Surgery Critical Care

PSGS Review
Bonaventure Plaza, Greenhills, San Juan
1-2 PM; April 26, 2013
Objectives

• To discuss key events and situations that have a major role in the outcome of critical care patient management
• To discuss areas where targeted goal management plays a major role in achieving positive outcomes in critical care
Discussion Points

• Conditions resulting to critical care status
• Inflammation state of ICU patient
• Optimizing nutrition therapy through:
  – Early enteral nutrition
  – Pharmaconutrients
  – Insulin resistance
  – Adequate intake
  – Role of the Nutrition Team
Surgical critical care

- Critical illness following surgery or trauma
- High risk surgical patients
- Shock and hemodynamic compromise
- Acute lung injury and ARDS following surgery, trauma, or pancreatitis
- Sepsis and severe infections
- Trauma evaluation and management
- Neurologic emergencies

- Post-transplantation
- Post-operative complications
- Peritonitis, perforated viscus, and abdominal sepsis
- Enterocutaneous fistulas
- Gastrointestinal hemorrhage
- Severe acute pancreatitis
- Multisystem organ failure
INFLAMMATION IN THE CRITICAL CARE STATE

- Shock and hemodynamic compromise
- Critical illness following surgery or trauma
- Acute lung injury following surgery or trauma
  - ARDS, complication of acute pancreatitis
- Sepsis and severe infections
- Peritonitis, perforated viscus, and abdominal sepsis
- Enterocutaneous fistulas
SHOCK AND HEMODYNAMIC COMPROMISE
Body compartment percentage of total body fluids:

a. 70%
b. 60%
c. 50%
d. 40%
Body composition, all ages

Body Composition at Various Ages

- Small premature infant (1 kg)
- Large premature infant (2 kg)
- Term infant (3.5 kg)
- 1 yr old (10.5 kg)
- Adult (70 kg)

Categories:
- Carbo
- Fat
- Prot
- Water
Body compartments in health and disease

NORMAL
- WATER (60%)
- PROTEIN (14%)
- FAT (25%)

OBESE
- WATER (55%)
- PROTEIN (14%)
- FAT (30%)

STARVATION
- WATER (72%)
- PROTEIN (12%)
- FAT (15%)

CRITICAL CARE
- WATER (70%)
- PROTEIN (6%)
- FAT (23%)

CARBO + OTHER (1%)
• Best solution for volume loss repletion:
  a. Isotonic saline
  b. Balanced electrolyte solution
  c. Colloid
  d. D5LR
Volume and electrolyte changes

- **Electrolytes = normal**
  - **Albumin = normal**

- **Electrolytes = normal**
  - **Albumin = low**

- **Hypernatremia**
  - **Albumin = normal**

- **Hyponatremia**
  - **Albumin = low**

- **ECF = loss**
  - **Intravascular loss**
  - **Interstitial = normal**

- **ECF = loss**
  - **Intravascular loss**
  - **Interstitial = swollen**

- **ECF = loss/none**
  - **Intravascular loss**
  - **Cell shrink**

- **ECF = loss/none**
  - **Intravascular loss**
  - **Cell swell**

- **Balanced electrolyte solutions**
  - **Colloid**

- **D5W**
  - **Colloid**

- **Hypertonic saline (3%SS)**
  - **Colloid**

- **Avoid D5W**
  - **Avoid 0.3% SS**

- **Cerebral edema**
Crystalloids

How to restore a depleted ECF volume to normal?

