Clinical Nutrition: Dispelling Myths, Embracing Realities

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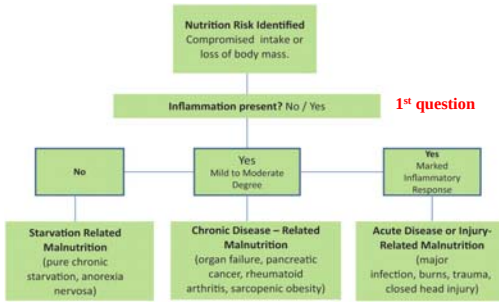
TODAY's TOPICS

- **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
  - increased infection rate
  - immune suppression
  - increased LOS
  - higher readmission rates
  - muscle wasting/functional loss → increased fall risk
  - higher treatment costs
  - increased mortality

Figure 1. Etiology-based malnutrition definitions.



Jane V. White et al. JPEN J Parenter Enteral Nutr 2012;36:275-283

JPEN JOURNAL OF PARENTERAL AND ENTERAL NUTRITION

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

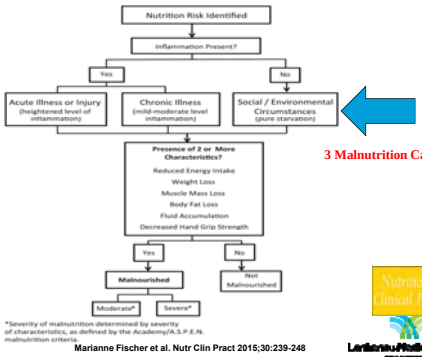
Jensen, G. ASPEN Adult Core Curriculum, 3rd edition, 2012

If inflammation, ? Acute Disease/Injury: Severe Inflammatory Response

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

Jensen G. ASPEN Adult Core Curriculum, 3rd edition, 2012

Figure 1. Schematic demonstrating practical application of the Academy of Nutrition and Dietetics (Academy)/American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) characteristics used to support the diagnosis of etiology-based malnutrition.



3 Malnutrition Categories

Marianne Fischer et al. Nutr Clin Pract 2015;30:239-248

Table 1. General Characteristics for the Diagnosis of Malnutrition. 4 .

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- Weight loss
- Inadequate energy intake
- Loss of muscle mass
- Loss of subcutaneous fat
- Fluid accumulation ← Caution
- Hand grip strength

***Notice NO protein marker!!**

Ainsley Malone, and Cynthia Hamilton Nutr Clin Pract 2013;28:639-650

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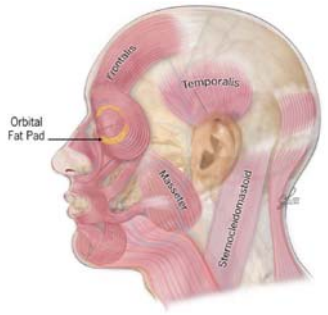
Malnutrition Definitions (need 2 criteria)

	Acute Illness/Injury		Chronic Illness		Social/Environmental	
	Moderate	Severe	Moderate	Severe	Moderate	Severe
5d Intake	<75%	≤ 50%	<75%	≤ 75%	<75%	≤ 50%
Weight Loss						
1 week	1-2%	>2%	1 month 5%	>5%	5%	>5%
1 month	5%	> 5%	3 month 7.5%	>7.5%	7.5%	>7.5%
3 months	7.5%	> 7.5%	6 month 10%	>10%	10%	>10%
			1 year 20%	>20%	20%	>20%
Body Fat	mild	mod	mild	severe	mild	severe
Muscle Loss	mild	mod	mild	severe	mild	severe

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

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



Marianne Fischer et al. Nutr Clin Pract 2015;30:239-248

Subcutaneous Fat Loss (Cont'd)

- Triceps (Triceps brachii)
  - Area on arm most identified with fat loss
  - Pinch skin (**Palpation**) between thumb and forefinger over the back of the upper arm over the tricep muscle
    - **Well-nourished:** Ample fat tissue between fold of skin
    - **Mild-moderate fat loss:** Fingers almost touch, some depth to pinch
    - **Severe fat loss:** Very thin layer of skin between folds or fingers touching

Subcutaneous Fat Loss (Cont'd)

- Chest/Lower Ribs
  - Inspect the mid-axillary line at the costal margin or lower ribs for Loss of fullness or loose skin
    - **Well-nourished:** ample fat tissue; chest wall and ribs should not be visible
    - **Mild-moderate fat loss:** loose skin, somewhat apparent ribs
    - **Severe:** Skin is stretched, prominent well-defined ribs
    - **Picture **

