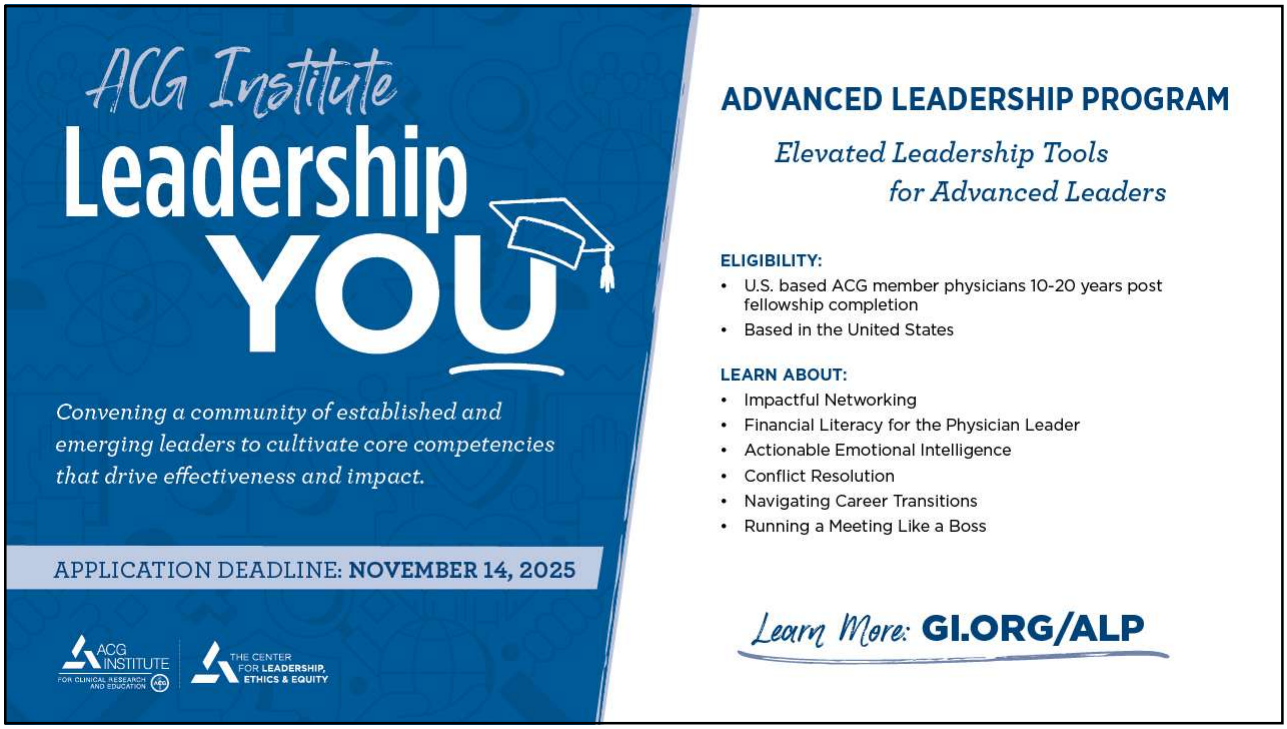
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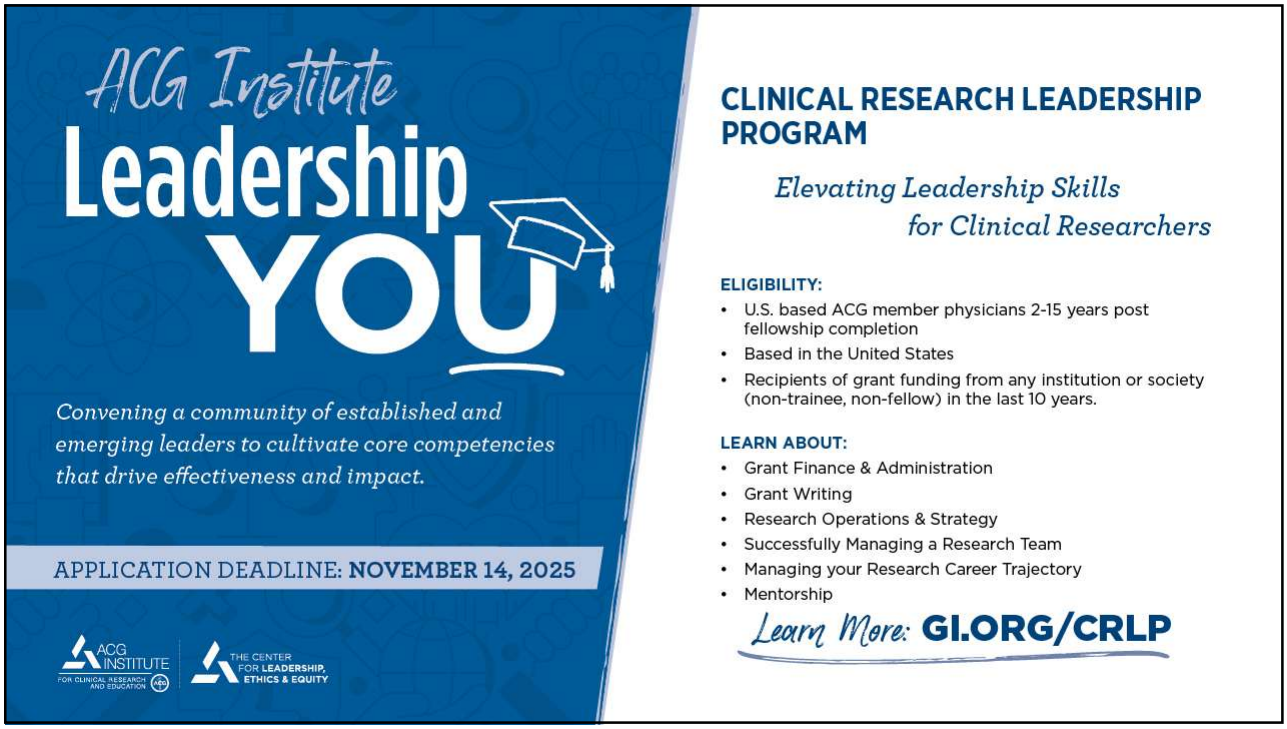