- NaCl → hyperchloraemic acidosis
- RL → hypotonicity, lactate
- BEL → compromise anion balance

∑ BEL: - deviation from normal↓
- potential of side effects↓
- intrinsic safety↑

⇒ trend: balanced electrolyte solutions (BEL)!
Pro’s and con’s of albumin infusion

- Maintenance or elevation of plasma colloid osmotic pressure
- Antioxidant action
- Transport function
- Anti-inflammatory and antiapoptotic actions
- No coagulation inhibiting effect

- Overhydration
- Viral transmission
- Anaphylaxis
- Costs
Hydroxyethyl Starch II

- Slowly degradable: MS > 0.5
- Rapidly degradable: MS < 0.5

450/0.7

70/0.5
- 200-260/0.5
- 200/0.62

130/0.4
- 130/0.42

balanced
- Mw > 500 kD, MS > 0.7
- (Hextend®)

4th generation?

balanced
- 130/0.4
- 130/0.42

- 1st generation HES: Hetastarch
- 2nd generation HES: Hexa-, Pentastarch
- 3rd generation HES: Tetrastarch
- Improved 3rd generation

Generation↑ ⇒ side effects↓
Crystalloids vs. HES vs. GEL

Plasma replacement: Colloids are more effective

Hydroxyethyl Starch and Hemostasis

- FVIII/vWF↓
- Fibrinpolymerisation↓
- Platelet aggregation↓
- Endothelial adhesion↓

⇒ HES interferes with fibrinogen, other coagulation factors and platelets more than from dilution alone

Kozek- Langenecker SA. Anesthesiology 2005; 103: 654
Colloids - Volume Effects

6% HES 130:
COP↑
volume effect 120%
half life 7h

4% GEL 30:
COP=
Volume effect 100%
half life 5h

*Oncometer: BMT 921, Membrane 20.000 D
## Resuscitation: fluids

<table>
<thead>
<tr>
<th>Use</th>
<th>Compartment</th>
<th>Composition</th>
<th>Examples</th>
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</thead>
<tbody>
<tr>
<td>Volume Replacement</td>
<td>Intravascular fluid volume</td>
<td>Iso-oncotic</td>
<td>6% HES 130 in balanced solution</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Isotonic Iso-ionic</td>
<td></td>
</tr>
<tr>
<td>Fluid Replacement</td>
<td>Extracellular fluid volume</td>
<td>Isotonic Iso-ionic</td>
<td>Balanced solution: normal saline; ringer’s lactate</td>
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<tr>
<td>Electrolyte or osmotherapy</td>
<td>Total body fluid volume</td>
<td>According to need for correction</td>
<td>KCL Glucose 5% Mannitol</td>
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<tr>
<td>(solutions for correction)</td>
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Reference: Zander R, Adams Ha, Boldt J. 2005; 40; 701-719
Compute fluids of a 70 kg person

- TBF=70kg x 60% = 42L (total body fluid)
- ECF=70kg x 20% = 14L (extracellular fluid)
- ICF=70kg x 40% = 28L (intracellular fluid)
- Total plasma volume = 70kg x 5% = 3.5L
- Total blood volume (hct=38) = 5.6L
- Total interstitial fluid = 14L – 5.6L = 8.4L
How much is the total blood volume?

- How to compute:

  \[
  \text{Total blood volume} = \text{total plasma volume} \times \frac{100}{100 - \text{hematocrit}}
  \]

  - Plasma volume is 5% of actual body weight
  - Weight=70 kg; hematocrit = 38
  - Total plasma volume = 5% \times 70\text{kg} = 3500 \text{ ml}
  - Total blood volume = 3500\text{ml} \times (100/[100-38])
  - TBV = 3500 \text{ ml} \times (100/62) = 3500\text{ml} \times 1.61
  - Total blood volume = 5645 \text{ ml or 5.6 liters}
Body composition and water

Human body composition (% of weight):

- **Water**: 60%
  - ECF (extracellular fluid): 20%
    - Intravascular fluid
    - Extravascular interstitial fluid
  - ICF (intracellular fluid): 40%
- **Mass**: 40%
  - Lean body mass
  - Fat mass

TBF = ICF + ECF = 42 liters (60% of weight)

- **ECF**: 14 liters
  - Plasma
  - Interstitial Fluid
- **ICF**: 28 liters

- **Computation of usual fluid requirement per day**:
  - 30 ml/kg
  - or 1.5 to 2.5 L/day
### Normal levels of electrolytes

