Clinical Nutrition: Dispelling Myths, Embracing Realities

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TODAY’s TOPICS
► Identifying/defining malnutrition—NO protein marker!!
► Vitamin deficiencies – what will you really see??
► Enteral nutrition (tube feedings)
  ● ICU when/why/where?
  ● bowel sounds
  ● gastric residuals
  ● diarrhea
► Parenteral nutrition (TPN)
  ● more is NOT better
  ● who/when?

Malnutrition Incidence and Adverse Outcomes
Associated w/ Malnutrition (JPEN July 2013;37(4):482-497)
• > 1/3 of pts in industrialized countries are malnourished upon hospital admission; if left untreated, approximately 2/3 will experience further decline in nutrition status during hospital stay
  ✔ Adverse Outcomes
  ➤ pressure ulcers ➤ higher treatment costs
  ➤ increased infection rate ➤ increased mortality
  ➤ immune suppression
  ➤ increased LOS
  ➤ higher readmission rates
  ➤ muscle wasting/functional loss ➤ increased fall risk

1st Question in Defining Malnutrition: Clinical Parameters - Inflammation
• Fever
• Hypothermia
• Infection
• UTI
• PNA
• Blood stream infection
• Wound or incisional infection
• Abscess

If inflammation, ? Chronic Disease/illness: Mild to Moderate Inflammatory Response
• CVD
• Celiac Disease
• Chronic pancreatitis
• COPD
• CHF
• CF
• Dementia
• DM
• IBD
• Hematologic malignancies
• Neuromuscular disease
• Obesity
• Organ failure/transplant
• Pressure ulcers
• RA
• Solid tumors

Figure 1. Etiology-based malnutrition definitions.
Jane V. White et al. JPEN J Parenter Enteral Nutr 2012;36:275-283
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If inflammation, ? Acute Disease/Injury: Severe Inflammatory Response

- ARDS
- CHI
- Critical illness
- Major abdominal surgery
- Major infection/sepsis
- multi-trauma
- SIRS
- Burns
- SAP
- Burns
- SAP


Table 1. General Characteristics for the Diagnosis of Malnutrition:

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Acute Illness/Injury</th>
<th>Chronic Illness</th>
<th>Social/Environmental</th>
</tr>
</thead>
<tbody>
<tr>
<td>5d Intake</td>
<td>&lt;75%</td>
<td>&lt;50%</td>
<td>&lt;75%</td>
</tr>
<tr>
<td>Weight Loss</td>
<td>1 week</td>
<td>1-2%</td>
<td>&gt;2%</td>
</tr>
<tr>
<td>Body Fat</td>
<td>mild</td>
<td>mod</td>
<td>mild</td>
</tr>
<tr>
<td>Muscle Loss</td>
<td>mild</td>
<td>mod</td>
<td>mild</td>
</tr>
</tbody>
</table>

Subcutaneous Fat Loss 3 areas:
orbital fat pad, triceps, and chest/lower ribs

Subcutaneous Fat Loss
- **Orbital fat pad**
  - Hollow, hollow...
  - Prominent brow bone
- **Triceps**
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
- **Chest/lower ribs**
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:
  - Well-nourished:

Figure 5. Fat pads and temporal muscle commonly used for inspection and palpation during a nutrition-focused physical assessment.

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Muscle Mass Loss 2 areas:
- Temple, interosseous muscle

Bilateral Muscle Wasting
- Temple
  - Observe patient straight on and from either side
  - Look for prominence of brow bone - scooping or hollowing
  - Scooping or hollowing at the temple indicates wasting of temporalis muscle
  - Mild wasting: slight depression of temporalis muscle
  - Severe wasting: hollowing, scooping depression

Subcutaneous Fat Loss (Cont’d)
- Triceps (Triceps brachii)
  - Area on arm most identified with fat loss
- Pinch skin (Paedoflies) between thumb and forefinger over the back of the upper arm over the triceps muscle
- Well-nourished: Arm fat tissue between fold of skin
- Mild-to-moderate fat loss: Finger clinical touch, severe atrophy to pinch
- Severe fat loss: Very thin layer of skin between folds or finger touching

Figure 3. Muscles of the hands (dorsal and palmar) used to inspect and palpate muscle loss during a nutrition-focused physical assessment: (a) dorsal interossei muscles, (b) palmar interossei muscles, and (c) thenar eminence.

Recommendation since 2009:
STOP USING ALBUMIN/PREALBUMIN AS MARKERS OF NUTRITION STATUS
How Can We Monitor Response to Nutrition Support?

