Nutrition Support

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GOALS

- Understand the medical decision processes for evidence based medicine.
- Comprehend the impact of the history and science of Parenteral and Enteral nutrition.
- Optimize outcomes and reduce complications in the nutritional and total care of patients.
OBJECTIVES

- Employ Evidence Based Medicine and other such analytic techniques to optimize patient care.

- Describe the indications for initiating Nutritional Support.

- Delineate the risks and benefits of Parenteral and Enteral support for common diseases.

- Synthesize nutritional support therapies for complex patients.
Definition

- Nutrition = to nurture or grow
- Support = to bolster or strengthen
Definition

- **Energy**
  - Generate ATP
  - Storage
- **Building Blocks**
  - Build lean tissue
  - Create enzymes
- **Supervisors**
  - Hormones
Players

- Energy – Protein 4 cal/gm, Lipid 9cal/gm and Carbohydrate 4 cal/gm
- Building blocks – protein, lipids, trace elements
- Supervisors – insulin, glucagon, growth hormone, IGF, epinephrine, endogenous steroids, prostaglandins, inflammatory mediators and vascular mediators
Definitions

Normal Metabolism

- Normal physiology – insulin
  - Maintain carcass – repair and rebuild
  - Store excess energy for lean times
  - In conjunction with work – build lean tissue
  - Marked by euglycemia and nitrogen balance
- Normal response - evolution
INSULIN

- Anabolic hormone
- Promotes glucose uptake (not rate of oxidation)
- Enhances protein, glycogen and fat synthesis
Metabolism of Starvation

- Driven by low insulin levels
- Sequential and linear decay
  - Consume carcass – no exogenous source of protein or energy
  - Consume energy stores
  - Reduce work – conserve energy
  - Marked by hypoglycemia and negative nitrogen balance
- Events – bedrest, infections, skin breakdown and death – at 70% IBW, about 120 days
Metabolic Response to Stress

- Counter-regulatory Hormones - catecholamines, glucagon, ACTH, etc....
- Ebb - Flow phases
  - Consume carcass – repair and fight infection
  - Consume excess energy
  - Reduce work – conserve energy
  - Marked by hyperglycemia and negative nitrogen balance
- Deleterious - prolongation / sequential
Mediators of the Hyperglycemic Response

- Glucagon
  - Increased gluconeogenesis
  - Increased hepatic glycogenolysis

- Epinephrine
  - Increased gluconeogenesis
  - Skeletal muscle insulin resistance
  - Increased lypolysis and FFA
  - Suppression of insulin resistance
Mediators of the Hyperglycemic Response

- Growth Hormone
  - Skeletal muscle insulin resistance
  - Increased lipolysis
  - Increased gluconeogenesis

- Tumor Necrosis Factor
  - Skeletal muscle insulin resistance
  - Hepatic insulin resistance
Mediators of the Hyperglycemic Response

- Norepinephrine
  - Increased lipolysis
  - Increased gluconeogenesis

- Glucocorticoids
  - Skeletal muscle insulin resistance
  - Increased lypolysis
  - Gluconeogenesis increased

- Cortisol
  - Reduced skeletal muscle insulin sensitivity
Evidence Based Medicine

- Best Practices based on documented efficacy
- Literature review techniques and analysis to derive guidelines.
Definitions - EBM and Guidelines

- “...the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients...”
  - Sackett, *BMJ* 1996

- “Systematically developed statements to assist practitioners and patients decisions about appropriate healthcare for specific clinical circumstances.”
  - Institute of Medicine
Evidence Based Medicine

- Lack of research
- Poor study design
- Faulty analysis
- Misinterpretation
- Extension and/or Misapplication
Alternatives to EBM

- Eminence - grey hair
- Vehemence - beat one’s chest
- Eloquence - Armani suit and big words
- Providence - What Would Jesus Do?