<table>
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<tr>
<th>Electrolyte</th>
<th>Normal Range</th>
<th>Unit</th>
<th>Location</th>
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<tbody>
<tr>
<td>Sodium</td>
<td>135-145</td>
<td>mEq/L</td>
<td>serum</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.5-5.5</td>
<td>mEq/L</td>
<td>serum</td>
</tr>
<tr>
<td>Calcium</td>
<td>8.8-10.4</td>
<td>mg/dL</td>
<td>serum</td>
</tr>
<tr>
<td>Calcium unbound</td>
<td>4.7-5.2</td>
<td>mg/dL</td>
<td>serum</td>
</tr>
<tr>
<td>Magnesium</td>
<td>1.4-2.1</td>
<td>mEq/L</td>
<td>plasma</td>
</tr>
<tr>
<td>Chloride</td>
<td>100-108</td>
<td>mEq/L</td>
<td>serum</td>
</tr>
<tr>
<td>Phosphate</td>
<td>2.5-4.5</td>
<td>mg/dL</td>
<td>plasma</td>
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Osmolality

- Normal cellular function requires normal serum osmolality
- Water homeostasis maintains serum osmolality
- The contributing factors to serum osmolality are: Na, glucose, and BUN
- Sodium is the major contributor (accounts for 90% of extracellular osmolality)
- Acute changes in serum osmolality will cause rapid changes in cell volume
How to compute for plasma osmolality

Osmolality = 2 x [Na] + [glucose]/18 + [BUN]/2.8

Na = 140 mmol/L
Glucose = 110 mg/dL
BUN = 20 mg/dL

Osmolality = (2x140) + (110/18) + (20/2.8)

Osmolality = 280 + 6.1 + 7.1

Osmolality = 293.2 mmol/L

Division of glucose and BUN by 18 and 2.8 converts these to mmol/L
Regulation of sodium and water balance

Aldosterone → ADRENAL GLAND
Sodium reabsorbed

HEART → ANH
Sodium excreted

ADH → KIDNEY
Water reabsorbed
• Antidiuretic hormone
  a. Leads to water retention
  b. Synthesized by the kidney
  c. Leads to water loss through the urine
  d. Stimulated by low plasma sodium level
Antidiuretic hormone (Vasopressin)

- Synthesized in hypothalamus
- Transported to the neural lobe/posterior pituitary
- Stored as secretory granules within the nerve terminals of neurohypophysis
- Depolarization of nerve terminal releases vasopressin into the circulation
- Hypertonicity/decreased ECF volume-arterial blood pressure stimulate secretion
- Vasopressin leads to water retention by the kidney
The post-resuscitation environment

1. Cardiopulmonary arrest
2. Cardio or pulmonary failure
3. Trauma/Injury

Shock/hypovolemia → ↓oxygenation

Microcirculation changes/effects

Cellular dysfunction, ↑free radicals, ↑eicosanoids, Acid-base imbalance
The post-resuscitation environment

1. Cardiopulmonary arrest
2. Cardio or pulmonary failure
3. Trauma/Injury

Shock/hypovolemia \( \rightarrow \) \( \downarrow \) oxygenation

Microcirculation changes/effects

Cellular dysfunction
\( \uparrow \) free radicals
\( \uparrow \) eicosanoids
Acid-base imbalance

\( \uparrow \) INFLAMMATION
The inflammation environment

STRESS INJURY

Monos, macros, lymphos, epithelia in inflammatory state

Adipose tissue

TNF, IL1, IL6

Liver

Platelets

Endothelium

Acute phase response

↑ fibrinogen

↓ HDL

↑ Aggregability

↑ Adhesion molecules

MICROCIRCULATION ENVIRONMENT

↑ LPL

LPL = lipoprotein lipase; HDL = high-density lipoprotein.
Organ status post-resuscitation

