Gastrointestinal Dimensions: Eating Disorders/Malnutrition

Daniel Jackson, MD
Clinical Professor
Department of Pediatrics
University of Utah School of Medicine
5/16/2019

Disclosures

None except:

- Bias toward our critical responsibility and value of effective sustained nutrition support

- Laced together with compassionate early psycho-behavioral intervention

Session Objective

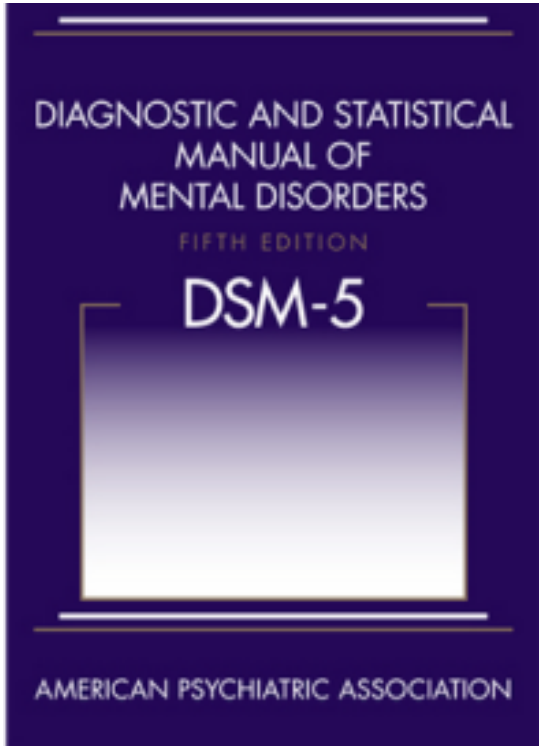
- **By the end of this session, participants will be able to:**
- Understand Malnutrition effects on GI Organ System tissue and function
- Understand Specific Eating Disorder GI comorbidities
- Understand GI Disorders mimicking or complicating Eating Disorders
- Review an Approach to GI dysfunction in Eating Disorders: diagnosis, prevention and treatment
- Know when to consult the GI specialist

Eating Disorders DSM-5

- Anorexia Nervosa (AN)
- Bulimia Nervosa (BN)
- Binge Eating Disorder (BED)
- Pica
- Rumination
- Avoidant/Restrictive Food Intake Disorder (ARFID)

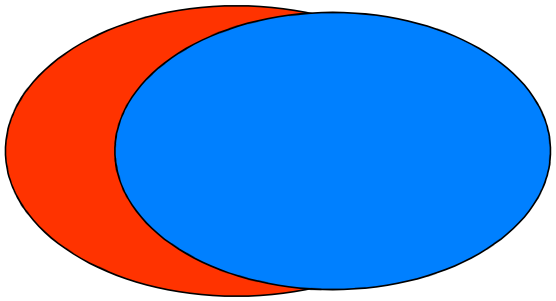
Also:

Many Mixed forms
EDO-NOS
Secondary DOE

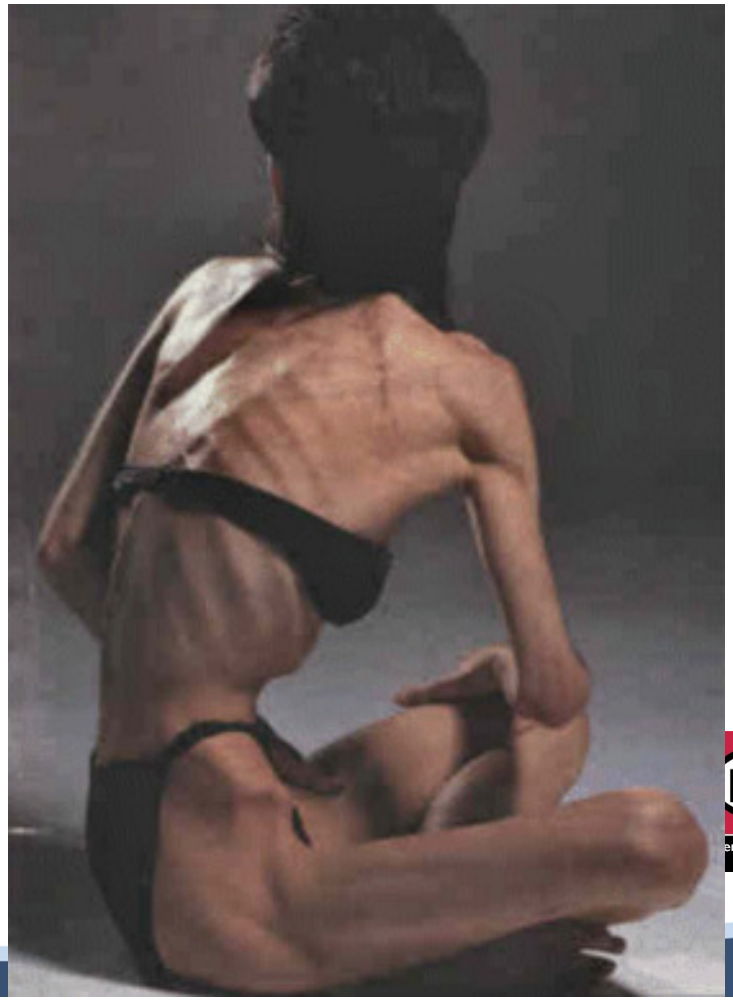


"Eating Disorders are 90% Mental…

The other half is Physical"



Marasmus *Gk: withering*



Effects of Starvation

- Food preoccupations/Food hoarding
- Abnormal taste preferences
- Binge eating
- Depression
- Obsessional behavior
- Apathy/irritability
- Inability to respond to feedback signals

Starvation Effects

Key, A et al (1950) The biology of human starvation.

- Study by Ancel Keys, 1944-5, Minnesota
  - Conscientious objectors to Korean Conflict
  - 32 healthy men of draft age “volunteers”
  - 3 months normal nutrition baseline
  - 6 months @ 50% intake; 25% weight loss
  - Altered Eating Behaviors:
    - Food preoccupation: ritualistic
    - Hoarding behavior
    - Gum chewing
    - Binge eating after experiment ended
    - Anxiety/Obsessive behaviors

