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 nuture or grow
- Support = to bolster or strengthen

Definition

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

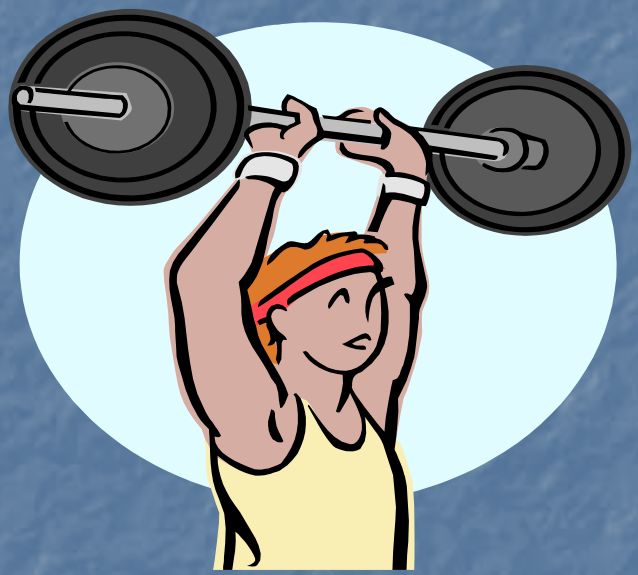
Players

- 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 practioners 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

- Primium 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

SUBJECTIVE	*OBJECTIVE*	*AUTOMATIC*
Appearance	Labs	Time
Disease	‘Metrics	Disease
SGA	Function	Technical
	Formulae	

Nutritional Assessment

- 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 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 JPEN 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
  - Heyland JPEN 27:355;2003, 2007

Where to Feed?

- No guts, no glory !
- Run it up the gut!
- **A mighty fortress is our gut.**

TPN v. Enteral History

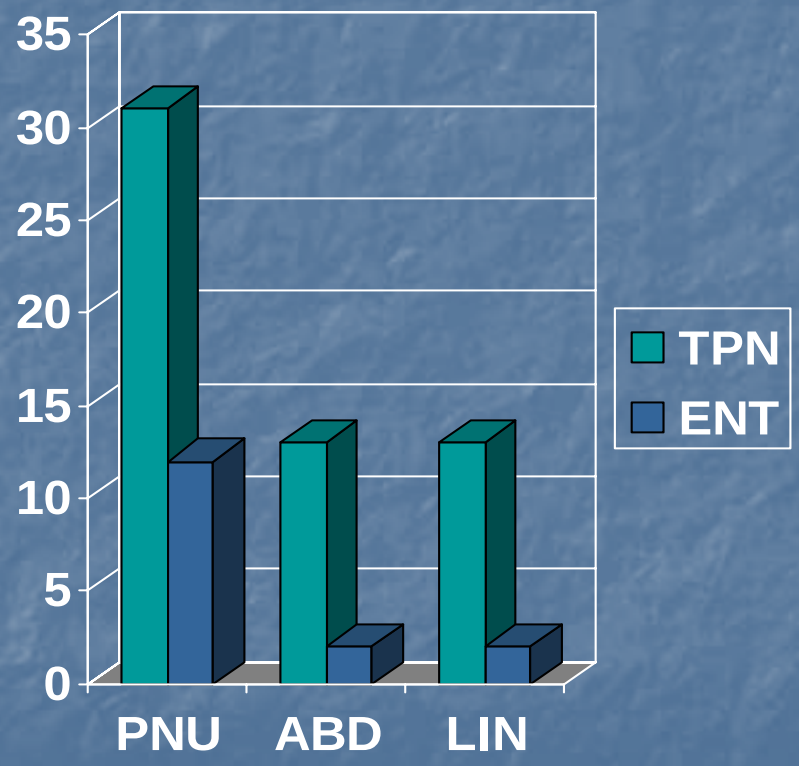
- Central Venous Access – Aubaniac 1952
- Dudrick and Wilmore 1968
- Widespread Application- late ‘70s and ’80s
- Moore – Needle J – late ’80s
- VA Cooperative – early ’90s
- Kudsk – Penetrating Patients - late ’90s