Cardiac status
• electrolyte status
• neural activity
• muscle activity

Lung status
• gas exchange
• mucosal immunity

GUT ISCHEMIA
1. ↑ free radicals
2. ↑ eicosanoids
3. Accumulation of inflammatory mediators
   ✓ Cytokines
   ✓ Complement
4. ↓ digestion and absorption

“FIRST HIT”
Organ status post-resuscitation

Cardiac status
- electrolyte status
- neural activity
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Lung status
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“FIRST HIT”

RESUSCITATION EFFORTS
1. Reperfusion
2. Laparotomy
3. ICU therapies
4. Gut disuse
Organ status post-resuscitation

**Cardiac status**
- electrolyte status
- neural activity
- muscle activity

**Lung status**
- gas exchange
- mucosal immunity

**GUT ISCHEMIA**
1. ↑free radicals
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   - ✓ Cytokines
   - ✓ complement
4. ↓digestion and absorption

**RESUSCITATION EFFORTS**
1. Reperfusion
2. Laparotomy
3. ICU therapies
4. Gut disuse

**“FIRST HIT”**

**“SECOND HIT”**
1. Gut dysfunction
2. Acute Lung Injury
3. Acute Kidney Injury

Within 24 hrs
Why?

Local inflammatory response:
1. Pro-inflammatory:
   - ↓mucosal blood flow
   - gastric alkalinization
   - ileus
   - impaired mucosal defense
2. Anti-inflammatory:
   - ↑apoptosis of lymphocytes,
   - PMN and monocyte deactivation,
   - shift from Th1 to Th2 phenotype (immuno-suppression)

Reperfusion →
promotes distribution of pro-inflammatory cytokines to the circulation:
- ↑PMN sequestration in target organs
- → Liver, lungs, kidney
- → MOF (multi organ failure)
Why?

Early isotonic crystalloid resuscitation →
- ↑inflammation
- ↑edema
- promote ileus.

Laparotomy with bowel manipulation →
- ↑gut inflammation
- ↑mucosal injury
- ileus

ICU interventions:
- Vasopressors = ↓mucosal perfusion
- Stress gastritis prophylaxis = ↑gastric alkalinization
- Opiates worsen ileus
- Antibiotics ↑bacterial overgrowth
- TPN = gut disuse → ↓local gut immunity and ↑systemic CARS.
Why?

NPO (nothing per os)
TPN (total parenteral nutrition) →
- Gut disuse →
- ↓ local gut immunity →
- worsening systemic CARS.

Mechanism:
1. ↓ mucosal blood flow
2. Epithelial and WBC apoptosis (↑ pro-inflammatory status)
3. ↓ mucosal defense to infection
4. ↓ secretory IgA
5. ↑ bacterial translocation
Immunologic phases of injury

↑inflammation → organ dysfunction

↑immunosuppression → infection → organ dysfunction

24 hours

Inflammation and organ failure in the ICU

**SIRS**
- TNFα, IL-1β, IL-6, IL-12, IFNγ, IL-3

**Tissue inflammation, Early organ failure (MOF) and Death**

**CARS**
- IL-10, IL-4, IL-1ra, Monocyte HLA-DR suppression

INSULIN RESISTANCE
Insulin resistance

Stress/Injury

- WBC, endothelium: ↑cytokines
- Adrenals: ↑cortisol
- Liver: ↑glycogenolysis, ↑gluconeogenesis
- ↑catecholamines, ↑glucagon
- ↓enteral nutrition

HYPERGLYCEMIA

1. ↑energy requirements → malnutrition
2. ↑inflammatory environment → SIRS
3. ↑susceptibility to infection → sepsis
4. ↑coagulable state of microcirculation → DIC
Blood glucose and mortality

Insulin resistance

Stress/Injury

- WBC, endothelium ↑ cytokines
- Adrenals ↑ cortisol
- Liver:
  - ↑ glycogenolysis
  - ↑ gluconeogenesis
- ↑ catecholamines
- ↑ glucagon
- ↓ enteral nutrition