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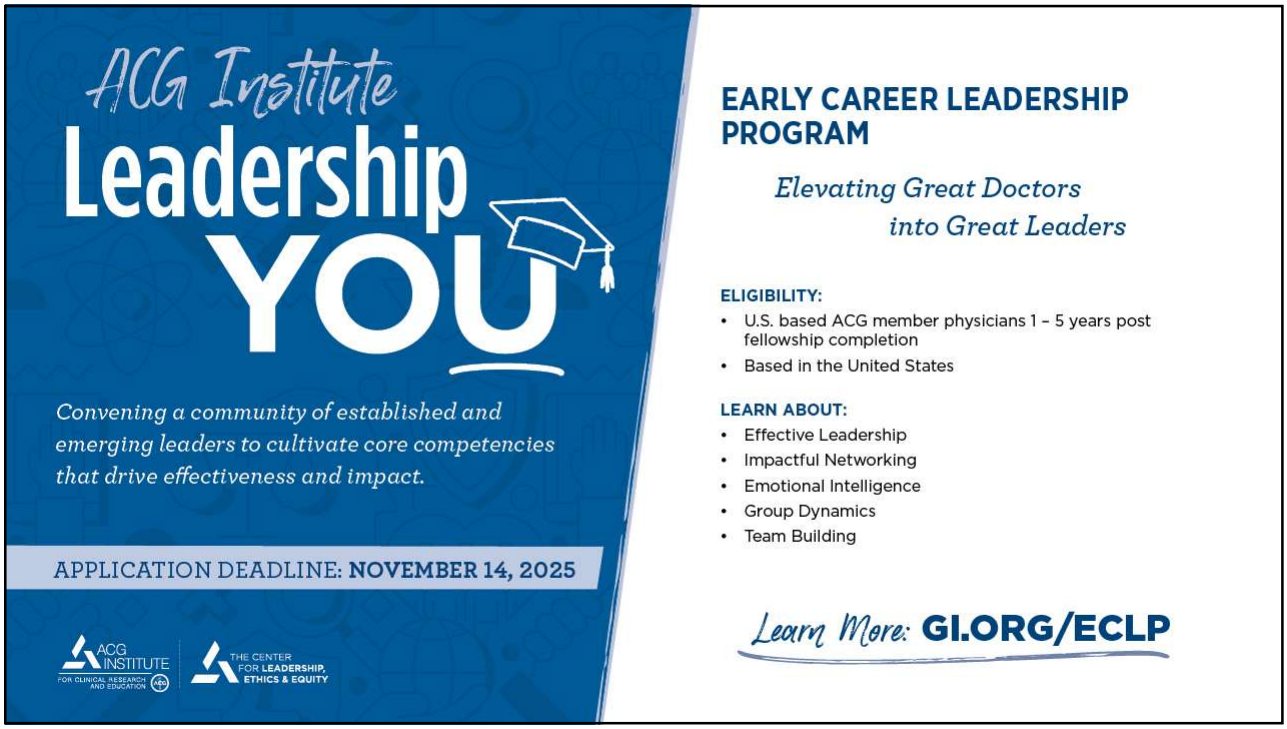
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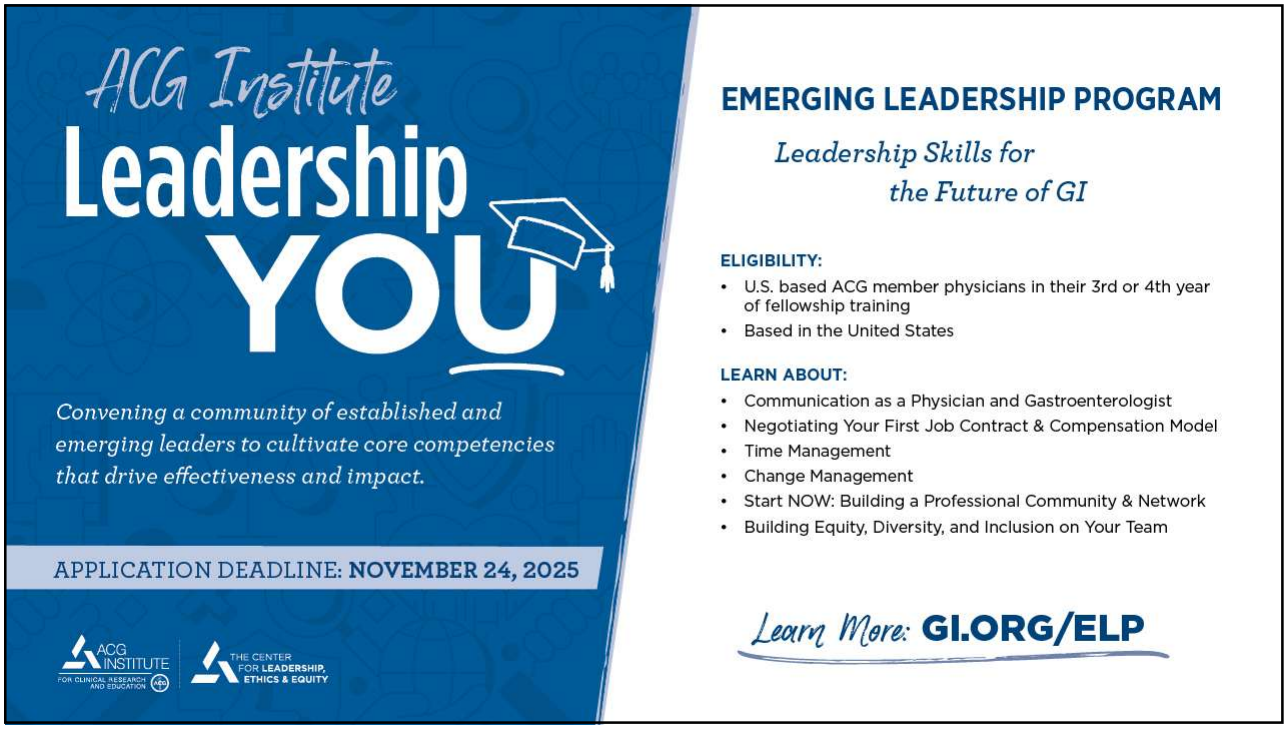
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NOTE: Must complete Prequalification Form (bit.ly/33guW6k) by November 3rd

