Critical Care Nutrition
Discussion points

• Nutrition screening and assessment
  • Prognosis of surgical patients with critical care status
  • Severe malnutrition and complication rates

• Nutrition care plan
  • Enteral nutrition
  • Immunonutrition
  • Adequate intake
  • Fluid management

• Prehabilitation
The metabolic picture of a critical care patient

Persistent Inflammatory immunosuppressed catabolic syndrome

The metabolic picture of a critical care patient

1 - understand the pathogenesis and how to manipulate the inflammatory process

2 - understand the impact of the inflammatory process on the microcirculation of the affected organ systems

3 – to appreciate how nutrition reserves and adequate intake through different access routes can make a difference in the critical care status

4 – to understand the role of the gut, microbiome, immuno-nutrition, lean body mass enhancement and fluid management in the outcome of the critical care patient

Persistent Inflammatory immunosuppressed catabolic syndrome

## Post-operative mortality – predictors

<table>
<thead>
<tr>
<th>Patient Factors</th>
<th>Outcome</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>OR: 1.03</td>
<td>Arenal et al (2003)</td>
</tr>
<tr>
<td></td>
<td>OR: 1.15</td>
<td>Cook et al (1998)</td>
</tr>
<tr>
<td>&gt;80y</td>
<td>OR: 3.77</td>
<td>Modini et al (2012)</td>
</tr>
<tr>
<td>Sex, male</td>
<td>OR: 1.05</td>
<td>Arenal et al (2003)</td>
</tr>
<tr>
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<td>OR: 5.88</td>
<td>Cook et al (1998)</td>
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<tr>
<td>ASA &gt;3</td>
<td>OR: 10.41</td>
<td>Leong et al (2009)</td>
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<td>OR: 3.87</td>
<td>Modini et al (2012)</td>
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<td>Hx of COPD</td>
<td>OR: 1.79</td>
<td>Kwok et al (2011)</td>
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<td>Hx of CHF</td>
<td>OR: 1.87</td>
<td>Kwok et al (2011)</td>
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<tr>
<td>Hx of neurologic disease</td>
<td>OR: 4.47</td>
<td>Modini et al (2012)</td>
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</table>

# Post-operative mortality – predictors

<table>
<thead>
<tr>
<th>Disease Factors</th>
<th>Outcome</th>
<th>Study</th>
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</thead>
<tbody>
<tr>
<td>APACHE 2 score</td>
<td>OR: 1.13</td>
<td>Fukuda et al (2012)</td>
</tr>
<tr>
<td>Creatinine &gt;1.5</td>
<td>▲ OR: 2.57</td>
<td>Kwok et al (2011)</td>
</tr>
<tr>
<td>&gt;2 failing organs</td>
<td>▲ OR: 5.51</td>
<td>Okubo et al (2008)</td>
</tr>
<tr>
<td>Metastatic disease</td>
<td>▲ OR: 5.26</td>
<td>McGillicuddy et al (2009)</td>
</tr>
</tbody>
</table>

Post-operative mortality – role of nutrition

Nutrition Risk Score:
- 1-3: Low Risk
- 4-6: Moderate Risk
- 7-9 High Risk

Patient need #1: Nutrition screening and assessment
Role of nutrition in surgical practice

• The need for nutrition screening and assessment of all preoperative patients
  • All severely malnourished or “nutritionally high risk” patients should be nutritionally built up
  • Prehabilitation

• The role of training
  • Survey for the presence of nutritional screening and assessment tools in all training institutions
  • There is an on-going seminar on critical care nutrition for all chapters of surgical training which may be completed within two years
Nutrition screening

Components:

- Height and weight – only accomplished in hospitals with a clinical nutrition program
- Body mass index – also accomplished only in hospitals with a clinical nutrition program
- Weight loss severity – rarely asked even in hospitals with a clinical nutrition program
- Acute loss in intake – only a few really understand why this is asked
- Critical care patient – only a few realize this information is part of the nutrition screening of the patient
The modified SGA form of PhilSPEN

SGA
- A (normal)
- B (mild/mod malnutrition)
- C (severe malnutrition)