HYPERGLYCEMIA

Managed By:
1. Glucose control (insulin – tight or pragmatic)
2. Early enteral feeding (effect of incretins, GLP-1 on glucagon/insulin)
Strict glucose control with insulin

EARLY ENTERAL NUTRITION
“NPO until further orders”
Early enteral nutrition

Mechanics:
1. Once vital signs are stable
   START Gastric/small bowel feeding
2. 10-15 ml/hr enteral pump
3. Gastric residual protocol
4. Pharmaconutrition:
   ✓ Fish oils
   ✓ Glutamine
   ✓ Arginine
   ✓ Antioxidants

1. Intraluminal nutrients reverse shock-induced mucosal hypoperfusion
2. Sustains mucosal cell quality and function
3. Mucosal immunity sustained
4. Reverse impaired intestinal transit
5. ↓ ileus mediated bacterial translocation
6. Reverses CARS

Window of opportunity = 24 to 48 hrs

• Requires protocols of feeding and gastric residual volume decision
• Needs calorie and protein counting practice
• Strict fluid balance
Early enteral nutrition

Early Enteral Nutrition in Critically Ill Patients With a High-Protein Diet Enriched with Arginine, Fiber, and Antioxidants Compared With a Standard High-Protein Diet. The Effect on Nosocomial Infections and Outcome

Feeding pathways

Can the GIT be used?

Yes

- Tube feed
  - More than 3-4 weeks
    - No: NGT
      - Nasoduodenal or nasojejunal
    - Yes: Oral
      - < 75% intake
        - No: Parenteral nutrition
          - Short term: Peripheral PN
        - Yes: Oral
          - More than 3-4 weeks
            - No: NGT
              - Nasoduodenal or nasojejunal
            - Yes: Gastrostomy
              - Jejunostomy

No

- “Inability to use the GIT”
  - “inadequate intake”
    - Parenteral nutrition
      - Short term: Peripheral PN
      - Long term: Central PN

Parenteral nutrition

**COMPOSITION**

1. Carbohydrates
2. Lipids
   - LCT (structural)
   - MCT (energy)
   - Fish Oils (immuno-modulation)
3. Protein
   - BCAA
   - Glutamine
4. Vitamins/Trace elements
5. Antioxidants

1. Sustains cellular metabolism and functions (MACRO & MICRONUTRIENTS)
2. Sustains mucosal cell quality and function (=GLUTAMINE)
3. Mucosal immunity sustained (GLUTAMINE & FISH OILS)
4. Reverses CARS (FISH OILS, GLUTAMINE, ANTIOXIDANTS)

- Requires protocols for access, feeding patterns, delivery
- Needs calorie and protein counting practice
- Strict fluid balance
- MAY BE TOTAL PARENTERAL OR SUPPLEMENTAL PARENTERAL NUTRITION
MCT vs. LCT

Gase exchange during lipid infusion in septic patients with ARDS

* repeated measures ANOVA
Smirniotis et al. Int Care Med 1998; 24: 1029-33
PHARMACONUTRITION
Diet and inflammation

TRAUMA / TUMOR

Diet ↑ In LCT

WBC

Oily Fish
Fish Oils
EPA/DHA

WBC

TNF, IL1, IL6, IL12, IL3, IFNg

Higher Inflammatory Reaction/State

Macrophage, T&B cells
Endothelium, fibroblasts
Epithelial cells, mast cells

Lesser Inflammatory Reaction/State

Macrophage, T&B cells
Endothelium, fibroblasts
Epithelial cells, mast cells

PRO-INFLAMMATORY PHASE

Griffiths RD 2001; Calder P, 2005
Diet and inflammation

TRIUMA / TUMOR

IL10, IL4, monocyte HLA-DR suppression

Higher Immunosuppression State

Monocytes, Th2 cells
Mast cells

Body

Diet ↑ In LCT

WBC

Monocytes, Th2 cells
Mast cells

Lesser Immunosuppression State

IL10, IL4, monocyte HLA-DR suppression

Oily Fish
Fish Oils
EPA/DHA

WBC

IMMUNOSUPPRESSION PHASE

Griffiths RD 2001; Calder P, 2005
Immune modulating nutrients

• Another name = pharmaconutrients
  – Fish oils
  – Glutamine
  – Antioxidants
  – Arginine

