critically Ill: Myths and Misconceptions. Critical Care Medicine April 2014, 42(4) 962-969
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