Nutrition Risk Score:
- 1-3: Low Risk
- 4-6: Moderate Risk
- 7-9 High Risk

Sensitivity: 94.7%
Specificity: 96.2%
Positive Predictive Value: 95.7%

Lacuesta-Corro L et al. The results of the validation process of a Modified SGA (Subjective Global Assessment) Nutrition Assessment and Risk Level Tool designed by the Clinical Nutrition Service of St. Luke's Medical Center, a tertiary care hospital in the Philippines.

http://philspenonlinejournal.com/POJ_0002.html
Malnutrition and surgical outcomes

SGA
- A (normal)
- B (mild/mod malnutrition)
- C (severe malnutrition)

Nutrition Risk Score:
- 1-3: Low Risk
- 4-6: Moderate Risk
- 7-9 High Risk

Patient need #2: Nutrition care plan
For pre-op and post-op critical care patients

• Physiology shows that healing and recovery require cell proliferation and enhanced metabolic activity
• Physiology dictates that the optimum inflammation environment should be sustained
• Physiology also dictates that there should be adequate calorie and protein to achieve the above ends and ensure minimum complications and low mortality especially for the malnourished patients
Wound Healing = increased cell proliferation and metabolic activity
Wound Healing = increased cell proliferation and metabolic activity
Wound Healing = increased cell proliferation and metabolic activity

FEATURES
- There is massive cellular proliferation and synthesis of growth factors
- There is need of huge amounts of macronutrients and micronutrients
- Bottom line – the need for adequate supply/reserves of nutrients
Wound Healing

FAILURE FACTORS

Malnutrition
- Poor protein reserves
- Less energy supply
- Fat > higher inflammatory state

Poor intake
- Poor nutrient supply
- Poor quality of wound healing
- Other complications like dehiscence, ulcers, fistulas

Resolution Process
- Success > good wound healing
- Failure > poor healing / sepsis

- Neutrophils
- Macrophages > active resolution

- Collagen
- Basement membrane
- Angiogenesis

Days  Weeks  Months +
Wound healing and the resolution process

RESOLUTION is an ACTIVE PROCESS

- Neutrophils pack up and leave
- Macrophages clear up the environment; starts the restoration to normal
- Fibroblasts start the rebuilding process

FAILURE of RESOLUTION

- Chronic inflammation:
  - Abscess formation
  - Excess scarring
  - Auto-immunity
Resolution is an active process

- **The pro-inflammatory mechanisms probably are counterbalanced by endogenous anti-inflammatory signals that serve to temper the severity and limit the duration of the early phases**, which leads to their **resolution**, an active rather than a passive process.

- The resolution of the inflammatory response is **mainly mediated by families of local-activity mediators that are biosynthesized from essential fatty acids eicosapentaenoic acid and docosahexaenoic acid.**

- These resolution mediators were **termed resolvins and protectins.**

- Inflammation resolution is **also mediated by lipoxins, trihydroxystearin-containing eicosanoids that are generated within the vascular lumen through platelet-leukocyte interactions.**

### Macrophage or WBC

<table>
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<tr>
<th>AA</th>
<th>AA</th>
<th>AA</th>
<th>EPA</th>
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<td>DHA</td>
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<td>EPA</td>
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<td>AA</td>
<td>EPA</td>
<td>AA</td>
<td>EPA</td>
<td>AA</td>
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</table>

**Cell Membrane – Lipid Layer**

- **Arachidonic Acid**
  - COX2
  - 5-LOX
  - 2S/PG & TX
  - 4S/LT
- **DHA**
  - COX2
  - DS/Resolvins
  - 3S/PG & TX
- **EPA**
  - COX2
  - ES/Resolvins
  - 5S/LT

**Phospholipase A2**

- **Injury**

**NFκB**

**↑ Inflammatory**

**↓ Inflammatory**

**Lipoxins, Protectins**

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**COX** = Cyclooxygenase; **LOX** = LipoOxygenase; **PG** = Prostaglandins; **LT** = Leukotrienes; **TX** = Thromboxanes; **NFκB** = Nuclear Factor Kappa B; **EPA** = Eicosapentanoic Acid; **DHA** = Docosahexanoic Acid