Canadian Clinical Practice Guidelines - 2007

- Strongly Recommend
  - Enteral
- Recommend
  - $<48^0$, Omega 3, Glut in Trauma/Burns, No Arg, Polymeric, SB feeds
- Consider/Insufficient Data
  - Everything else

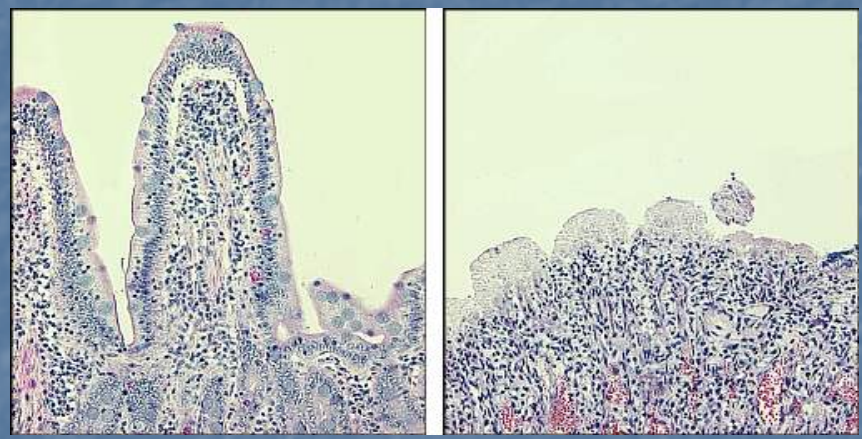
Enteral v. Parenteral

- Kudsk et al. *Ann Surg 1992*
- PRCT 98 pts.,ATI >15
- TPN v. jejunostomy, <24 hrs. fed
- Septic morbidity
- Reduced pneumonia, intra-abd, line sepsis in enteral



GUT

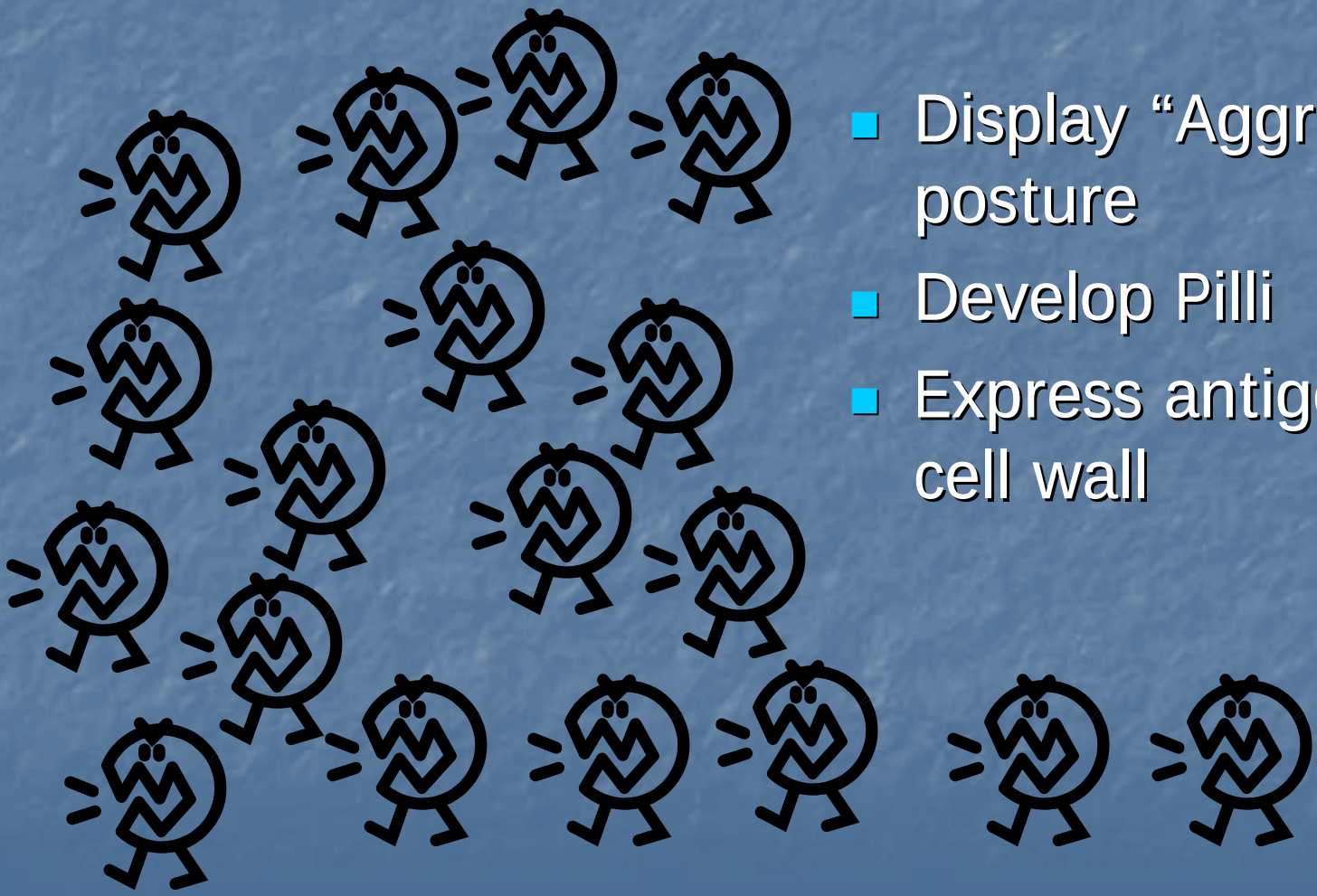
- Alterations in blood flow
  - No autoregulation
  - Digestion continues
- Alterations in permeability
- Decreased cell replication/ villi height
  - Lack of enteral feeding
  - Loss of glutamine