Postop Albumin/Prealbumin/C-reactive protein

- SCCM/ASPEN 2016: Traditional nutrition assessment tools (albumin, prealbumin, transferrin) are not validated in critical care and should not be used as markers of nutrition status.
- "Albumin, prealbumin, transferrin, and RBP reflect the acute phase response (increases in vascular permeability and reprioritization of hepatic protein synthesis) and do not accurately represent nutrition status in the ICU setting. … serum albumin concentrations would not be expected to change through the course of management until the stress metabolism abates. Thus, serum protein concentrations have no use postoperatively to measure adequacy of nutrition therapy”.
- North American Surgical Nutrition Summit, 2013:
  - "Hypoalbuminemia is a valid prognosticator of preop risk, correlating significantly with increased LOS, infection, and mortality. However, it should not be followed over time in hospitalized patients. Use of any marker (albumin, prealbumin, or transferrin) for nutrition status is controversial, since they represent 'negative acute phase proteins' levels altered by any stress, injury, infection, organ failure, or acute phase response. Such proteins are poor indicators of actual nutrition state.”

Postop: do we care about Albumin/Prealbumin? NO!!

- Little value in assessment of nutritional status in critical illness/infection/postop due to:
  - Increased transcapillary escape of albumin into interstitial/intercellular fluid
  - Decreased synthesis with critical illness/surgery when positive acute phase production increases

Vitamin Deficiencies – what will you really see??!!

The micronutrient deficiency seen in some long-term Metformin patients
What Disease States and Patients May Require Vitamin B12 Supplements?

- IBD resections or ileal involvement
- Pernicious anemia
- Atrophic gastritis
- Total/partial gastrectomy
- Gastric Bypass, Sleeve Gastrectomy
- Vegans

Lack of Intrinsic Factor

Micronutrient Supplements Required by Bariatric Surgery Patients

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Supplements Required</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastric Bypass (GBP) (5 sup)</td>
<td>- chewable MVI b.i.d.</td>
</tr>
<tr>
<td></td>
<td>- chewable iron 10mg b.i.d.</td>
</tr>
<tr>
<td></td>
<td>- chewable Calcium Citrate 800mg b.i.d. with 5000 units liquid Vit D daily</td>
</tr>
<tr>
<td></td>
<td>- B12 1000mcg oral 3x/week or IM 1000mcg/month</td>
</tr>
<tr>
<td>Lap Adjustable Gastric Band (LAGB) (5 sup)</td>
<td>- chewable MVI b.i.d.</td>
</tr>
<tr>
<td></td>
<td>- chewable iron 10mg b.i.d.</td>
</tr>
<tr>
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<td>Sleeve Gastrectomy (5 sup)</td>
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The micronutrient deficiency with neurologic (peripheral neuropathy, confusion) and cardiovascular (tachycardia, cardiomegaly, CHF) implications.
**Answer: What is Thiamine?**

- **Dry Beriberi** → peripheral neuropathy: symmetric impairment of sensory, motor, and reflex functions

- **Wet Beriberi** → mental confusion, muscular atrophy, edema, tachycardia, cardiomegaly, CHF, + peripheral neuropathy

- rapid improvement win 24 hours after Rx, however peripheral neuropathy may take several months to recover

- What is the most frequent cause of Thiamine deficiency in Western countries?

**What other diseases/patients require thiamine supplementation?**

- Alcoholism
- GBP c/b chronic vomiting
- High-dose lasix (> 80mg/day)
- Malnutrition/refeeding syndrome

**B1/Thiamine Deficiency spectrum—Wernicke's Encephalopathy (WE)**

- **At risk:** alcoholics, severe malnutrition, malabsorption, thiamine-free TPN, high-dose diuretics

- **Signs/symptoms:**
  - ophthalmoplegia, nystagmus, ataxia, confusion and markedly deranged mental function

- **Rx:** IV Thiamine 500mg tid x 2-3 days; 250mg/day thereafter

- **Recovery:** If treated early, recovery is rapid and complete. If untreated → Korsakoff's Psychosis/Syndrome (a continuum of WE)

**B1/Thiamine Deficiency – Korsakoff’s Psychosis**

- **Major Symptoms:**
  1. amnesia
  2. confabulation – invented memories due to memory gaps/blackouts
  3. limited conversation
  4. lack of insight
  5. apathy

- **Etiology of Sx:** thiamine deficiency → damage to thalamus and hypothalamus; cerebral atrophy

- **Rx:** IV or IM Thiamine. If Rx successful, improvement will be seen win 2 years. Only 20% of cases are reversible.

