**Metabolic Stress**

- Cellular response to stress
  - Inappropriate accumulation of substances within the cell
  - Changes in size, number, or shape
  - Inflammatory response
- Highest level of nutritional risk
  - PEM
Starvation

- Malnutrition – body is unable to utilize nutrients or needs are so high that current intake cannot meet demands
- Body responds through physiological adaptations
  - lower BMR - Energy needs decrease
  - Lipolysis occurs – ketone production
- See Table 25.1 and Fig. 25.1
Metabolism Response to Starvation (Short Term)
No Injury or “Stress” (Protective Adaptation Occurs)

- Overall energy needs decrease
- Metabolic rate decreases 20-25 kcal/kg/d
- Energy from fat storage >90% of kcal
- Energy from protein <10% for gluconeogenesis
- Protein store protected

ENERGY DEPOT
FAT, FATTY ACID

90% kcal
Ketones

ENERGY PRODUCTION

Energy for protein synthesis
To tissues

LEAN MASS
Minimal catabolism to meet glucose needs
EROSION MINIMAL

LIVER

Gluconeogenesis (10% kcal)
Glucose
For obligate users (brain)

Oxygen

Micronutrients needed
Amino Acids

Alanine
Protein synthesis

Hormone adaptation preserves protein

Intact skin
Heat loss blocked

Glucose
Pyruvate
Urea

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Response to Stress

- Hypermetabolic, catabolic response to acute injury or disease
- Degree of metabolic stress correlates with seriousness of injury
- Energy needs dramatically increase, normal adaptations do not occur
Response to Stress

• Etiology –
  ✓ Hormone release, acute-phase protein synthesis, hypermetabolism, increased reliance on gluconeogenesis, shift in fluid balance and decreased urine output
Response to Stress

- Clinical manifestations
  - Ebb phase
    - 2-48 hours
    - Shock resulting in hypovolemia
    - Decreased oxygen available to tissues
    - Decreased cardiac and urinary output
  - Flow phase
    - Hypermetabolism, catabolism
    - Altered immune and hormonal response
  - Recovery phase
    - Return to anabolism and normal metabolic rate
Response to Stress

- Pathophysiology
  ✓ “Fight or flight” response
    - Glucagon - gluconeogenesis
    - Cortisol - gluconeogenesis and fatty acid mobilization
    - Epinephrine, norepinephrine - glycogenolysis
    - Mobilization of nutrients to meet immediate demands
    - May result in hyperglycemia
Response to Stress

- Pathophysiology
  - Hyperglycemia and insulin resistance may be controlled by *insulin therapy*
  - Also may reduce catabolism and inflammation
Response to Stress

• Pathophysiology
  ✓ Gluconeogenesis
    • Reliance on protein as source of glucose
    • Alanine and glutamine in particular
    • Increased catabolism of skeletal muscle
    • Negative nitrogen balance
    • Complications: immunosuppression, increased infection rates, delayed wound healing, increased mortality
Response to Stress

• Pathophysiology
  ✓ Acute-phase proteins - markers of stress response
    • Fibronectin
    • C-reactive protein (CRP)
    • Ceruloplasmin
    • Serum amyloid A
    • Regulated by cytokines; interleukins, leukotrienes, interferon, tumor necrosis factor – see Table 25.3
Catabolic Insult-Induced Protein-Energy Malnutrition
(Protein and Energy Production Abnormal)

- No adaptive responses activated
- Increase metabolism rate 35-40 kcal/kg/d
- Increase glucose production in excess of need
- Increase use of protein for fuel (glucose)

Prior to nutrition support

LIVER

Gluconeogenesis
Amino acids
Oxygen

To tissues
Glucose
Micronutrients

To liver
Alanine

Lean Mass
Rapid erosion (catabolism) of muscle protein. Visceral protein for glucose production

Energy Depot
Fat
Fatty acid
36C
Body heat
30% used
70% recycled

Energy Production
Mainly from glucose and amino acids

To tissues
To wound
Glutamine

Glutamine depletion
Antioxidant depletion
Micronutrient depletion

Net protein loss

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Response to Stress - Nutrition Therapy

- Balance between prevention of PEM and complications of nutrition support
- Consider status prior to illness, level of injury, current metabolic changes
Response to Stress - Nutrition Therapy

- Assessment
  - Many standard measures not valid or reliable
  - Family members important source of information
  - Measured weight and visceral protein status may be affected by fluid balance
  - Indirect calorimetry most accurate for estimating energy requirements
Response to Stress - Nutrition Therapy

• Assessment
  ✓ Energy estimates – equations
    • Mifflin-St. Jeor or Harris-Benedict
    • Use stress and injury factors – see Table 25.4
  • Initial caloric goals: 25-35 kcal/kg

✓ Protein
  • 1.2-1.5 g protein/kg
✓ “Permissive underfeeding”
  • 14 kcal/kg, 1.2 g protein/kg
Response to Stress - Nutrition Therapy

• Interventions
  ✓ Oral preferred route
  ✓ Early initiation of nutrition support with specific dg - see Fig. 25.4
  ✓ First consider enteral – see Fig. 25.5
  ✓ Specialty formulas available – see Table 25.5
Response to Stress - Nutrition Therapy