Gut Epithelium

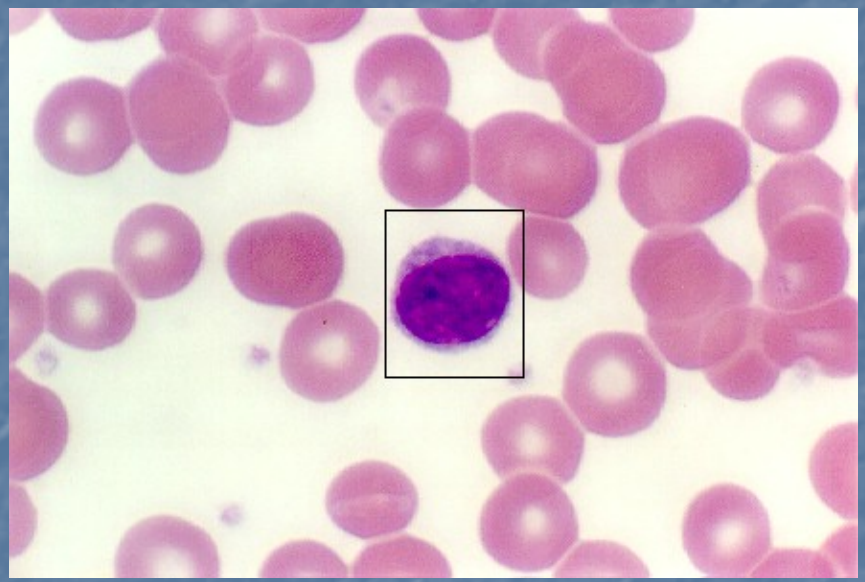
- Largest Surface Area (300 m^2)
- Cellular junction width
- Cellular Proliferation or Apoptosis
- Neuroendocrine potential