**Refeeding Syndrome – Definition and Risk Identification**

- Metabolic and physiologic complications seen in severely malnourished patients when aggressively fed (oral, TF, TPN)
- caused by intracellular shifts of Mg/K/Phos and vitamin deficiencies

- **Identifying At-Risk Patients – NICE Criteria**
  - **Patient has ≥ 1 of the following:**
    1. BMI < 16
    2. unintentional weight loss > 15% w/in past 3-6 months
    3. minimal nutritional intake > 10 days
    4. hypophosphatemia, hypokalemia, hypomagnesemia prior to feeding

  - **Patient has ≥ 2 of the following:**
    1. BMI < 18.5
    2. unintentional weight loss > 10% w/in past 3-6 months
    3. minimal nutritional intake for > 5 days
    4. history of alcohol abuse, chemotherapy, chronic diuretics

**Pathophysiology of Refeeding**

- **Change from fat catabolism → CHO metabolism** → increased insulin production → intracellular uptake of glucose, Phos, Mg, K+ → low serum levels Phos/Mg/K+

- **sudden increase in CHO** → decreased sodium and water excretion → expanded ECF compartment, fluid overload → pulmonary edema; “refeeding edema”

- **CHO metabolism/anabolism increases use of thiamine** (cofactor in enzyme systems)

- **Susceptible timeframe:** 1st 3-7 days after aggressive nutrition
Pathophysiology of Refeeding Syndrome

- **Thiamine Functions**: cofactor in CHO metabolism (glycolysis); in deficiency state: 1) pyruvate converted to lactate instead of acetyl CoA → lactic acidosis and death due to wet beriberi in patients receiving thiamine-free TPN

- **Phosphorus Functions**: required for ATP production, cofactor in enzyme systems. Lack of RBC phosphorus → hemolysis, anemia, inadequate tissue oxygenation → hyperventilation
  - **Severe hypophosphatemia (<1.5mg/dl)**: 1) neuromuscular - confusion, seizures, coma; weakness, rhabdomyolysis; 2) cardiac - decreased MAP; 3) respiratory - hypoxia, impaired diaphragmatic contractility

Management Guidelines – IV Phosphate Replacement **

- **Mild hypophosphatemia, asymptomatic**
  - 2.3-2.7mg/dl 0.08-0.16mmol/kg
- **Moderate hypophosphatemia, asymptomatic**
  - 1.5-2.2mg/dl 0.16-0.32mmol/kg
- **Severe, symptomatic**
  - < 1.5mg/dl 0.32-0.64mmol/kg

  **For normal renal function. Patients with renal insufficiency: < 50% standard dose. Use adjusted BW for BMI > 30 or > 130% IBW.**

Pathophysiology of Refeeding Syndrome

- **Potassium Functions**: cellular metabolism; glycogen and protein synthesis
  - **Severe hypokalemia (< 2.5mEq/L)**: 1) neurologic – paralysis
  2) cardiac – altered myocardial contraction and signal conduction; arrhythmias, cardiac arrest

- **Magnesium Functions**: cofactor in many enzyme systems including ATP production and oxidative phosphorylation
  - **Moderate to severe hypomagnesemia (< 1.0mg/dl)**
    1) cardiac – EKG changes, arrhythmias
    2) neuromuscular – tremor, seizures, coma
    3) hypomagnesemia-induced hypokalemia
    4) hypomagnesemia-induced hypocalcemia

Management Guidelines – IV Potassium Replacement *

- **Serum K+**
  - 2.5-3.4 mEq/L 20-40mEq (10-20mEq/h)**
  - < 2.5 mEq/L or if symptomatic 40-80mEq

  **For normal renal function. Patients with renal insufficiency: < 50% standard dose. Continuous cardiac monitoring and infusion via CVC for infusion rate > 10mEq K+/hr.**

Management Guidelines – IV Magnesium Replacement

- **Mild/moderate hypomagnesemia, asymptomatic**
  - (serum Mg 1-1.5 mg/dl)
    1-4g MagSulfate (8-32mEq magnesium), ≤ 1 mEq/kg*

- **Severe or symptomatic hypomagnesemia**
  - (serum Mg < 1 mg/dl)
    4-8g MagSulfate (32-64mEq magnesium), ≤ 1.5 mEq/kg*

  **For normal renal function. Patients w/ renal insufficiency: < 50% standard dose. Use adjusted BW for BMI ≥ 30 or > 130% IBW.**

The micronutrient deficiency associated with prolonged diarrhea in Crohn’s/Ulcerative Colitis patients
Answer: What is zinc?

- Zinc deficiency → diarrhea, anorexia, dysgeusia
- Active diarrhea: Rx 220mg Zinc Sulfate/day
- What other micronutrient deficiencies are common in Crohn's/Ulcerative Colitis patients?