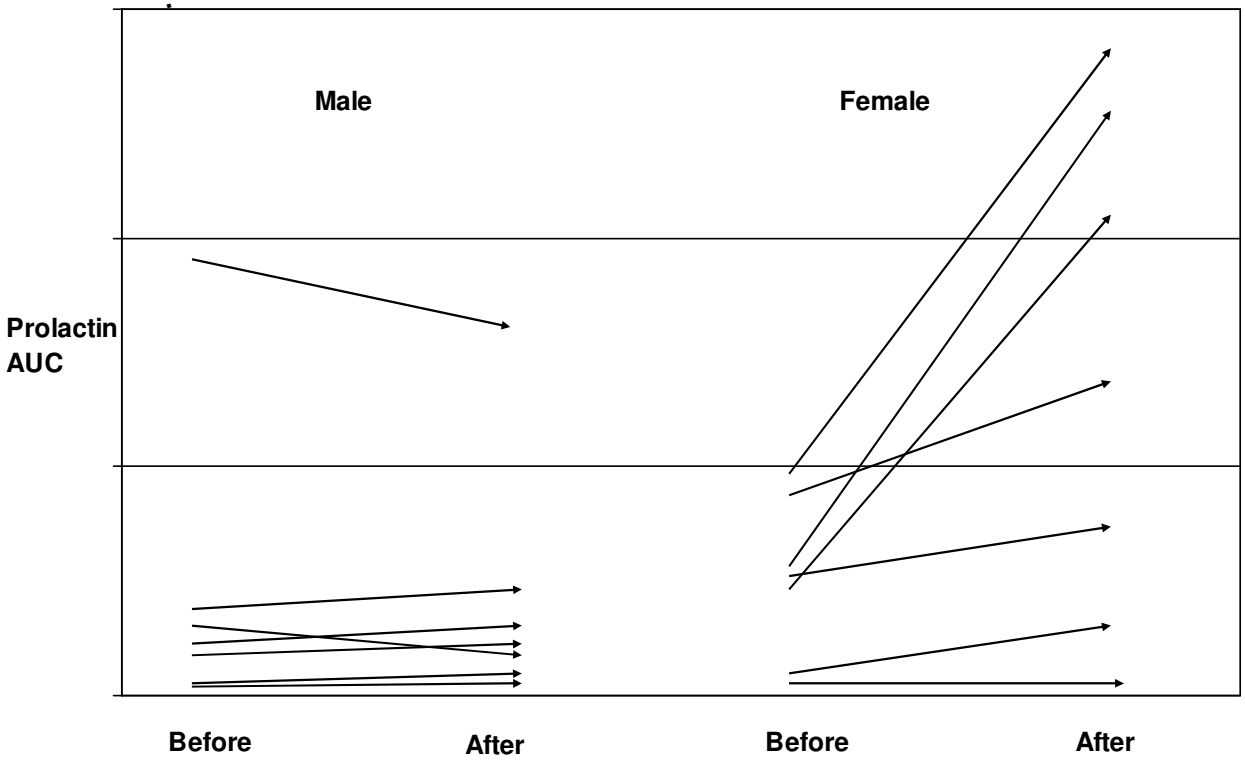
Starvation and the Brain

- Demonstrated atrophy of the starved brain
- Alterations in brain function:
  - Adaptive responses: conserve energy, increase nutrient intake.
  - Maladaptive responses:
  - Alterations in neurochemistry
  - Enhanced behaviors/traits
- Acute vs. Chronic undernutrition

Sustained Hypocaloric Intake

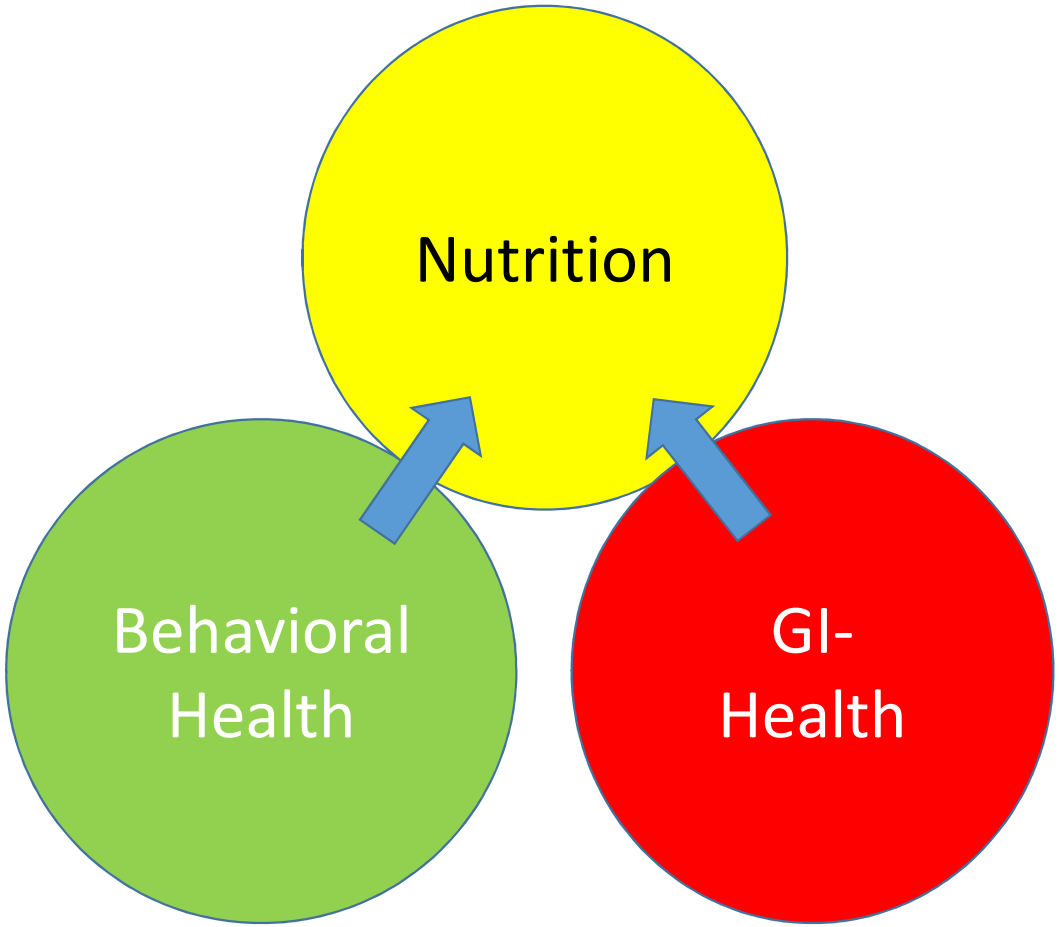
- 3 week hypocaloric (1000 kcal) diet
- 6 men and 6 women lost 6% and 4.5 % body weight
- L-Tryptophan infusion at 0 and 3 weeks
- TRH injection after each LTP infusion
- Prolactin measured after infusions,
  - Bio-marker for 5HT receptor status
  - Dramatic rise in women, not men at 3 weeks
- Consider upregulated 5HT receptors in dietary restriction in women
  - 5 or more 5HT receptors are satiety receptors.
  - Dieting reduces synaptic 5-HT:
    - Short term effect of increased appetite
    - Long term effect of upregulated receptors → increased satiety response
    - Inability to increase intake despite appetite