Angry Bacteria



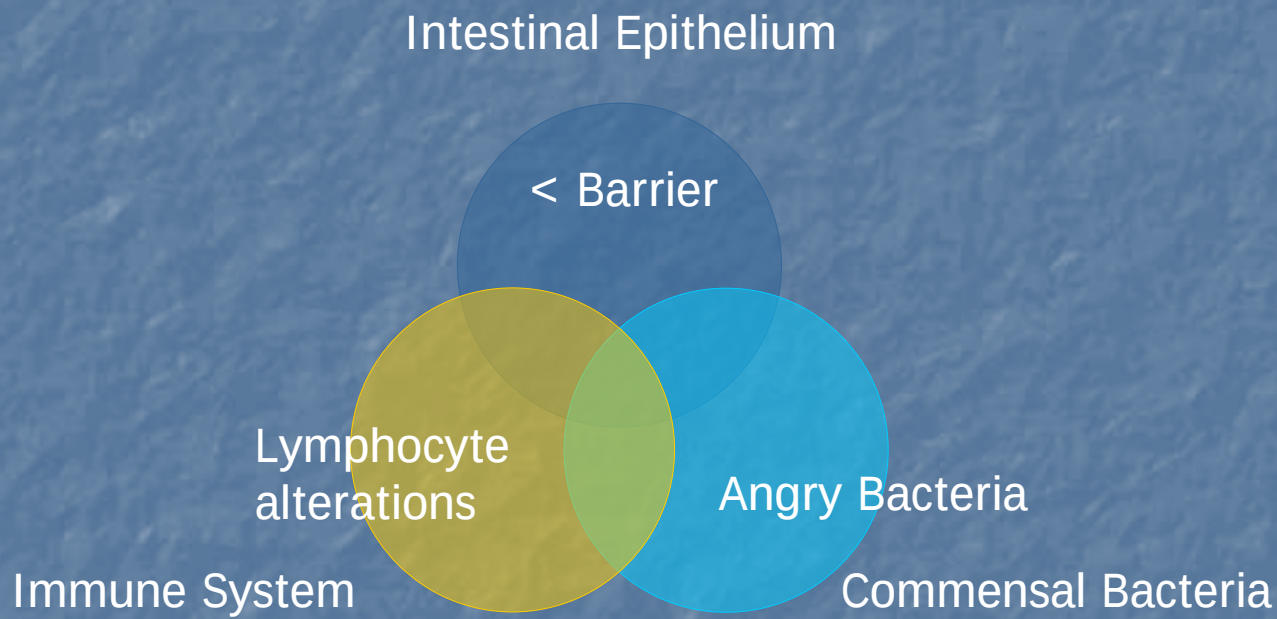
- Display “Aggressive” posture
- Develop Pilli
- Express antigens on cell wall

Mucosal Immune System



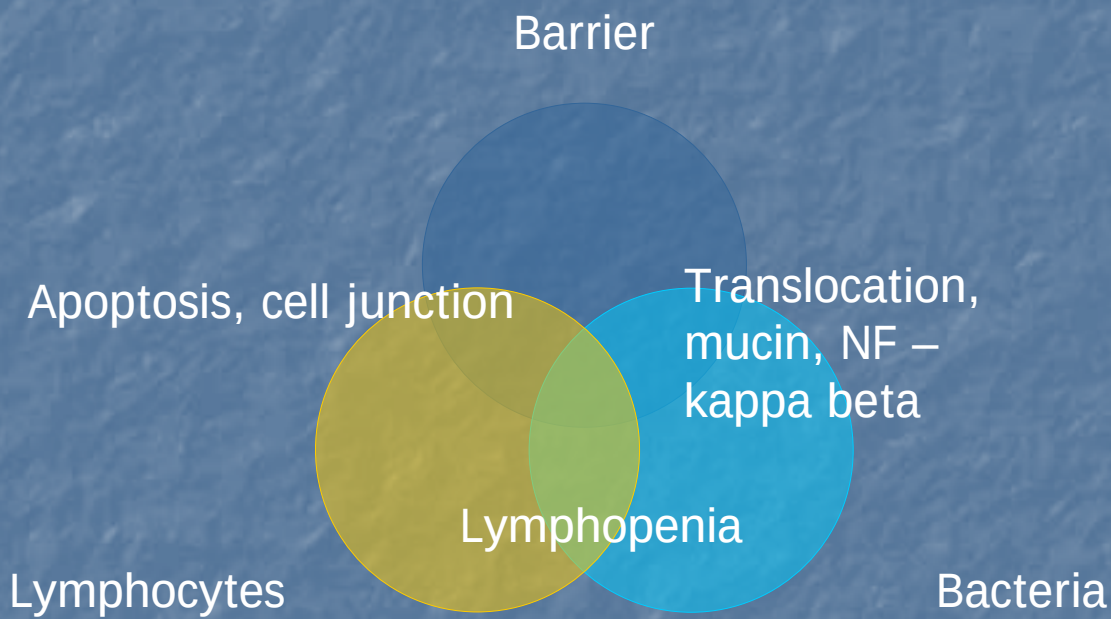
- Largest lymph system
- Peyer's patches, LN, lamina propria and IE lymphocytes
- Altered # with stress