**Calder Philip,** *Polynsaturated fatty acids, inflammation, and immunity: Nutrition, immune functions and health; Euroconferences, Paris; June 9-10, 2005*
Macrophage or WBC

CELL MEMBRANE - LIPID LAYER

<table>
<thead>
<tr>
<th>AA</th>
<th>AA</th>
<th>AA</th>
<th>EPA</th>
<th>AA</th>
<th>AA</th>
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<th>EPA</th>
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<td>AA</td>
<td>AA</td>
<td>AA</td>
<td>AA</td>
<td>EPA</td>
<td>AA</td>
</tr>
</tbody>
</table>

Phospholipase A2

FISH OILS

GLUTAMINE

ARACHIDONIC ACID

COX2

5-LOX

2S/PG & TX

4S/LT

COX2

DHA

EPA

COX2

DS/Resolvins

3S/PG & TX

3S/Resolvins

5S/LT

ES/Resolvins

5S/Resolvins

↑ INFLAMMATORY

↓ INFLAMMATORY

Lipoxins, Protectins

COX=Cyclooxygenase; LOX=Lipoxygenase; PG=Prostaglandins; LT=Leukotrienes; TX=Thromboxanes; NFKB=Nuclear Factor Kappa B; EPA=Eicosapentaenoic Acid; DHA=DocosaHexanoic Acid

Calder Philip, Polyunsaturated fatty acids, inflammation, and immunity: Nutrition, immune functions and health; Euroconferences, Paris; June 9-10, 2005
Fish oils (EPA / DHA)


EPA=500mg-1gm/day
GLA=500mg/day

Old 1999 data

Reduction in
- Ventilator days
- ICU days
Fish oils (EPA / DHA)


Conclusion: Omega-3 nutritional supplementation may reduce ICU length of stay and duration of mechanical ventilation without significantly affecting mortality
Glutamine

* P < 0.05

Old 2008 data

Reduction of pro-inflammatory cytokines

Glutamine

Animal study, sepsis model (cecal ligation)


- significantly reduces hospital mortality, infectious complication rates, and hospital LOS
- analysis indicates the importance of delivering glutamine dipeptides together with adequate parenteral energy and nitrogen so that the administered glutamine serves as precursor in various biosynthetic pathways rather than simply as a fuel.
<table>
<thead>
<tr>
<th>Complication</th>
<th>Standard Enteral (n=153)</th>
<th>Immunonutrition (n=152)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious complications</td>
<td>60 (39.22)</td>
<td>43 (28.29)</td>
<td>0.043</td>
</tr>
<tr>
<td>Surgical wound infection</td>
<td>27 (17.65)</td>
<td>12 (7.89)</td>
<td>0.010</td>
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<tr>
<td>Mortality</td>
<td>9 (5.88)</td>
<td>2 (1.32)</td>
<td>0.032</td>
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<tr>
<td>Overall morbidity</td>
<td>72 (47.06)</td>
<td>51 (33.55)</td>
<td>0.016</td>
</tr>
<tr>
<td>Other infectious complications</td>
<td></td>
<td>No significant difference</td>
<td></td>
</tr>
<tr>
<td>Other complications</td>
<td></td>
<td>No significant difference</td>
<td></td>
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### Macronutrients and micronutrients

<table>
<thead>
<tr>
<th>Requirements</th>
<th>Dose</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total calories per day</td>
<td>25-30 kcal/kg actual body weight</td>
<td>For obese or BMI &gt;30 use ideal body weight</td>
</tr>
<tr>
<td>Total protein per day</td>
<td>1.5 -2.5 g/kg actual body weight</td>
<td>If serum creatinine is normal</td>
</tr>
<tr>
<td>Non-protein calories</td>
<td>50% carbo: 50% fat to 60% carbo to 40% fat</td>
<td>Lipids range from saturated to unsaturated fatty acids</td>
</tr>
<tr>
<td>Vitamins</td>
<td>Water and fat soluble vitamins daily</td>
<td>Both EN / PN</td>
</tr>
<tr>
<td>Trace elements</td>
<td>Daily</td>
<td>Both EN / PN</td>
</tr>
<tr>
<td>Immuno-nutrition</td>
<td>Fish oils, glutamine, antioxidants, arginine</td>
<td>Either EN / PN</td>
</tr>
</tbody>
</table>
Feeding pathway