EPA, GLA, antioxidants (enteral)

EPA, antioxidants, zinc, selenium

Lipid emulsions with Fish Oils

Meta-analysis: effect of lipid emulsion containing fish oil on infectious morbidity

Injury induced immunosuppression - management

Surgery

Arginine Deficiency (resistant to post-operative supplementation)

↑CD16+ granulocytes express arginase 1

↓Plasma arginine by 50%

↓T-lymphocyte growth and function

Impairment of Acquired Immunity

Fish oils inhibits arginase 1
1. Banzal V et al. JPEN 2005

Glutamine enhances T-cell proliferation and activity

Banzal V et al. JPEN 2005
Asprer JM et al. Nutrition 2006
Effect of $\Omega$3 and $\Omega$6 lipids on liver function in cancer surgery patients

Glutamine

- Low glutamine independent of mortality
- Doubled mortality
  - Gln < 0.42 mmol/L
  - \( p = 0.013 \)

Figure 3. Risk ratios (RR) and associated 95% confidence intervals (CI) for the effect of glutamine on infectious complications.

Figure 2. Risk ratios (RR) and associated 95% confidence intervals (CI) for the effect of glutamine on mortality.

Antioxidants

- Superoxide dismutase
- Oxygen radicals $O_{2}^{•}$
- Glutathione peroxidase
- Glutathione reductase
- Catalase
- Hydrogen peroxide $H_2O_2$
- Glutathione
- Vitamin C
- ONOO$^-$
- Hydrogen peroxide $H_2O$
- ONO$^-$ + H$_2$O

Munoz C. Trace elements and immunity: Nutrition, immune functions and health; Euroconferences, Paris; June 9-10, 2005;
Antioxidants

1. α-tocopherol
   1,000 IU (20 mL) q 8h per naso- or orogastric tube
2. ascorbic acid
   1,000 mg given IV in 100 mL D5W q 8h for the shorter of the duration of admission to the ICU or 28 days.

Inflammation and organ failure in the ICU

**SIRS**
- TNFα, IL-1β, IL-6, IL-12, IFNγ, IL-3

**CARS**
- IL-10, IL-4, IL-1ra, Monocyte HLA-DR suppression

Insult (trauma, sepsis)

Tissue inflammation, Early organ failure and death

Early EN + pharmaconutrition

ANTIBIOTICS AND PRO/PRE-BIOTICS
Guidelines

• Prophylaxis: single pre-operative dose; one hour before incision
• Patients on antibiotics: continue
• Prolonged surgery: repeat antibiotics within 4 hours of procedure
• Per 1500 ml of blood loss: give antibiotics
• Type of antibiotics: broad spectrum recommended by infection committee

EBM guidelines: ACS
Antibiotics and gut microflora

• Antibiotics → alterations in gastrointestinal microbiota composition → ↑disease risk → by ↑susceptibility to gastrointestinal infections.
  – Antibiotic-associated diarrhea and colitis → *Clostridium difficile* or *Clostridium perfringens* (human study)
  – Increased susceptibility to invasive *salmonellosis* after streptomycin and vancomycin (animal study)
  – Antibiotic therapy for children infected with *E. coli* strain O157:H7 → ↑*risk of hemolytic-uremic syndrome* (human study)
  – Antimicrobial treatment for *Helicobacter pylori* induces marked disturbances in the intestinal microbiota. (human study)

Prebiotics


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Figure 2. The effect of probiotics supplementation vs. without probiotics on eradication rates by intention-to-treat analysis. (*n* = number of successful eradication; *N* = number of participants).
ADEQUATE INTAKE
Nutrition strategies in critical care