“Motor of Sepsis”



Clark and Coopersmith.
SHOCK 2007

Crosstalk



CYTOKINES, NO, LYMPHOKINES, RECEPTORS

Enteral Nutrition Indications

- Everyone
- Everybody
- Need access and egress

Any medical or surgical diagnosis

Contraindications to Enteral Feedings

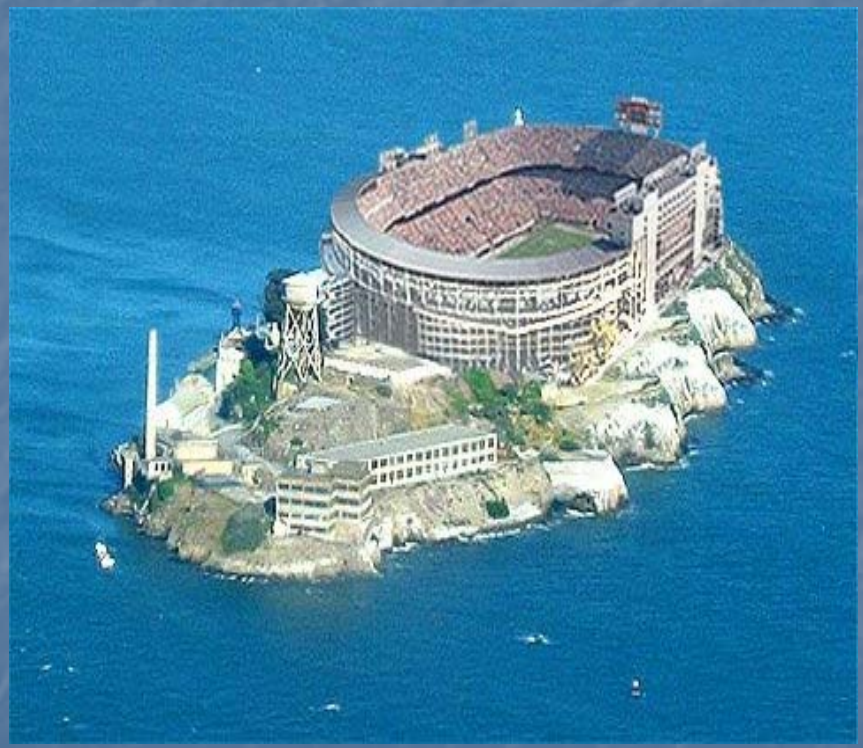
- High output Fistula
- Obstruction
- Metabolic needs
- Enteral intolerance

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

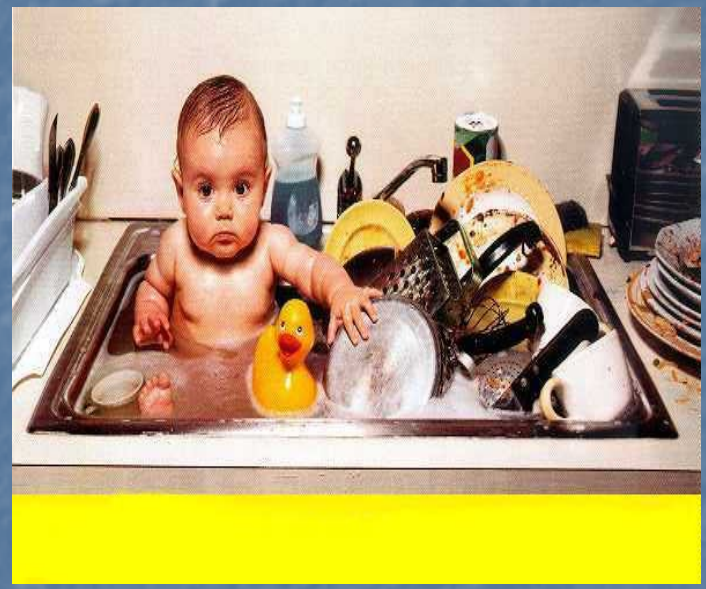
- Allows non-interactive feedings
- Can restore nutrient or energy deficits
- Metabolic and electrolyte resuscitation