Muscle Mass Loss 2 areas: temple, interosseous muscle

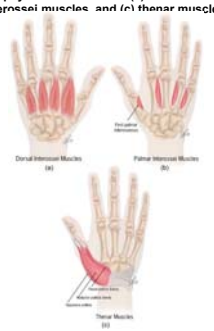
Bilateral Muscle Wasting

- Temple
  - Observe patient straight on and from either side .
  - Look for prominence of brow bone - scooping or hollowing
  - Scooping or hallowing at the temple indicates wasting of temporalis muscle
    - **Well-nourished:** observe well-defined muscle
    - **Mild-mod wasting:** slight depressing of temporalis muscle
    - **Severe wasting:** hollowing, scooping depression

Bilateral Muscle Wasting (Cont'd)

- Interosseous Muscle (dorsal interossei)
  - Observe muscle between thumb and index finger on back of hand (palm down). Have patient press thumb and forefinger back and forth with pressure to inspect muscle
    - **Well-nourished:** May bulge in male and be flat/bulge in female
    - **Mild-Mod:** Slightly depressed or flat
    - **Severe:** flat or depressed area between thumb and forefinger

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 muscles.



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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

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- **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 ***no***t 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" with levels altered by any stress, injury, infection, organ failure, or acute phase response. Such proteins are poor indicators of actual state.".

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

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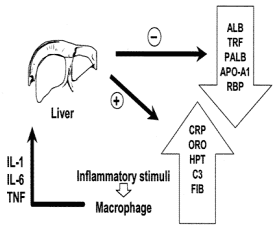
Prealbumin is better ***marker of severity of illness or "nutritional risk"*** than nutritional status/malnutrition or adequacy of nutrition support (except in late illness)



Bauer. Intensive Care Medicine 2000.
Davis. JPEN 2012.
Jenson. JPEN 2009.
Raguso. Curr Opin Clin Nutr Metab Care 2003.

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

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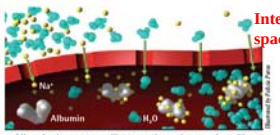
- Little value in assessment of nutritional status in critical illness/infection/postop due to:
- *increased transcapillary escape of albumin into interstitial/intercellular fluid*
- *decreased synthesis* with critical illness/surgery when positive acute phase production increases



▲ Albumin does not readily move through normal capillary pores, while water and smaller biologic structures move freely. Sodium is highly attracted to albumin, and together, they help maintain COP by attracting water into the intravascular space.

Interstitial/intercellular space

Intravascular Space

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Nutrition in Clinical Practice

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

The micronutrient deficiency seen in some long-term Metformin patients

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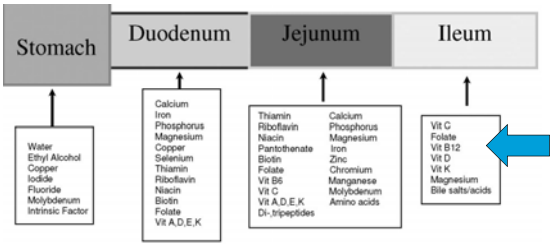
Answer: What is Vitamin B12

What other disease states require B12 supplementation?

GI Tract – Sites of Nutrient Absorption



Source: Shikora SA et al. "Nutrition and Gastrointestinal Complications of Bariatric Surgery" Nutrition in Clinical Practice Feb. 2007;22:29-40.

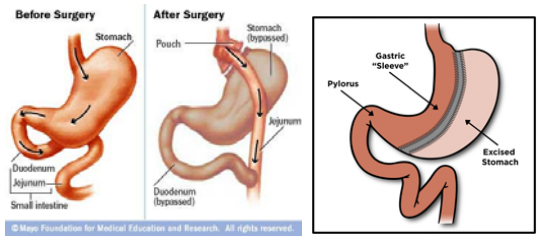
What Disease States and Patients May Require Vitamin B12 Supplements?

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

Lack of Intrinsic Factor

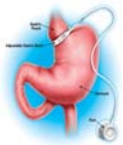
Lap Roux-n-Y

Lap Vertical Sleeve Gastrectomy



Micronutrient Supplements Required by Bariatric Surgery Patients

Gastric Bypass (GBP) (need 5 supplements)	Lap Adjustable Gastric Band (LAGB)	Sleeve Gastrectomy (need 5 supplements)
chewable MVI bid	chewable MVI 1x/day	chewable MVI bid
chewable iron 18mg bid		chewable iron 18mg/day
Chewable Calcium Citrate/D tid (or Calcium Citrate lozenge tid with 5000units liquid Vit D/day)	chewable Ca Citrate/D tid	chewable Ca Citrate/D tid
B12 1000mcg oral/day or IM 1000mcg/month		B12 1000mcg oral 3x/week or IM 1000mcg/month



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 w/in 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?**

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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:** opthalmoplegia, 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 w/in 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?

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Potential Micronutrient Deficiencies in IBD

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

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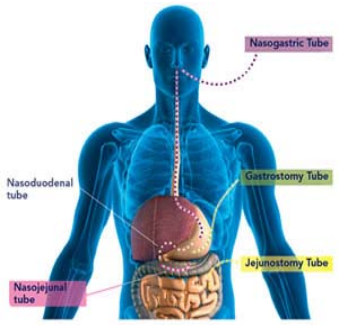
Summary: Reality of Micronutrient Deficiencies

- **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: ICU when/where/why?, bowel sounds, gastric residuals, diarrhea



Enteral Nutrition/Tube Feeds – When/Why?