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Moderator:
Omar Jamil, MD

All attendees will be muted and will remain in "Listen Only Mode"

Type your questions here so that the moderator can see them.
Not all questions will be answered but we will get to as many as possible.

A handout with the slides and room to take notes can be downloaded from your control panel.

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ACG Virtual Grand Rounds

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Week 47 – Thursday, November 20, 2025
Vaccine Update for Gastroenterologist- IBD and Beyond
Faculty: Freddy Caldera, DO, MS, PhD, FACG
Moderator: Francis A. Farraye, MD, MSc, MACG
At Noon and 8pm Eastern

*Week 48 - No VGR in observance of the Thanksgiving Holiday

Week 49 – Thursday, December 4, 2025
ACG's 2025 Practice Management Summit – Course Highlights
Faculty: Andy Tau, MD
Moderator: Kunjali Padhya, MD, FACG
At Noon and 8pm Eastern

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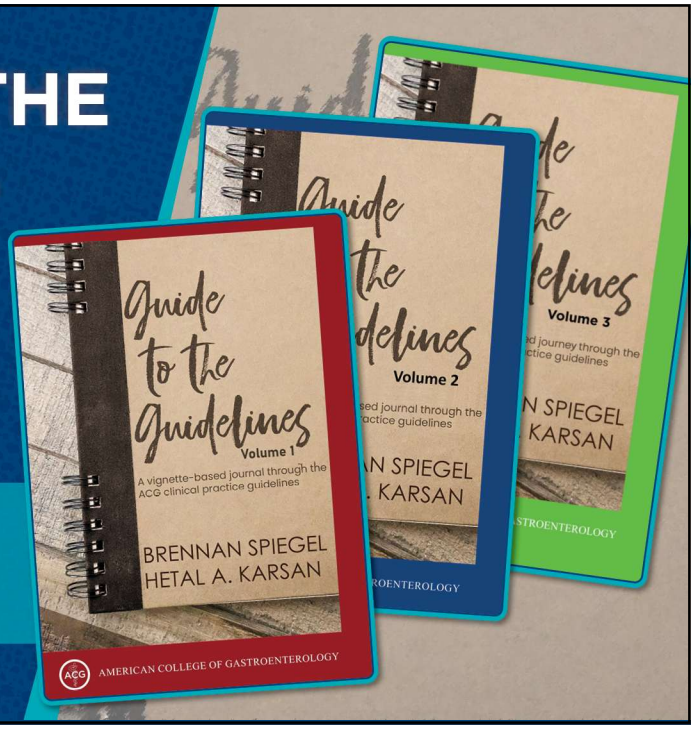

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2025-26 ACG SBS Series

- Small Bowel Nutrient and Fluid Absorption: Key Concepts to Manage Short Bowel Syndrome
- Inpatient Management of the Newly Diagnosed Short Bowel Patient: Consult to Discharge
- Short Bowel Syndrome/Intestinal Failure: Recognition, Complications, and Basic Management
- Short Bowel Syndrome: Maximizing Management to Convert Intestinal Failure to Intestinal Insufficiency
- Cases of Non-Short Bowel/Intestinal Failure: Pearls for Recognition and Management

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Disclosures

George Ou, MD

Baxter: Consult; Knight Therapeutics & Celltrion: Consultant.

Omar Jamil, MD

No relevant financial relationships with ineligible companies.