- Diffendence - no problem, mon’
- Nervousness - litigataphobia
- Confidence - Surgeonesque

Issacs and Fitzgerald. *BMJ* 1999
Resources

- AHRQ
- Cochrane Database
- EAST (www.east.org)
  - Primer
Nutrition Guidelines

- ASPEN
- ACCP
- Canadian
  - Newest
  - Supported
  - Validated
  - criticalcarenutrition.com
Nutrition Game Plan

Why feed?
Who to feed?
What to feed?
When to feed?
Where to feed?
Prime Directive

- Primum non nocere - “First, Do no harm”!
- Live long and prosper.
Why Feed?

- Intuitively logical
- Improve outcomes:
  - Survival, Decreased infections, Complications, Cost
- Therapeutic manipulation
Cumulative Energy Deficit

- Cumulative deficit directly related to infections, LOS, complications, vent days
  - Chiolero Clin Nutrit 24:503:2005

- Reducing deficit improves:
  - Infections (24%) and complications (24%), LOS (16 days).
    - Taylor CCM 27:2525;1999
  - LOS (10 days), Mortality (10%)
    - Martin CMAJ 170:197;2004
Who to Feed

- Lean Tissue - Lost or Threatened
- Time - 5-7 day rule
- Metabolism - Hypo-, Hyper-... Normal
- Supporting data: Intuitive, Teleological, PRCT - equivocal, VA cooperative
- Particular to general reasoning
## Who to Feed
### Nutritional Assessment

<table>
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<th>SUBJECTIVE</th>
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Recent unintended weight loss
- 10#/month

Decreased lean or lipid tissues

Decreased functionality

Albumin levels

Disease Process affecting Intake or digestion
What to Feed?

- Energy - glucose, lipids, protein
- Precursors - protein, lipids, trace elements, phosphorus
- Slimy, yet satisfying - prepared products
- Adapt to patient status
- Obey the Prime Directive!
Substrate Utilization and the Stress Response

- Protein Catabolism
  - Used for synthesis of acute phase response proteins
  - Required for thermogenesis
  - Precursors for tissue repair
  - Immune function
  - Amino acids provided for hepatic gluconeogenesis
Substrate Utilization and the Stress Response

- Protein Catabolism
- Fatty Acids
  - Energy source for cardiac and skeletal muscles
  - Utilization in the liver and other tissues
  - Epinephrine-induced lipolysis
  - Hyperglycemia and resulting elevated insulin levels prevents mobilization from fat stores
Substrate Utilization and the Stress Response

- Protein Catabolism
- Fatty Acids
- Glucose
  - Fuel for central nervous system
  - Required for wound healing
  - Energy for the activation of the immune system
  - Hepatic gluconeogenesis NOT suppressed by glucose administration!
How to Feed

- Goal directed - 25 kcal/kg, 1.5 - 2 gm protein/kg
- Adjust for body and disease
- Avoid overfeeding - more is not better
- Manipulate metabolic metabolic milieu
- Constant critical care
- Do no harm
When to Feed

- Patient and Disease specific - availability of route
- Five day rule
- Metabolic resuscitation
- Supporting data: Many studies (Moore, Daly, Alexander, ...) improved outcome, # infections with early feeding
Enteral v. IVF

- Infections decrease 28%, (p<0.03)
- LOS decreases 1 day (p<0.001)
- Mortality decreases 74% (p<0.06)
  - Lewis BMJ 323:1;2001
  - McClave J PEN 30:143;2006
Early v. Later Enteral

- Decrease infection by 55%, (p<0.0006)
- Decrease LOS 2.2 days, (p<0.0004)
- Decrease mortality by 35%, (p<0.06)
  - Marik CCM 29:2265; 2001
Where to Feed?