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



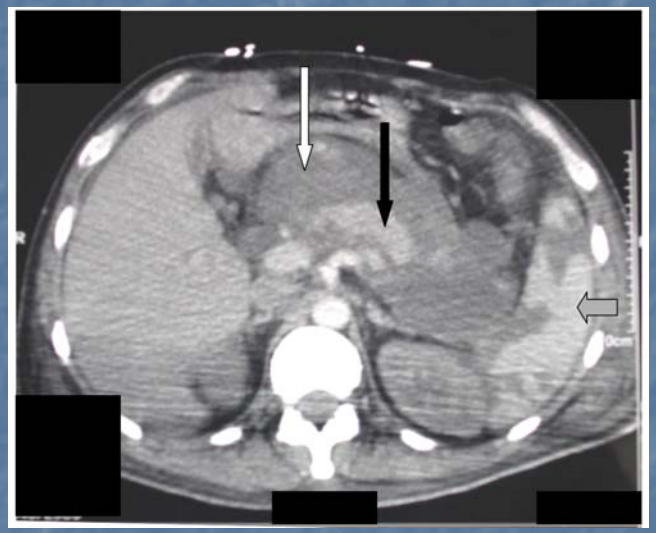
Enteral V. Parenteral

- Early Enteral
  - <48 hours
  - Goal
- 5 day rule
- TPN only after 7 days
- Convert to enteral ASAP

ED

- 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

Grise et al. Pancreas 2002 24:90.