- It is not about adequate nutrition in the first or second week post-op
- It is all about adequately delivering nutrition requirements for the whole post-op period (which may take weeks to months)

Enteral nutrition

• All guidelines point to enteral nutrition as the feeding of choice
• Major issues/choices:
  • Type of access: NGT, PEG, gastrostomy, jejunostomy
  • Timing of feeding: continuous, intermittent
  • Adequacy of intake: 70% to 80% of calculated intake
### Physiologic basis for early enteral feeding

<table>
<thead>
<tr>
<th>Metabolism Maintenance</th>
<th>Motility</th>
<th>Maintenance</th>
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</thead>
<tbody>
<tr>
<td>Gastrin</td>
<td>Gastric acid, pepsin, <strong>mucosa growth/repair</strong></td>
<td><strong>Glycogenolysis, gluconeogenesis, lipolysis</strong></td>
</tr>
<tr>
<td>Glucagon (A cells)</td>
<td>↑bicarbonate secretion (panc duct, bile duct)</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
</tr>
<tr>
<td>Secretin</td>
<td>Stimulates insulin secretion (gliptin)</td>
<td><strong>Stimulates insulin secretion (gliptin)</strong></td>
</tr>
<tr>
<td>CCK</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>(2) <strong>Muscle contraction</strong></td>
</tr>
<tr>
<td>GIP</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Inhibits gastrin, secretin, VIP, GIP, motilin</td>
</tr>
<tr>
<td>Motilin</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Gastrin secretion</td>
</tr>
<tr>
<td>Neurotensin</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Inhibits food intake, gastric inhibitory peptide</td>
</tr>
<tr>
<td>VIP</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Inhibits food intake, gastric inhibitory peptide</td>
</tr>
<tr>
<td>Substance P</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Inhibits food intake, gastric inhibitory peptide</td>
</tr>
<tr>
<td>Glicentin (L cells)</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Inhibits food intake, gastric inhibitory peptide</td>
</tr>
<tr>
<td>Somatostatin</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Inhibits food intake, gastric inhibitory peptide</td>
</tr>
<tr>
<td>GRP</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Inhibits food intake, gastric inhibitory peptide</td>
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<tr>
<td>Guanylin</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Inhibits food intake, gastric inhibitory peptide</td>
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<tr>
<td>Peptide YY</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Inhibits food intake, gastric inhibitory peptide</td>
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<tr>
<td>Ghrelin</td>
<td><strong>Gallbladder contraction, ↑pancreatic juice rich in enzymes</strong></td>
<td>Inhibits food intake, gastric inhibitory peptide</td>
</tr>
</tbody>
</table>

Physiologic basis for early enteral feeding

Feed within 24 to 48 hours post-op

Metabolism

Maintenance

Motility

Gastric acid, pepsin, **mucosa growth/repair**

Glycogenolysis, gluconeogenesis, lipolysis

↑ bicarbonate secretion (panc duct, bile duct)

Gallbladder contraction, ↑ pancreatic juice rich in enzymes

Stimulates insulin secretion (gliptin)

(1) Muscle contraction

↑ GI motility, ↑ ileal blood flow

↑ secretion of electrolytes and water; relaxes smooth muscle including sphincters

(2) Muscle contraction

Glucagon (GLP-1, GLP-2) - Glycogenolysis, gluconeogenesis, lipolysis

Inhibits gastrin, secretin, VIP, GIP, motilin

Gastrin secretion

↑ growth hormone, central control of food intake

NORMAL

NON-UTILIZATION IN ONE WEEK

Dog Intestinal epithelium

Critical Point: 3-5 days

http://www.vivo.colostate.edu/hbooks/pathphys/digestion/stomach/gibARRIER.html
Physiologic basis for early enteral feeding

**BENEFITS of ENTERAL FEEDING**
- Early bowel motility recovery
- Gut mucosa defense is maintained
- Gut microbiome is maintained
- Faster wound healing
- Preserved immune status