Potential Micronutrient Deficiencies in IBD

- Folate
- B12 – ileal involvement/resection
- Ca/Vit D – malabsorption, poor calcium intake
- Iron – poor intake, bloody diarrhea (UC)

Summary: Reality of Micronutrient Deficiencies

- Alcoholics: folate 1mg, thiamine 100mg, Vit B6 (50mg or MVI)
- Metformin: Vitamin B12
- IBD: Calcium, Vit D, iron, B12, folate, zinc (active diarrhea)
- Refeeding syndrome – thiamine, folate
- Weight loss surgeries:
  - GBP: MVI, iron bid, Ca, Vit D, B12 oral daily
  - Sleeve Gastrectomy: MVI, iron 1/day, Ca, Vit D, B12 oral 3x/week
  - Lap Band: MVI, Ca, Vit D

Enteral Nutrition/Tube Feeds – When/Why?

- No bowel sounds/flatus/stool required to start TF (SCCM/ASPEN 2016 B3)
  - 'While GI factors should be evaluated when initiating EN, overt signs of contractility should not be required prior to initiation of EN'.
  - Bowel sounds are indicative only of contractility and do not necessarily relate to mucosal integrity, barrier function, or absorptive capacity
- TF w/in 24-48h in critically ill unable to eat (SCCM/ASPEN 2016 B1)
  - Supports functional integrity by maintaining tight junctions b/t intraepithelial cells, stimulating blood flow, and triggering release of trophic agents (CCK, gastrin, bile salts)
  - Maintains structural integrity – villous height
  - Stimulates production of immunocytes composing GALT
  - Contributes to organ mucosal-associated lymphoid tissue (lungs, liver, kidneys)

Does the gut play a role in MSOF? YES!!

- Fed gut produces B/T lymphocytes → lymph nodes → systemic circulation
- Unfed gut/decreased contractility → bacterial overgrowth
- Increased cytokines
- Increased gut permeability
- Macrophage activation
- Lungs, liver, kidneys
Do We Need Bowel Sounds? NO!!

SCCM/ASPEN 2016 In the ICU population, neither the presence or absence of bowel sounds nor evidence of passage of flatus and stool is required for the initiation of enteral feeding.

Bowel sounds only indicative of contractility; don’t relate to integrity of GI mucosa or absorptive capacity

SCCM/ASPEN 2016 In the ICU setting, evidence of bowel motility (resolution of clinical ileus) is not required in order to initiate EN in the ICU.

As long as the patient remains hemodynamically stable, it is safe and appropriate to feed through mild to moderate ileus.

Is TF Contraindicated in Pressor-Dependent Patients? NO!!

Some pressors may increase splanchnic blood flow:

Dopamine (<10mcg/kg/min)
Levophed (<3mcg/min; 0.5-3ml/min)

Intestinal Vasooconstrictive Effects:

Dopamine >10mcg/kg/min
Levophed >4mcg/min
Phenylephrine/Neosynephrine
Vasopressin

Summary: TF Recommendations with Hypotension/Vasopressive Agents

EN when hemodynamically stable (fluid resuscitated, stable pressor doses, MAP > 60 mmHg)

STOP TF if:

sustained MAP < 60
increasing doses pressors
increased vent support (increasing PEEP, FiO2)
signs of GI intolerance (abd distention/pain, increased NGT output if nasoenteric feeds, cessation of stooling, abd Xray/CT → significant small bowel or colon dilation)

Isotonic, fiber-free formula; fiber (preferred bacterial substrate) in setting of decreased gut motility → incr bowel distention, bacterial overgrowth → stretched bowel wall more susceptible to decreased integrity

Gastric Residual Volumes (GRVs)

Gastric residual volumes should NOT be used as part of routine care. If protocol calls for gastric residuals, avoid holding TF for GRVs < 500ml in absence of other signs of intolerance. GRVs do not correlate w/ incidence of PNA, regurgitation, or aspiration. (SCCM/ASPEN 2016 D2a, D2b)

*** DO NOT CHECK GASTRIC RESIDUALS IN JTUBES!!! ***

Flaws in the GRV Rationale

Daily volume of gastric (3000ml) and salivary (1500ml) secretions averages an hourly rate approximately 188ml/hr in a normally-fed adult. Gastric capacity averages 1500-1900ml.

Most GRVs < 150ml and no significant difference in pattern of GRVs in critically ill patients vs. healthy volunteers.