- Goodwin GM et al. Psycholog Med 1987; 17:839-42


Male
Female
Prolactin
AUC
Before
After
Before
After

Food-Restriction Induced Hyperactivity

- Rats restricted to chow for 90 mins./d lose weight and stabilize.
- Access to running wheel → compulsive running → lethal weight loss
- A model: OCD, hyperactivity and/or AN ?
  - Motor activity → increases 5-HIAA, catecholamines
- Fluoxetine attenuated the FRIH:
  - serotonin receptor mediated
- No response to imipramine
    - Altemus M et al. Pharm Biochem Behav 1996;53:123-31


Nutrition
Behavioral Health
GI-Health

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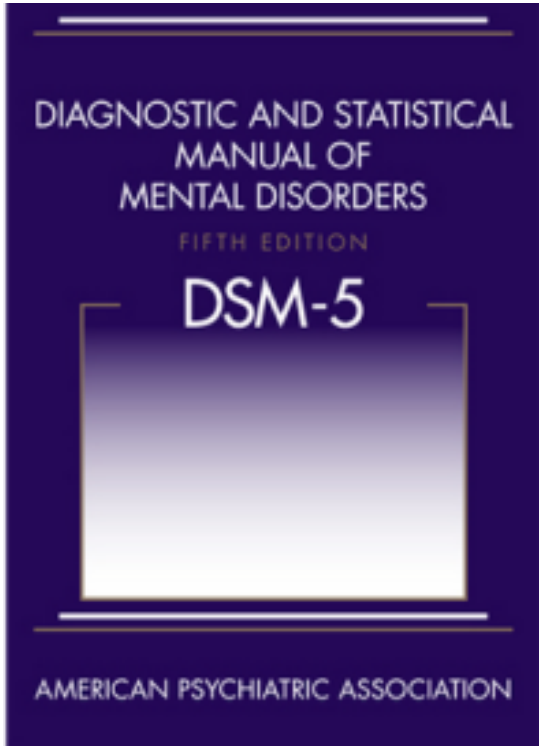