---

**Fundus Antrum Duodenum Jejunum Ileum Colon**

- **Gastrin**
  - Gastric acid, pepsin, *mucosa growth/repair*
- **Glucagon (A cells)**
  - Glycogenolysis, gluconeogenesis, lipolysis
- **Secretin**
  - Bicarbonate secretion (pancreas, bile duct)
- **CCK**
  - Gallbladder contraction, ↑pancreatic juice
- **GIP**
- **Motilin**
  - Muscle contraction
  - Inhibits gastrin, secretin, VIP, GIP, motilin
  - Secretion of chloride to lumen
- **Neurotensin**
- **VIP**
  - Inhibits food intake, gastric inhibitory peptide
  - ↑growth hormone, central control of food intake
- **Glicentin (L cells)**
- **Somatostatin**
- **GRP**
  - Secretion of chloride to lumen
- **Guanylin**
- **Peptide YY**
- **Chrelin**

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Gut associated lymphoid tissues
Relationship of GALT and MALT

**GIT**
- **M-cells: Antigen Presenting Cells (APC)**
- Peyer’s Patches - GALT
- Lymphocyte activation
  - VITAMIN D3
  - T-Cells upregulate gut homing receptors
  - Lamina propria –
    - Assist IgA forming B-Cells
    - Secretory IgA
    - T-Cell/B-Cell clones

**PULMONARY**
- Alveolar macrophage (APC)
- Respiratory LN - MALT
- The role of the gut in immune competence

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The microbiome

Normal adult gut microbiota by age three (3) years old

- Total cells in the body = 37 trillion
- Total bacteria in the body = 100 trillion

**Proteobacteria**
- wide variety of pathogens
  - Class: Beta-proteobacteria: *Bordetella pertussis*
  - Class: Gamma-proteobacteria: *Acinetobacter, Pseudomonas, Escherichia coli, Salmonella, Serratia marcescens, Helicobacter, Yersinia*

Microbiome: protective/beneficial mechanisms

**Gut mucosa**
- Intact mucosal immune defense
- Intact maturation and growth
- Better response to inflammatory insults
- Tolerance to diverse microbiota

**GALT**
- Active and fully competent
- Effective T-cell maturation to cellular or humoral immunity
- Concomitant protection of the MALT

**Liver and spleen**
- Intact immune cells/nodes

**Antibody**
- Full secretion of IgA

**Immune response**
- Controls NFkB activity
- Good immune-modulation
- SCFA provision

**Metabolism**
- Liver metabolism (macro & micronutrient)
- Drug disposal
- Xenobiotic metabolism

**Angiogenesis**
- Wound healing
Collapse of the Microbiome, Emergence of the Pathobiome, and the Immunopathology of Sepsis

John C. Alverdy, MD, FACS, Monika A. Krezalek, MD

Abstract: The definition of sepsis has been recently modified to accommodate emerging knowledge in the field, while at the same time being recognized as challenging, if not impossible, to define. Here, we seek to clarify the current understanding of sepsis as one that has been typically framed as a disorder of inflammation to one in which the competing interests of the microbiota, pathobiota, and host immune cells lead to loss of resilience and nonresolving organ dysfunction. Here, we challenge the existence of the idea of noninfectious sepsis given that critically ill humans never exist in a germ-free state. Finally, we propose a new vision of the pathophysiology of sepsis that includes the invariable loss of the host’s microbiome with the emergence of a pathobiome consisting of both “healthcare-acquired and healthcare-adapted pathobiota.” Under this framework, the critically ill patient is viewed as pathway-blocking agents remains a pervasive line of inquiry. Virtually, every report on sepsis begins with the declaration that the problem is increasingly affecting more than 750,000 patients each year and that more than 200,000 patients in the United States die each year of sepsis (2). Grants and review articles continue to claim that sepsis and its associated mortality rate are escalating at an alarming rate. Like cancer, there is now a war on sepsis, and, with proper funding, the cure is right around the corner.