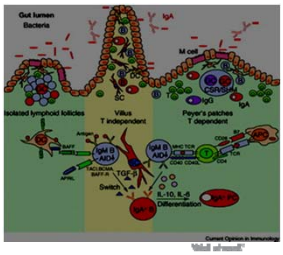
- **No bowel sounds/flatus/stool required to start TF**
  (SCCM/ASPEN 2016 B3)
  ► "While GI factors should be evaluated when initiating EN, overt signs of contractility should not be required prior to initiation of EN".
  ► Bowel sounds are indicative only of contractility and do not necessarily relate 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!

GALT = Gut-associated lymphoid tissue

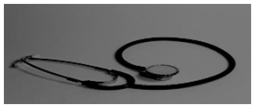
Fed gut: gut produces B/T lymphocytes → lymph nodes → systemic circulation

- **Unfed gut/decr 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 Vasoconstrictive 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

Enteral Nutrition – Where?

- **Gastric feeds for most critically ill patients** (SCCM/ASPEN 2016 B4b)
  ► technically easier, decreases time to EN start
  ► largest multicenter RCT gastric vs SB TF: no difference in clinical outcomes including LOS, mortality, nutrient delivery, incidence of PNA (Davies, Critical Care Medicine 2012)
  ► 13 RCTs do demonstrate lower rates PNA w/ SB TF
- **Divert TF to SB for those at high-risk aspiration or intolerant to gastric feeds** (SCCM/ASPEN B4a, D4a)
- **SURGERY PATIENTS:**

  It is appropriate to attempt to provide postop EN judiciously in a patient above a gut anastomosis, in one with an open abdomen, in a setting of bowel wall edema, in a stable patient on vasopressor therapy, or in one with hypoactive bowel sounds and postop ileus. (SCCM/ASPEN 2016 O4; 2013 N. American Surgical Nutrition Summit) Barlow, Clin Nutr 2011; Collier, JPEN 2007; Khalid, Am J Crit Care 2010; Caddell, Curr Gastroenterol Rep 2011; Dissanaike, J Am Coll Surg 2008)

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, JPEN 2002; 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)		Osmolality of Meds: (mOsm/kg)	
► gingerale	565	► Lasix	3940
► apple juice	700	► Acetaminophen (elixir)	5400
► popsicles	720		
► Jello	735	► MVI elixir	5700
► water ice	1065	► NaPhosphate	7250
► sherbert	1225	► Reglan	8350

- ***if truly malasorbing 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

TPN: Who, When, How Much?

1968: Stanley Dudrick, MD and Doug Wilmore, MD (HUP); 1st PN via central vein (SVC)

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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 POD5-7.

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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TPN How Much?: Surgery Calorie/Protein Guidelines

NONOBESE (BMI < 30)

Calories

25-30 calories/kg ABW
30-35 calories/kg ABW
(high o/p ECF, Burns, TBI, low BMI)

Protein
1.5-2.5g/kg ABW

(upper end for trauma, burns TBI, OA, high o/p ECF, CRRT)

OBESE (BMI ≥ 30)

Calories

BMI 30-50: 11-14/kg ABW
BMI > 50: 22-25/kg IBW
IC x 0.65-0.70

Protein
BMI 30-40: 2g/kg IBW

BMI > 40: 2-2.5g/kg IBW

****Obesity Permissive Underfeeding (SCCM/ASPEN 2016)**
1. avoidance of complications of hyperglycemia
2. decreased CO2 production
3. ability to utilize endogenous fat stores while avoiding protein catabolism
4. Meta-analysis – significantly decreased infectious complications and hospital LOS; no difference in mortality

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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 2009 G2. (Martindale et al, Critical Care Medicine 2009;37(5):1-30).
- North American Summit Surgical Nutrition 2013 (McClave et al, JPEN Sept 2013;37(1):73S-82S)
- 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

(Battistella, J Trauma 1997 -- ? Overfeeding contributed to poor outcomes)

Fish-oil based IVFE – International Nutrition Survey Data

Shorter IUC LOS, trend toward decr vent days
(Cahill, Critical Care Medicine 2010)

- U.S. soy IVFE content of omega-6:omega-3 = 7:1 (recommendation in critical illness is 2:1)

NEW SMOFLIPID – FDA-approved 8/2016
(30% soybean oil, 30% MCT, 25% olive oil, 15% fish oil)

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Summary: TPN Contributors to PNALD (Parenteral Nutrition-Associated Liver Disease)

- Calorie overload → hyperinsulinemia which promotes lipogenesis 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. Hypertriglyceremidia → increased FFA's to liver. Limit to 4-5 mg/kg/min.

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