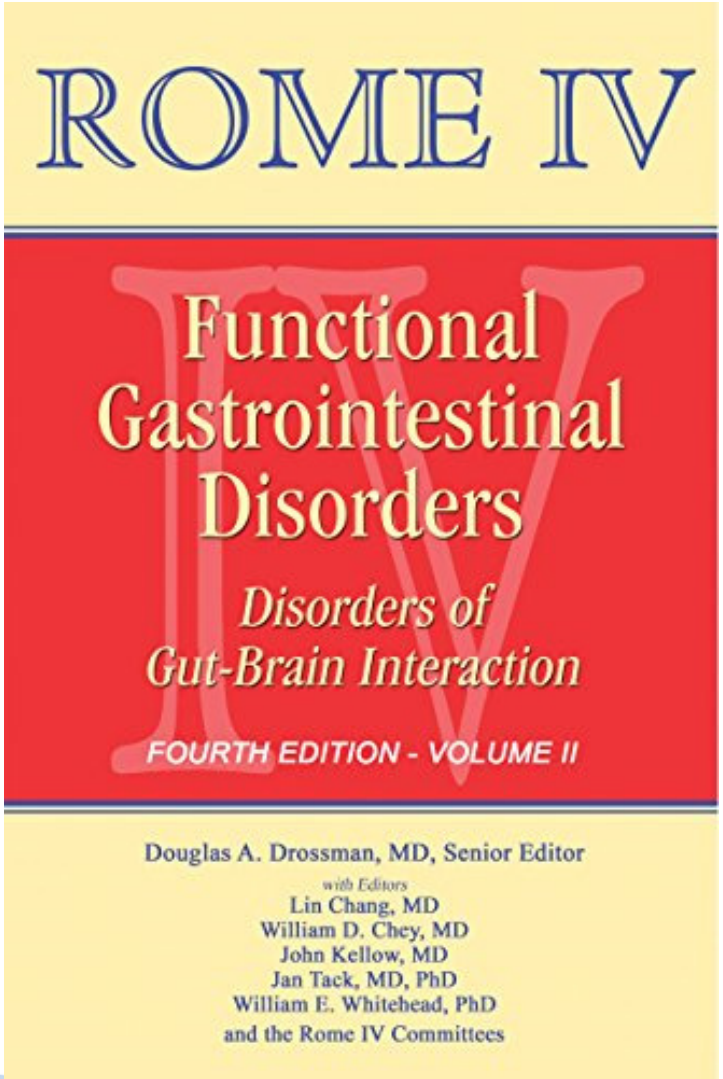

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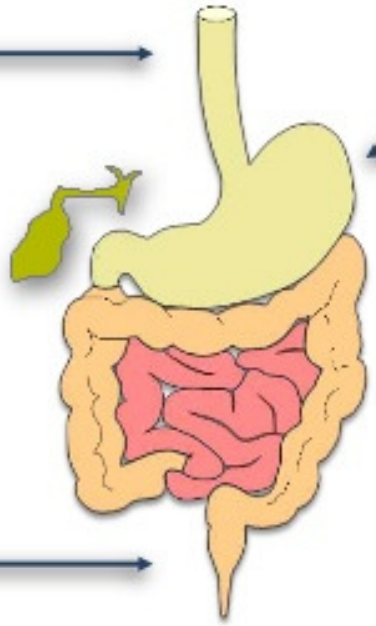
Functional Gastro-Intestinal Disorders (FGID)

(Symptoms arising from the GI tract not explained by any detectable disease)

Esophagus
- Functional heartburn
- Gastro-Esophageal Reflux
- Functional Dysphagia
- Globus

Gastro-Duodenal
- Functional dyspepsia
- Belching Disorders
- Nausea and Vomiting Disorders