- No guts, no glory!
- Run it up the gut!
- A mighty fortress is our gut.
TPN v. Enteral History

- Central Venous Access – Aubaniac 1952
- Dudrick and Wilmore 1968
- Widespread Application- late ‘70s and ‘80s
- Moore – Needle J – late ’80s
- VA Cooperative – early ’90s
- Kudsk – Penetrating Patients - late ’90s
Canadian Clinical Practice Guidelines - 2007

- **Strongly Recommend**
  - Enteral

- **Recommend**
  - <48<sup>0</sup>, Omega 3, Glut in Trauma/Burns, No Arg, Polymeric, SB feeds

- **Consider/Insufficient Data**
  - Everything else
Enteral v. Parenteral

- PRCT 98 pts., ATI >15
- TPN v. jejunostomy, <24 hrs. fed
- Septic morbidity
- Reduced pneumonia, intra-abd, line sepsis in enteral
GUT

- Alterations in blood flow
  - No autoregulation
  - Digestion continues

- Alterations in permeability

- Decreased cell replication/ villi height
  - Lack of enteral feeding
  - Loss of glutamine
Gut Epithelium

- Largest Surface Area
  (300 m²)
- Cellular junction width
- Cellular Proliferation
  or Apoptosis
- Neuroendocrine potential
Angry Bacteria

- Display “Aggressive” posture
- Develop Pilli
- Express antigens on cell wall
Mucosal Immune System

- Largest lymph system
- Peyer’s patches, LN, lamina propria and IE lymphocytes
- Altered # with stress
“Motor of Sepsis”

Intestinal Epithelium

< Barrier

Lymphocyte alterations

Immune System

Angry Bacteria

Commensal Bacteria

Clark and Coopersmith. 
SHOCK 2007
Crosstalk

Barrier

Apoptosis, cell junction

Translocation, mucin, NF - kappa beta

Lymphopenia

Lymphocytes

Bacteria

CYTOKINES, NO, LYMPHOKINES, RECEPTORS
Enteral Nutrition Indications

- Everyone
- Everybody
- Need access and egress

Any medical or surgical diagnosis
Contraindications to Enteral Feedings

- High output Fistula
- Obstruction
- Metabolic needs
- Enteral intolerance
Impact of the Amount of Enteral Formula Delivered to the ICU Patient

- Actual formula delivery
  - Delivery of 14-18 kcal/kg/day or 60-70% of enteral feeding goal associated with shortened LOS and ventilator days and with reduced infectious complications
  - Obese patients receiving < 18 kcal/kg had a shorter ICU LOS and fewer antibiotic days than those receiving greater energy delivery
  - No adequate studies demonstrate an impact on mortality or hospital cost related to enteral formula delivery
Complications of Enteral Nutrition

- Tube related
  - Malposition
- GI related
  - Diarrhea
  - Dietary
  - Demand
Parenteral Nutrition

- Hyperosmolar concentrations of glucose and amino acids... and maybe lipids
- Requires central venous access
TPN

- Allows non-interactive feedings
- Can restore nutrient or energy deficits
- Metabolic and electrolyte resuscitation
TPN Troubles

- Requires critical care
  - NKHHC
  - Electrolytes
  - Hyperglycemia
- Absent nutrients
  - Glutamine
  - Omega 3 oils
- Lipid infusions
  - Omega 6
- Central line problems
- Cost
Indications for TPN

- Fistula
- Failure of enteral diet
- Short gut
Enteral V. Parenteral

- Early Enteral
  - <48 hours
  - Goal
- 5 day rule
- TPN only after 7 days
- Convert to enteral ASAP
38 y/o man – epigastric pain, vomiting
Chronic ETOH, chronic pancreatitis
BP 90’s, BD = -10, Febrile
CT = edema
ED

- CT scan
- Fluids
- Nutrition??
Early Enteral In Pancreatitis

- Improve Outcome
- Ameliorate disease
- Avoid Complications
Reducing Inflammation

- Pancreatic rest
- Resuscitation and avoiding Second hit/MSOF
- Stimulate inhibitors
Pancreas On/Off