Know the nutritional status/risk level to give accurate nutrient requirements

Correct the imbalances

Deliver all requirements through the most appropriate route

Make sure adequate intake is achieved

Readjust mgt as soon as there is need

Use special substrates

Body composition changes
- protein loss
- carbohydrate loss
- fat loss
- fluid imbalance
- electrolyte imbalance
- trace element changes

Organ changes:
- dysfunction
- failure

More organs affected →
- more morbidity
- higher mortality

Factors influencing nutrition:
- inflammation factors
- stress factors, hormones
- wound healing, repair
- function priorities
Adequacy of feeding in ICU

Comparison of recommended vs. actual calorie intake, geriatric intensive care patients

Nutrition intake and infection(s)

Adequate intake and outcome

Nutrition team and intake

Llido et al. Nutrition support team supervision improves intake of critical care patients in a private tertiary care hospital in the Philippines: report from years 2000 to 2011
Nutrition team and intake

Llido et al. Nutrition team supervision improves intake of critical care patients in a private tertiary care hospital in the Philippines: report from years 2000 to 2011

* p < 0.05, T-Test
Nutrition intake and risk reduction

Refeeding syndrome

- Severely malnourished, geriatric, low electrolyte values, artificial nutrition
  1. **Sodium and water retention** → fluid overload, edema, heart failure
  2. **Hypophosphatemia** → ventilatory failure, rhabdomyolysis
  3. **Hypokalemia** → cardiac arrhythmia, ventilatory failure, rhabdomyolysis, ileus
  4. **Hypomagnesemia** → cardiac arrhythmia, rhabdomyolysis
  5. **Vitamin deficits (thiamine)** → encephalopathy, lactic acidosis

E. Fiaccadori. Fluids and electrolytes. PN Workshop 2009, Kuala Lumpur, Malaysia
Targeted nutrition

• Burns
• Surgical site infections
• Pressure ulcers
MANAGEMENT STRATEGIES
### Surgical critical care

- Critical illness following surgery or trauma
- High risk surgical patients
- Shock and hemodynamic compromise
- Acute lung injury and ARDS following surgery, trauma, or pancreatitis
- Sepsis and severe infections
- Trauma evaluation and management
- Neurologic emergencies

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<td>Severe acute pancreatitis</td>
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Management strategies

• Shock and hemodynamic compromise / Acute lung injury following surgery, trauma, pancreatitis
  – Resuscitation
    • Choose appropriate solutions
  – Stabilize microcirculation
    • Maintain oxygen delivery and perfusion
    • Renal support
  – Early feeding within 24 hrs – pharmaconutrition
    • Increasing daily vitamin, trace elements, glutamine, fish oils
    • Pre/probiotics with antibiotics
  – Strict glucose control
Management strategies

- High risk surgical patients / critical illness following surgery or trauma
  - Severely malnourished
  - Immunosuppressed
  - Management:
    - Pre-operative build up
    - Zero fluid balance
    - Post-op early feeding
    - Adequate intake + pharmaconutrition
    - Targeted nutrition
Management strategies

• Sepsis and severe infections/postoperative complications/peritonitis, ruptured viscus
  – Prevention:
    • Pre-op - severely malnourished build up
    • Intra-op: zero-fluid balance
    • Post op: early enteral nutrition and zero-fluid balance
    • Adequate nutrient delivery
      – Macronutrients
      – Daily vitamins and trace elements
    • Pharmaconutrition
    • Antibiotics, culture& sensitivity, pre/probiotics
Management strategies

• Enterocutaneous fistulas
  – severely malnourished: build up
  – Post op:
    • High output: parenteral nutrition
    • Medium to low: combined enteral and parenteral nutrition
  – Adequate nutrient delivery
    • Macronutrients
    • Daily vitamins and trace elements
  – Pharmaconutrition
  – New substrates