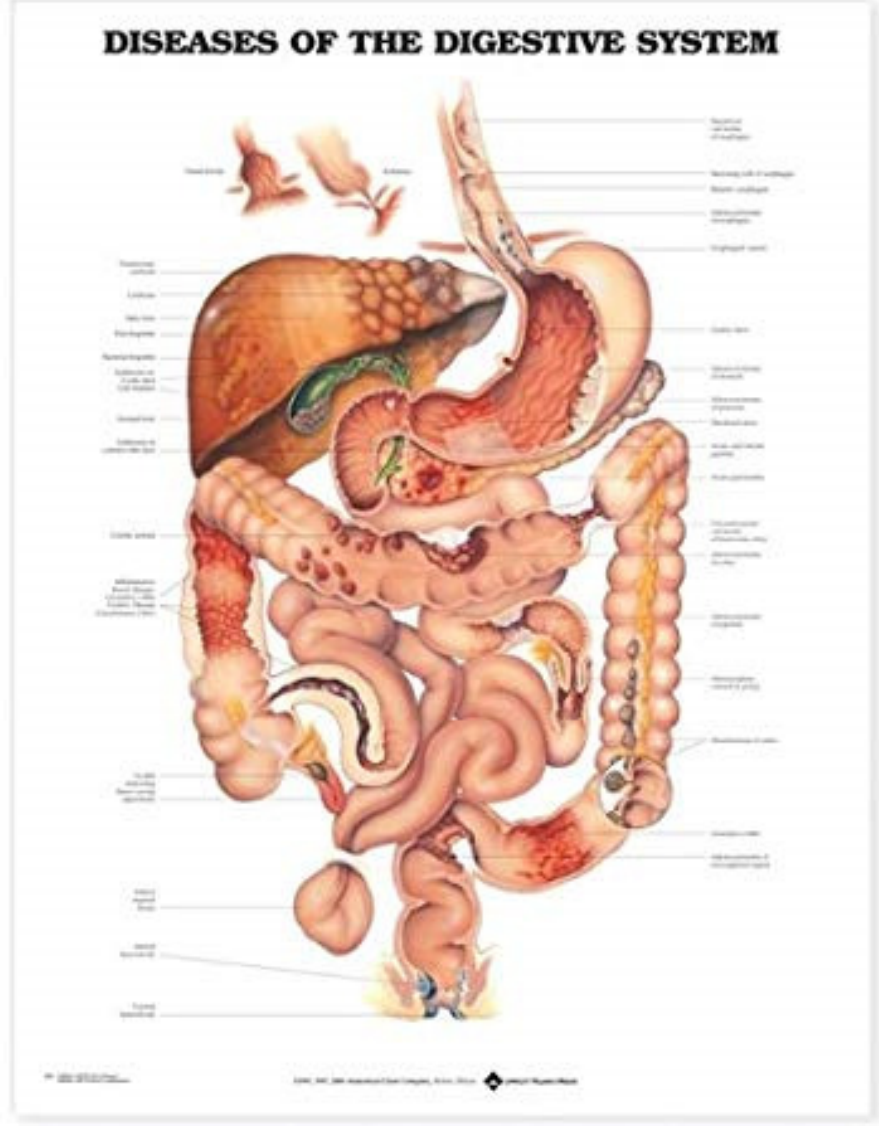


Gallbladder/Sphincter of Oddi
- Functional gallbladder disorder
- Functional SO disorder

Functional Bowel Disoders
- **Irritable Bowel Syndrome**
- **Functional Constipation**
- Functional Bloating
- Functional Diarrhea
- Functional Abdominal Pain

Ano-Recutum
- Functional Incontinence
- Functional Anorectal Pain
- Dischezia

Drossman DA. Gastroenterology 2006;130:1377-90



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GI disease/dysfunction Meets EDO Behavior

- 1. GI disease/dysfunction → EDOB → Malnutrition
  - Celiac, Crohn Disease, Irritable Bowel Syndrome
- 2. GI disease/dysfunction → Malnutrition→ EDOB
  - Cystic Fibrosis, Crohn Disease, Type I DM/gastroparesis
- 3. EDOB → GI disease/dysfunction
  - Constipation-IBS, Oral-Esophageal injury, Gastric Distention/gastroparesis
- 4. EDOB → Malnutrition → GI disease/dysfunction
  - Fatty liver disease; villus atrophy, Sup. Mes. Art. (Duodenal Obstruction) Syn.

GI DDx of "Eating Disorder Patient"

- Inflammatory disease: Celiac disease, IBD (Crohn's or UC)
- Chronic hepatitis, Chronic pancreatitis, Cystic Fibrosis
- Esophagitis- Eosinophilic
- Gastroparesis- idiopathic, post-viral, CGD, Myopathy, Type 1 DM
- Irritable Bowel Syndrome- stool retention