On
- Feedings
- Fat
- Gastric

Off
- Peptides
  - GLP – 1
  - PYY
- Low Fat Feeds
- PYY demonstrated to reduce histo changes, IL – 6 levels, prevent death

Therapy

- Aggressive resuscitation
- <48 hours feeding
- Low in fat
- Reduction in ICU/MSOF
- 7 day Mortality
  - MSOF = 50%
  - No MSOF = 0%

McClave J PEN 2006 30:143
Johnson et al. Gut 2005
Case Two

- 75 y/o woman
  - Sigmoid colectomy for cancer
  - PMH – DM, HTN
- POD #5
  - Fever, >BP, peritonitis
  - Anastomotic dehiscence at OR
- ICU
  - P::F<250, BD = -10, u/o marginal
ICU

- When to Feed?
- Where to Feed?
- What to Feed?
Manipulate Metabolism

- Maintain Euglycemia
- Omega 3 fatty acids
  - Alter PG and EC
- Glutamine
  - Gut fuel
- Probiotics
  - Restore normal flora
Risk factors for stress hyperglycemia in critical illness

- Pre-existing diabetes mellitus
  - Insulin Deficiency
- Infusion of pressors
  - Insulin resistance
- Obesity
  - Insulin resistance
- Aging
  - Insulin deficiency
- Sepsis
  - Insulin deficiency & resistance
- Glucocorticoid therapy
  - Insulin resistance
- Bed rest
  - Reduced skeletal muscle insulin sensitivity
Observational Studies

- **Morbidity**
  - Admit FBG >126/2 random BG > 200 associated with 29% more ICU admits in non-DM patients. *Umpierrez 2002*
  - Admit BG > 8.0-10 mmol in non-DM with MI associated with risk of CHF or cardiogenic shock. *Capes 2000*
  - Single BG > 220 in DM POD #1 had serious infection rates 5.7 x higher than DM post op with BG < 220. *Pomposelli 1998*
Observational Studies

- **Mortality (cont’d)**
  - Mean BG values in ICU med/surg patients were higher in non-survivors (163 mg/dl) than survivors (124 mg/dl). Lowest mortality occurred among patients with mean BG 80-99 mg/dl. *Krinsley 2003*
Intervention Trial

- **Van den Berghe, 2001**
  - Prospective, randomized controlled trial
  - Subjects: surgical ventilated ICU patients, n=1548.
  - Treatment groups
    - Group 1: Conventional treatment - IV insulin infusion only if BG >215, then maintenance of BG 180-200.
    - Group 2: IIT - IV insulin infusion to maintain BG 80-110.
Bacteremia

Prolonged Abx (>10d)

Dialysis/CVVH

Critical illness polyneuropathy

Prolonged ventilation (>14 d)

Prolonged ICU stay (>14d)

Relative Risk Reduction (%)

Van den Berghe, 2001
Mortality in ICU patients receiving IIT vs conventional therapy

Van den Berghe, 2001
Recommendations

- In the critically ill patient with diabetes:
  - Scheduled insulin will likely be required while receiving nutrition support if previously on oral agent or insulin prior to admission (OK to use SQ intermediates)
  - Aggressive Sliding Scale to accompany SQ scheduled insulin
  - If unable to attain BG below 180 mg/dL (in ICU’s) with SQ scheduled insulin regimen, consider the use of intravenous infusion
OR

- 24 y/o
  +9 mm
  +RUQ >>L Flank
  How many tubes?
OR

- J-tube ?
- G-tube ?
- Formula ?
- Route ?
Pre-Op/Post Op

- Post pyloric feeding
  - Reduce NPO Time
- Open Abdomen Feedings
  - Speed
    - Closure/Improve outcome
- Trophic Effects
  - Fistula closure
Questions ?
Review

- Nutrition Support
- Provide specific needs
- Provide therapeutic care
- Enteral >> Parenteral
- Prevent Complications
- Use available Evidence Based Data
Thanks