4 RCTs: increasing GRV from 50-150 to 250-500 does not increase the incidence of regurgitation, aspiration, or PNA. (Taylor, Critical Care Medicine 1999; Montego, Intensive Care Medicine 2010; Pinilla, JPEN 2001; McClave, Critical Care Medicine 2005)

What About Diarrhea?

Diarrhea Defined: Frequent watery, loose bowel movements; > 500ml every 8 hours OR > 3 stools/day for ≥2 consecutive days

Questions to ask:
1) does it meet the definition of diarrhea?
2) Cdiff or infectious cause?
3) antibiotic/med-induced diarrhea?

What about Osmolality? NO! NEVER dilute formulas.

Saliva, pancreatic enzymes, bile salts, neutralize pH in first 10-45cm of small bowel

Infused gastrically, formulas achieve isotonicity (250-300mOsm/kg) by the Ligament of Treitz; infused into Ligament of Treitz, formulas achieve isotonicity by the jejunum
**Diarrhea: What about TF Osmolality?**

- Hypertonic TF formulas: 500-800mOsm/kg
- Osmolality of clear liquids (mOsm/kg)
  - ginger ale: 565
  - Lasix: 3940
  - apple juice: 700
  - Acetaminophen: 5400
  - popsicles: 720
  - NaCl solution: 500
  - Jello: 730
  - water ice: 1065
  - MVI elixir: 5700
  - shortbread: 1225
  - Neoglucan: 8300

- if truly malabsorbing TF:
  1. consider if intolerance to FOS (fructo-oligosaccharides)
  2. if Cdiff negative, try anti-diarrheals
  3. banana flakes
  4. consider change to peptide-based or elemental TF

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**Who Needs TPN? 2016 ASPEN/SCCM Guidelines**

- If gut is dysfunctional, for patients previously healthy prior to critical illness with no evidence of protein-calorie malnutrition, use of TPN should be reserved and initiated only after the first 7 days of hospitalization when EN is not feasible. 2011 NEJM PRCCT n=4650; significantly decreased infections and significantly increased likelihood of earlier discharge from ICU and hospital in late-initiation group
- high nutrition risk (NRS > 5) or severely malnourished, start TPN ASAP if TF not feasible; for NRS < 5, hold off on TPN until PODS-7.

Heyland, *JAMA* 1998: fewer overall complications than STD
Braunshweig, *ACJN* 2001: signif lower risk mortality and trend toward lower infection risk

- Malnourished patients (≥ 10% weight loss over 3 months) w/ dysfunctional guts receiving preop TPN (5-7 days) resulted in 10% reduction in postop complications vs. patients receiving no specialized nutrition therapy.

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**Permissive Underfeeding of TPN in surgical ICU patients**

- **1st week ICU:** 80% of estimated calorie requirements or ≤ 20 calories/kg with adequate protein provision; decreases potential for hyperglycemia and insulin resistance.
- Meta-analysis of 5 studies (trauma, GI cancer, pancreatitis, intestinal obstruction, abdominal/chest procedures) resulted in:
  1. 40% decreased infections, decreased vent days, decreased hospital LOS
  2. decreased hospital LOS by 2.49 days vs. patients randomized to full caloric provision

SCCM/ASPEN 2016 H2.

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**Lipid-free TPN 1st week postop**

- **SCCM/ASPEN 2016 H3a.** In the first week of hospitalization in the ICU, when PN is required and EN is not feasible, patients should be given a parenteral formulation without soy-based lipids. If concern for EFAD, maximum 100g lipid/week
- **soy-based lipid-free parenteral nutrition**
  1. significant reduction in infectious morbidity (PNA and cath-related sepsis)
  2. decreased hospital and ICU length of stay
  3. shorter duration of mechanical ventilation
  4. Meta-analysis – significantly decreased infectious complications and hospital LOS, no difference in mortality

**Fish-oil based INFE** – International Nutrition Survey Data
Shorter ICU LOS; trend toward decr vent days
(Crit. Care Med. 2010)
U.S. any INFE content of omega-3:omega-6 = 7:1 (recommndation in critical illness is 2:1)
NEW: SMOLIPID – FDA-approved 8/2016
(30% soybean oil, 20% MCT, 25% olive oil, 15% fish oil)
Calorie overload → hyperinsulinemia which promotes lipoegenesis and inhibition of FA oxidation

Lipid overload: >1g/kg/day → cholestasis due to incr cholesterol, TGs, and phospholipid concentrations in liver. Limit fat to 30% kcals or 1g/kg.

Dextrose overload → excess CHO converted to fat in liver. Hyperglycemia → increased FFA’s to liver. Limit to 4-5 mg/kg/min.
References