Yet an often overlooked fact of sepsis is that in the overwhelming majority of cases, the actual cause of death in a patient with sepsis remains ill-defined and obscured by the term itself. The main reason for this is three-fold: 1) the clinical syndrome of sepsis cannot be precisely defined in any meaningful clinical or biological context despite numerous and recent attempts (2), 2)
The microbiome

**THE PATHOBIOME**

- **Proteobacteria**
  - wide variety of pathogens
  - Class: Beta-proteobacteria: *Bordetella pertussis*
  - Class: Gamma-proteobacteria: *Acinetobacter, Pseudomonas, Escherichia coli, Salmonella, Serratia marcescens, Helicobacter, Yersinia*

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# Early enteral nutrition guidelines for critical care patients

**Grade B recommendation**

<table>
<thead>
<tr>
<th>Hours</th>
<th>Early EN: Guideline</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 48 hours</td>
<td>1 Canadian</td>
<td>Evidence of trend</td>
</tr>
<tr>
<td>&lt; 24 hours</td>
<td>2 ACCEPT</td>
<td>Significant evidence</td>
</tr>
<tr>
<td>&lt; 24 hours</td>
<td>3 Australian/New Zealand</td>
<td>Significant evidence</td>
</tr>
<tr>
<td>&lt; 24 hours</td>
<td>4 ESPEN</td>
<td>Significant evidence</td>
</tr>
<tr>
<td>&lt; 48 hours</td>
<td>5 ASPEN</td>
<td>Evidence of trend</td>
</tr>
</tbody>
</table>

3. Doig GS and Simpson F. EvidenceBased.net
Adequate intake

• There are still some nutrition groups who have not yet agreed on the issue of how much calorie and protein to give

• Local and ESPEN data show the pre-2014 guidelines are still working well and emphasize on delivering adequate intake

• Current focus is on the negative impact of hypocaloric feeding and need for higher protein doses
How much energy?

Usual: 20-25 kcal/kg/day
Very sick: 15-20 kcal/kg/day

How much protein?

Adequate intake and survival in surgery

St., Luke’s Medical Center, General Surgery (From admission to discharge)
Del Rosario et al. Available at:
http://www.philspenonlinejournal.com/POJ_0006.html
Adequate intake and survival in the ICU

Enteral feeding alone vs. Supplemental PN

Supplemental parenteral nutrition allows achievement of full adequacy of intake

Supplementary figure 2: Kaplan-Meier analysis for nosocomial infections in per-protocol analysis
SPN = supplemental parenteral nutrition. EN = enteral nutrition. *Statistically significant with Benjamini-Hochberg correction.

Daily intake and fluid balance should be done

<table>
<thead>
<tr>
<th>Date And Shift</th>
<th>Nutrient Source</th>
<th>Calorie Intake</th>
<th>TCR</th>
<th>% Calorie Intake</th>
<th>Protein Intake</th>
<th>TPR</th>
<th>% Protein Intake</th>
<th>Total Fluid Intake</th>
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<tbody>
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<td>2/1/07</td>
<td>Oral</td>
<td></td>
<td></td>
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<td></td>
<td>Tube Feed</td>
<td>900</td>
<td>1600</td>
<td>72%</td>
<td>36 g</td>
<td>52 g</td>
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<td>1100</td>
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<td>IV Dextrose</td>
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<tr>
<td></td>
<td>TOTAL</td>
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<td>36 g</td>
<td>52 g</td>
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<tr>
<td></td>
<td>Tube Feed</td>
<td>200</td>
<td>1600</td>
<td>84%</td>
<td>8 g</td>
<td>52 g</td>
<td>135%</td>
<td>240</td>
</tr>
<tr>
<td></td>
<td>IV Dextrose</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Parenteral</td>
<td>1150</td>
<td>84%</td>
<td></td>
<td>62 g</td>
<td>52 g</td>
<td>135%</td>
<td>1200</td>
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<tr>
<td></td>
<td>TOTAL</td>
<td>1350 kcal</td>
<td>84%</td>
<td></td>
<td>70 g</td>
<td>52 g</td>
<td>135%</td>
<td>1440 ml</td>
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<td>2/6/07</td>
<td>Oral</td>
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</tr>
<tr>
<td></td>
<td>Tube Feed</td>
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<td></td>
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<tr>
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<td></td>
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</tbody>
</table>
Fluid management

• There is a need to make a survey of the practice of fluid management, electrolyte status understanding and fluid balances in all training institutions