Therapy

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

McClave JPEN 2006 30:143

Johnson et al. Gut 2005

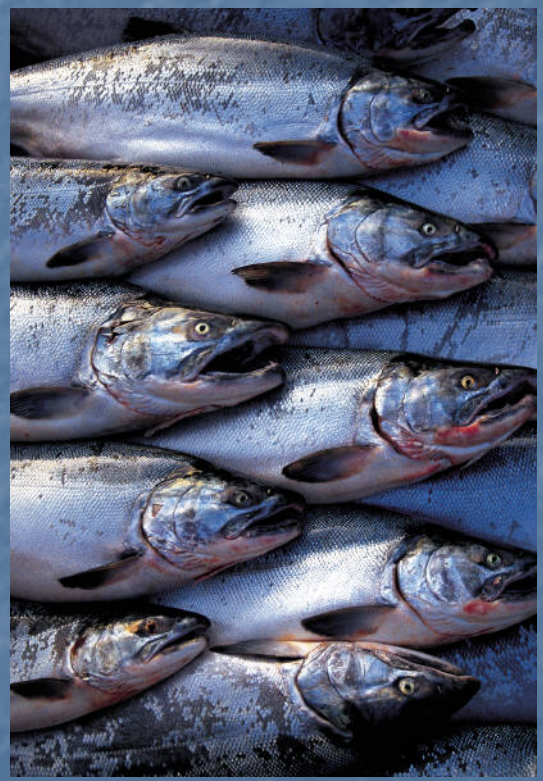
Case Two

- 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

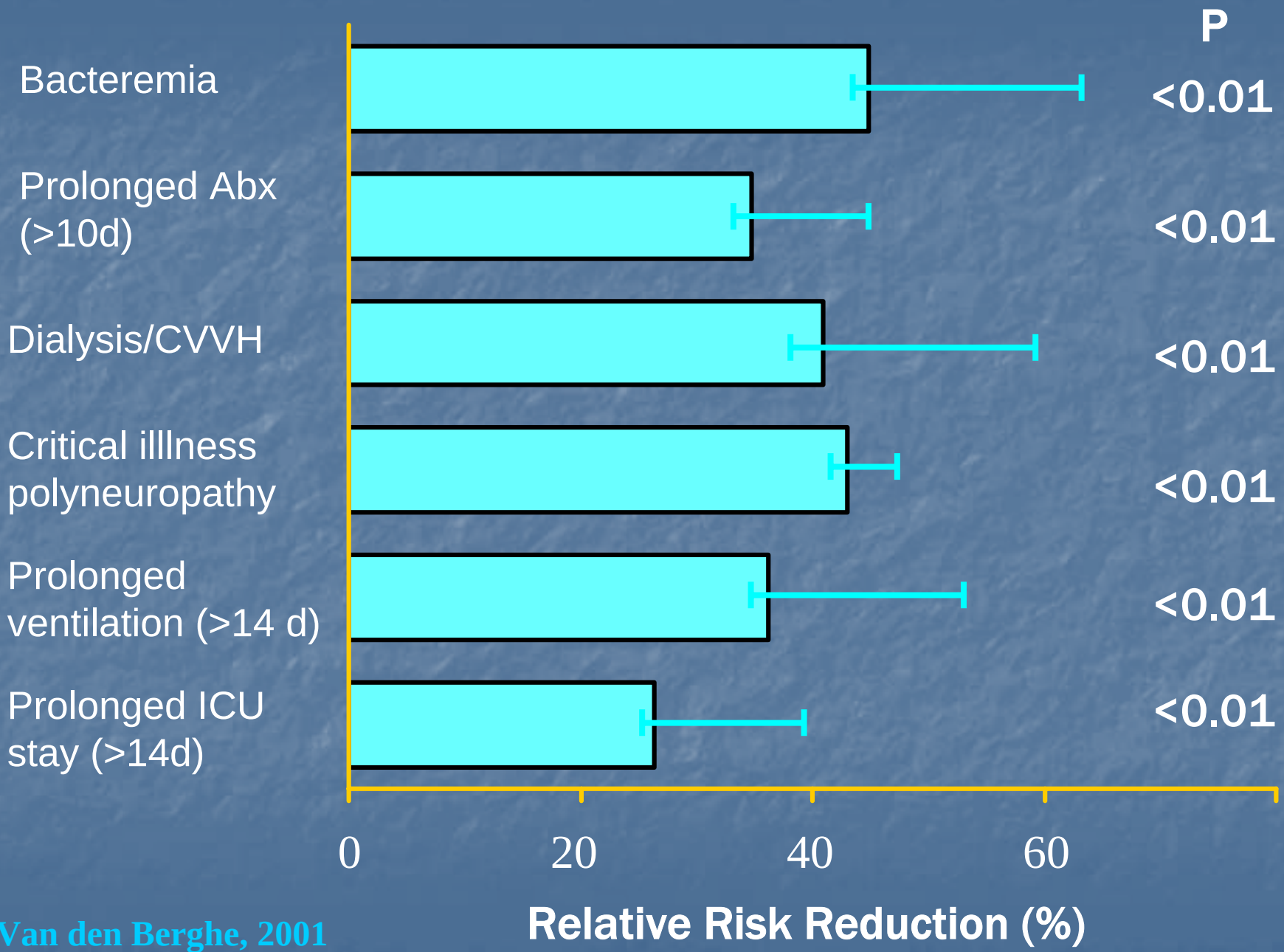
- Morbidity
  - Admit FBG >126/2 random BG > 200 associated with 29% more ICU admits in non-DM patients. Umpierrez 2002
  - Admit BG > 8.0-10 mmol in non-DM with MI associated with risk of CHF or cardiogenic shock. Capes 2000
  - Single BG > 220 in DM POD #1 had serious infection rates 5.7 x higher than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