• Interventions
  ✓ Supplemental nutrients to consider:
    • Arginine, glutamine
    • Branched-chain amino acids: isoleucine, leucine, valine
    • Omega-3 fatty acids
    • Modify type of lipid; menhaden oil, marine oil, structured lipids
    • Sources of fiber
    • Probiotics, prebiotics, synbiotics
Response to Stress - Nutrition Therapy

- Interventions
  - Complications of enteral include
    - Hyperglycemia
    - Electrolyte imbalances
    - Aspiration
    - Mechanical complications
Response to Stress - Nutrition Therapy

• Interventions
  ✓ Total parenteral nutrition (TPN)
    • Reserved for NPO status, if enteral access not viable or unable to meet needs (volume)
    • Hyperglycemia most critical concern
    • Other concerns: catheter occlusion, infection, hypertriglyceridemia, intestinal atrophy, electrolyte disturbances, refeeding syndrome
Burns

- Tissue injury caused by exposure to heat, chemicals, radiation, or electricity
- Depth of wound and body surface are used to classify
  - Superficial
  - Superficial partial thickness
  - Deep partial thickness
  - Full thickness
  - See Table 25.6
Burns

- Rule of “Nines” used to estimate BSA
  - See Fig. 25.6
  - Used in assessment of extent of injury, basis for fluid and medication recommendations
Burns

- Pathophysiology
  - Excessive inflammatory process
  - Rapid fluid shifts and accumulation
  - Fluid loss from wound
  - Metabolic stress; hypermetabolism, catabolism, immune, hormonal response
  - Respiratory complications
Burns

• Treatment
  ✓ Topical agents
  ✓ Clean, debride, dress wounds
  ✓ Skin grafting
Burns

• Nutrition Therapy/ Implications
  ✔ 20% body protein can be lost
  ✔ Fluid imbalance, pain, immobility
  ✔ Wound healing requires optimum nutrition
  ✔ Weight fluctuations (d/t fluid shifts and resuscitation)
Burns

• **Nutrition Therapy/ Assessment**
  - Estimate energy using indirect calorimetry
  - Curreri equation can be used at peak of burn injury
    - Needs do not increase beyond 50-60% total body surface area burn
  - Mifflin-St. Jeor equation with injury factor 1.3-1.5
  - Energy needs increase with fever, infection, sepsis
Burns

- Nutrition Therapy/ Assessment
  - Protein 1.5-2 g protein/kg
  - Negative nitrogen balance may not be totally prevented
  - Set goal to minimize losses and promote wound healing
**Burns**

- **Nutrition Therapy/ Interventions**
  - Nutrition support - enteral
    - Early feeding associated with prevention of infections and Curling’s ulcer, and reduction in protein catabolism
    - Focus on higher protein (20-25% of kcal)
    - Supplemental arginine, glutamine, omega-3 fatty acids
  - PN if enteral cannot meet needs
Burns

- Nutrition Therapy/ Interventions
  - Nutrition support - PN
    - Avoid overfeeding, control hyperglycemia
  - Oxandrolone (anabolic steroid)
    - Used to promote protein synthesis
  - Additional vitamins, minerals, trace elements
    - Vitamins C, A, E, zinc routinely used
  - Wean from nutrition support when pt. can meet at least 60% of needs orally
Surgery

- Nutritional implications if...
  - Patient enters surgery malnourished or overnourished
  - Surgery will interrupt normal nutrition processes
  - Preoperative changes in weight, albumin, CRP
Surgery

- Clinical manifestations... depend on type of procedure
  - 12 hours pre-op NPO
  - May have nasogastric tube
  - Anesthesia may result in postop. ileus (lack of motility), general paralysis of GI tract
  - PO resumed with bowel sounds and gas production
Surgery

- Nutrition Implications/ Interventions
  - Post-operative metabolic stress
  - Progression for postoperative feeding individualized
    - NPO to solid foods as quickly as possible
  - Individualize energy and protein using REE and activity and injury factors
  - Nutrition support if NPO prolonged
Sepsis, SIRS, MODS

- **Sepsis** – immunosuppressive response preventing adequate response to infection
- **Systemic Inflammatory Response System (SIRS)** – sepsis without infection
- **Multi-Organ Distress Syndrome (MODS)** – complication of sepsis and SIRS; altered function in 2 or more organs
Sepsis, SIRS, MODS

- Etiology/Pathophysiology
  - Originating infection/trauma
  - Complex cascade of proinflammatory cytokine release, altered cellular metabolism, hypoperfusion, hypotension
  - Release of inflammatory mediators
  - Sepsis continues; shift to anti-inflammatory response with anergy
  - Increased rate of gluconeogenesis and catabolism
Sepsis, SIRS, MODS

- Clinical manifestations
  - Increased WBC, heart rate, respiration
  - Fever or hypothermia
  - Elevated CRP, fibrinogen, complement proteins, other acute-phase proteins
  - Increased serum lactate and serum glucose
Sepsis, SIRS, MODS

• Nutrition Implications
  ✓ Nutrition support to meet abnormalities in metabolism, nutritional requirements, and fluid/volume restrictions