• We have a guidebook on surgical critical care which discusses fluids and electrolytes – do the residents have a copy?
Fluid overload and mortality

• Overload criteria: > 10% weight gain from pre-admission weight

• Weight gain and mortality:
  • 5% weight gain -> 10% mortality
  • 15% weight gain -> 20% mortality
  • 32% weight gain -> 100% mortality

The plasma and crystalloids given

<table>
<thead>
<tr>
<th></th>
<th>Plasma</th>
<th>0.9% Saline</th>
<th>Ringer's Lactate</th>
<th>Sterofundin</th>
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<tbody>
<tr>
<td>Na</td>
<td>135-145</td>
<td>154</td>
<td>131</td>
<td>140</td>
</tr>
<tr>
<td>K</td>
<td>3.5-5.3</td>
<td>-</td>
<td>5</td>
<td>5</td>
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<tr>
<td>Ca</td>
<td>2.2-2.6</td>
<td>-</td>
<td>2</td>
<td>0</td>
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<tr>
<td>Mg</td>
<td>0.7-1.2</td>
<td>-</td>
<td>0</td>
<td>1.5</td>
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<tr>
<td>Cl</td>
<td>95-105</td>
<td>154</td>
<td>111</td>
<td>98</td>
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<td>Bicarb precursor</td>
<td>24-32</td>
<td>-</td>
<td>Lactate 29</td>
<td>Acetate 27</td>
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<td>Na:Cl ratio</td>
<td>1.28-1.45:1</td>
<td>1:1</td>
<td>1.18:1</td>
<td>1.43:1</td>
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<tr>
<td>Osmolality</td>
<td>275-295</td>
<td>308</td>
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When you add dextrose to all of the above > HYPO-OSMOLAR

*Balanced electrolyte solution*
The effect of fluid overload

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<tr>
<th></th>
<th>Restricted Group n=69</th>
<th>Standard Group n=72</th>
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<tbody>
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<td>Overall complications</td>
<td>21</td>
<td>40</td>
<td>0.003</td>
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<td>Major complications</td>
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<td>18</td>
<td>0.040</td>
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<tr>
<td>Minor complications</td>
<td>15</td>
<td>36</td>
<td>&lt;0.001</td>
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<tr>
<td>Tissue-healing complications</td>
<td>11</td>
<td>22</td>
<td>0.040</td>
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<tr>
<td>Cardiopulmonary complications</td>
<td>11</td>
<td>22</td>
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</tr>
<tr>
<td>Deaths</td>
<td>0</td>
<td>4</td>
<td>0.12</td>
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</table>

The effect of water and salt overload

Salt and water overload

↑ intra-abdominal pressure

↓ mesentery blood flow

STAT3 activation
↓ myosin phosphorylation

↓ muscle contractility

ILEUS

Intestinal edema

↓ tissue OH-proline

Intramucosal acidosis

Impaired wound healing

DEHISCENCE

The effect of water and salt overload

Salt and water overload

↑ intra-abdominal pressure
↓ mesentery blood flow
STAT3 activation
↓ myosin phosphorylation
↓ muscle contractility

Intestinal edema

The daily accumulated fluid balance should not go beyond 75% of the interstitial fluid volume (=12% of the patient’s weight)

ILEUS

DEHISCENCE

Prehabilitation > Can help reduce critical care situations

• Observation and proposal:
  • Patients who are elderly, malnourished, anxious, and have a low physical function before surgery are likely to have suboptimal recovery.
  • A multimodal prehabilitation program is proposed, consisting of exercise training and nutritional and psychological support, which increases physiologic reserve before the stress of surgery.
  • The integration of exercise, adequate nutrition, and psychosocial components, with medical and pharmacologic optimization in the presurgical period, deserves to receive more attention by clinicians.

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Summary: Critical care nutrition

• Involves understanding of the critical care environment and mechanics including the microbiome
• Involves identifying the patients who are at high risk of going into a critical care state
• Involves providing macro and micronutrients through access routes that ensure adequate levels daily
• Involves the practice of early enteral nutrition and supplementary parenteral nutrition
• Involves prudent fluid management
• Will be improved on with the practice of prehabilitation
Thank You