- Other: Addison's, Lymphoma/Leukemia

Screen patients for primary GI conditions that mimic EDO's

Presentations: GI conditions in EDO

- Dysphagia/Pyrosis: **acid reflux** vs eosinophilic esophagitis
- Epigastric pain: Esophagitis, gastritis**, dyspepsia**, nocturnal: PUD, pancreatitis, cholecystitis
- Supraumbilical pain: **Stool retention**, IBD- ileitis/proximal colitis, lymphoma
- Infraumbilical pain: **Constipation**, diverticulosis/itis, IBD- distal colitis
- Back pain: Scapula/shoulder: pancreas/biliary; post- duodenal ulcer, UTI/pyelo/stone; lumbosacral constipation
- Early satiety: Chronic stool retention(cologastric inhibition); gastroparesis (post–viral vs metabolic); small intestinal distention due to gas/distention consequences: “bloating, cramping, urgency”; IBS- irritable bowel syndrome-(diarrhea vs constipation dominant)
- Nausea/Vomiting: gastroparesis, GERD, Hiatal Hernia, Superior Mesenteric Artery syndrome, Stool Retention:ascending/transverse colon; Acute gastric distention
- Diarrhea, gas, distention, pain**: lactose intolerance**(lactase deficiency vs. post-viral); celiac disease, giardiasis/cryptosporidium; HIV, post-viral enteropathy
- Inflammatory: fever, chills, rash, oral/perianal lesions, blood, nocturnal,mucu urgency/frequency of stools vs. tenesmus and “Crohnstipation”

GI tract changes in general undernutrition:

- Motility:
  - Cephalic phase: *dysregulated appetite*
  - Oropharyngeal initiation- *rare*
  - Esophageal peristalsis/sphincter function – *preserved*
  - Gastroparesis – slowed gastric emptying- atrophy-distention
    - Gastric Emptying: *solids delayed*
  - Gastrocolic response: *with gastroparesis- is impaired*
  - Cologastric inhibition: *stool retention/colonic distention*
  - Prolonged intestinal transit
  - Stool Retention/constipation
- Lumenal Digestion:
  - Hypochlorhydria
  - Pancreatic insufficiency
  - Biliary insufficiency
- Mucosal Digestion:
  - Brush border membrane
  - Small bowel bacterial overgrowth
- Mucosal Absorption:
  - Crypt hypoplasia, Villus atrophy
  - Epithelial disruption/dis-integration-increased permeability

Consequence: Reduced Nutrient intake and Assimilation

Most reverse or improve with nutrition

Nutrition/EDO Related Acquired GI Pathology

- Oral:
  - Dental erosions, caries, -enamel loss/polished surface.
  - Gingival periodontitis: rare in children- palatal ulcers –Vitamin C
  - Stomatitis: cheilosis- angular stomatitis: iron, zinc, B2
  - Nutritional deficits:
    - Vit C: gingivitis/bleeding (scurvy)
    - B vitamins(B1-3,6,12): mucosal atrophy, atrophic glossitis, glossodynia(burning tongue)
    - Oral candidiasis(thrush)
  - Sialadenosis: salivary gland(acinar) hyperplasia: parotitis→necrosis
  - Rx: PPI treatment, consider motility agent for gastric emptying, manage stool retention that slows gastric emptying– observe for 2 hours after scheduled meals.

- Esophagus:
  - Pyrosis(heartburn), chest pain, dysphagia complaints prevalent
  - GERD: Acid/Pepsin exposure: esophagitis- metaplasia→dysplasia (Barrett's)
  - Esophageal motility usually normal despite c/o dysphagia
  - Mallory Weiss tears– rare rupture (Boerhaave syndrome) with BN
- Stomach:
  - Gastroparesis vs Functional dyspepsia
    - Impaired fundus accommodation vs antral hypomotility
    - Gastric bezoar
    - Muscle atrophy
    - Autonomic dysfunction
  - Acute gastric distention-→rupture rare

- Small Intestine:
  - Prolonged gastro-cecal transit
  - Superior mesenteric artery syndrome : Rx: Transpyloric (NJ) feeding
  - Absorption – relatively preserved
- Large Intestine:
  - IBS-Constipation– may persist after restored nutrition
  - Anorectal/pelvic floor dysfunction prevalent 42% AN
  - Rectal prolapse[DDx: celiac, cystic fibrosis]
  - Laxative abuse– less risky unless castor oil or anthraquinones (senna and cascara) melanosis coli staining
  - Necrotizing Colitis
  - Rx: PEG 3350(Miralax) bowel prep cleanout→ sufficient prn to maintain daily complete BMs→ link toilet to meal—short walk stimulation.