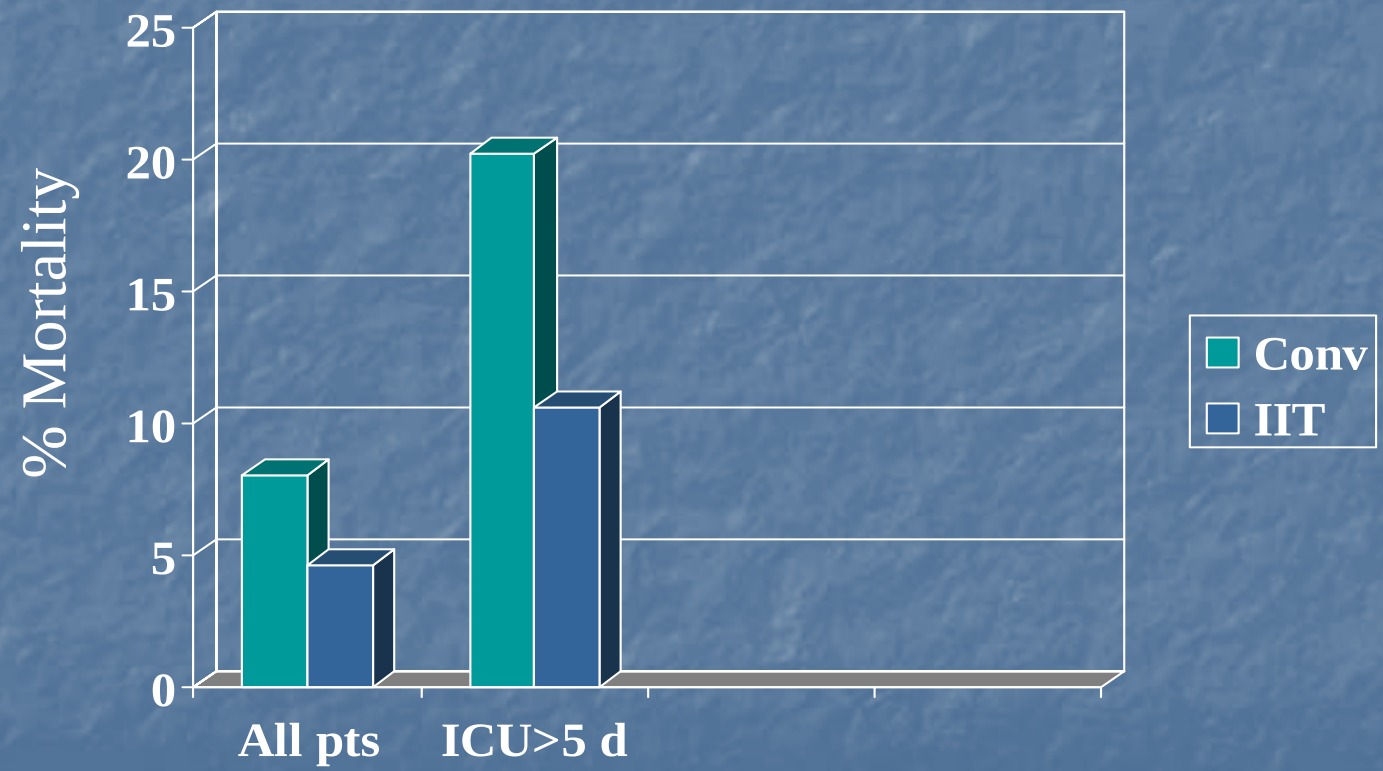
Intervention Trial

- ***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.



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?

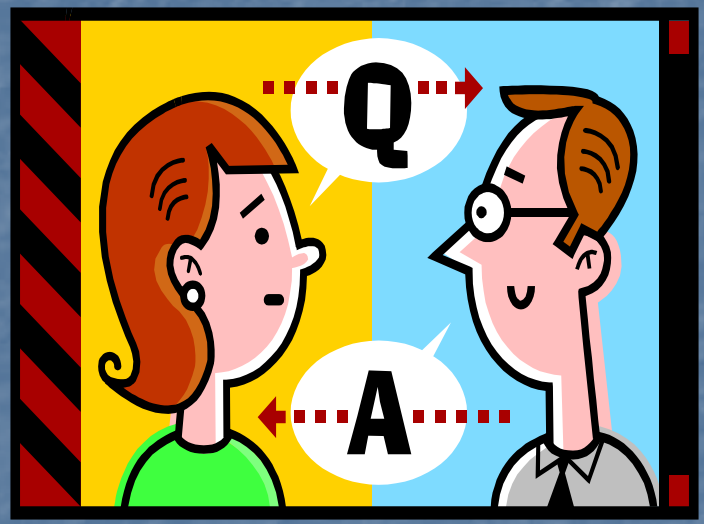
OR

- 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