- Pancreas:
  - Fibrosis in general with Protein-Energy Malnutrition– atrophy
  - Reduced amylase, lipase, trypsin (but normal bicarbonate excretion)
  - Reports of acute pancreatitis – not chronic—
    - Confounders: elevated serum amylase levels may be salivary origin

    Elevated serum lipase is seen in diabetic ketoacidosis– or ketosis.

  Rx: test fecal elastase, stool qualitative fat—consider pancrelipase
- Liver:
  - Transaminase elevation– improves with refeeding
  - NAFLD – hepatic steatosis→steatohepatitis
  - Starvation induced autophagy of hepatocytes and liver failure
  - Rx: Consider Vitamin E 800 IU supplementation for high ALT

Approach:

- Eating Disorder Phenotypes overlap with Chronic GI phenotypes:
  - Much of EDO Behavioral and GI dysfunction will improve with nutrition
- EDO does not logically exclude underlying or associated GI disorder
  - Cause, Consequence or Comorbidity
- Severe chronic malnutrition due to chronic energy deficit and/or somatic disease: can mimic EDO, including secondary psychiatric features of anxiety-OCD/depression and paradoxical anorexia related to disturbance of hypothalamic satiety regulation.
- Nutrition Support is a critical objectively measurable effective intervention related to prognosis.
- Cognitive based and pharmacologic based interventions may depend on restored brain-body nutritional and metabolic status.

Pharmacologic Interventions: Little Data

- Cyproheptadine-anti-histamine/anti-serotonin: Appetite stimulation
  - Also increases gastric accommodation/relaxation/reduce vomiting
- Zinc supplementation: increases NPY and orexin: 1 RCT-increased BMI
- Vitamin B12 , Selenium: if plasma levels low
- PUFA-omega 3 and 6: may help
- Motility: metoclopramide, erythromycin, azithromycin, domperidone
- Acid suppression: PPI
- Laxatives: non-irritant osmotic laxative - polyethylene glycol, lactulose
- Probiotics: Hypothesis

When to Consult GI

- When Symptoms do not respond to Nutrition Support
- When Nutrition support cannot be provided due to GI symptoms
- For red flags of hematemesis, hematochezia, abdominal distention, persistent pain or tenderness, nocturnal pain, odynophagia or dysphagia.
- For positive celiac screen, occult blood, lactoferrin/calprotectin, elevated ESR/CRP in setting of GI dysfunction

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1. Bern EM, Woods, ER and Rodriguez L. Gastrointestinal Manifestations of Eating Disorders. JPGN 2016; 63: e77-e85
2. McClain CJ, Humphries LL, Hill KK, Nickl NJ,. Gastrointestinal and nutritional aspects of eating disorders. J Am Coll Nutr. 1993; 12: 466-74
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4. Quick VM, Byrd-Bredbenner, C and Neumark-Sztainer D. Chronic illness and disordered eating: A discussion and review of the literature. Adv Nutr 2013; 4:277-86
5. Blanchet C et al Medication in AN: A Multidisciplinary Overview of Meta-Analyses and Systematic Reviews J Clin Med 2019; 8,278 doi: 10.3390/jcm8010278

Hub Team Contacts

Liz Joy, MD, MPH
Medical Director | Clinical Lead
Liz.Joy@imail.org | 801-442-3721

Janae Decker, MPH, RD
Adult Dietitian
Janae.Decker@imail.org | 801-507-6084

Steve Varechok, LCSW, CSCS
Clinical Social Worker
svarechok@gmail.com| 801-277-8100

Sadie Wilde, MA
Clinic Supervisor
Sadie.Wilde@imail.org | 385-282-2722

Amelia Davidson, MS, RDN, CSP, CD
Pediatric Dietitian
Amelia.Davidson@imail.org | 801-662-5320

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NEXT SESSION:

June 20, 2019
12:00-1:30 p.m.

Outpatient Nutrition Care for Patients with Eating Disorders

Amelia Davidson, MS, RDN, CSP, CD
Pediatric Dietitian
Intermountain Healthcare

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