**All of the relevant financial relationships listed for these individuals have been mitigated*

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Cases of Non-Short Bowel/Intestinal Failure: Pearls for Recognition and Management

George Ou, MD, MPH
St. Paul's Hospital
University of British Columbia

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Objectives

- Recognize non-short bowel causes of intestinal failure
- Understand basic principles in the diagnosis and nutrition management of non-short bowel causes of intestinal failure.

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Case #1

- 28yo M with hx multiple abdo surgeries for SBOs post previous trauma → **chronic abdominal pain and vomiting**. 39kg (86lbs); BMI 15.7.
- GI anatomy = intact
- PMH: Chronic alternating diarrhea/constipation, hx Gr D erosive esophagitis, intellectual disability
- Meds: metoclopramide, pantoprazole, PEG3350, haloperidol, ondansetron, hyoscine
- P/E: **++temporal wasting, deltoid squaring, paucity of adipose tissue**, **edema**. **++abdo distension with tenderness**. Absent bowel sounds.

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Case #2

- 37yo F with **metastatic ovarian Ca** and **progressive disease with malignant SBO**. No further oncologic treatment planned. 41kg (90lbs); BMI 16.
- Anatomy: SBO + venting G tube.
- PMH: No other health concerns
- Meds: fentanyl patch, hydromorphone, haloperidol, octreotide, dexamethasone, ondansetron prn
- P/E: **Significant muscle wasting and low adiposity**. Abdomen distended with 20Fr venting G tube.

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Case #3

- 20yo F w/ chronic N/V, pain, diarrhea/constipation → **worsening oral intolerance** post cholecystectomy
- PMH: POTS, hEDS, impaired gastric emptying, Migraines, Complex regional pain syndrome, PTSD, somatization, lap chole, urinary retention
- Investigations: EGD, Colonoscopy, MRE, CTA, SBFT, serum tryptase levels all within normal
- Meds tried: Prucalopride, metoclopramide, domperidone, erythromycin, prochlorperazine, ondansetron, diMENhydrinate, CBD oil, nabilone, pantoprazole, esomeprazole, mirtazapine, bupropion, duloxetine, sumatriptan, topiramate, pregabalin, senna, PEG3350, linaclotide, plecanatide, tenapanor, ivrabidine, midodrine
- P/E: No clear signs of malnutrition with **good muscle tone and adipose tissue. Stable weight.**

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Intestinal failure

- When the intestines *fail*
- Many past definitions

Pironi et al. Clinical Nutrition 2015

Table 2
Main original definitions and classifications of Intestinal Failure reported in the literature prior to March 15th 2014, in order of publication date. Bold characters indicate the original contribution of each paper.

Author, date (ref)	Definition and classification of intestinal failure
Fleming CR and Remington M. 1981 [1]	**A reduction in the functioning gut mass below the minimum amount necessary for adequate digestion and absorption of food**
Irving M. 1995 [10]	The **spectrum of intestinal failure** covers a wide range of diseases but essentially they can be placed in **four major categories:** short bowel syndrome, motility disorders of the bowel (chronic pseudoobstruction), small bowel parenchymal disease, intestinal fistula
Irving M. 2000 [11]	Intestinal failure can be **complete or partial**, the former typically following total small bowel enterectomy, whilst the latter is seen following partial resection. The condition can be **acute and temporary**, as seen with recoverable motility disorders such as ileus and obstruction, or chronic and permanent. Although a wide spectrum of conditions can be associated with IF, four **major underlying causes** can be identified. These are: (i) the short bowel syndrome; (ii) total parenchymal bowel disease (e.g. Crohn's disease); (iii) motility disorders, such as visceral myopathy and chronic intestinal obstruction; and (iv) small bowel fistulation causing premature loss of enteric content. **The principal resulting nutritional disorders are starvation and dehydration**, but loss of body mass is frequently made worse by catabolism from associated sepsis. **Treatment** is complicated, but has at its core the provision of **nutritional support, principally through the intravenous route**. **Resolution of IF can occur spontaneously** by the process of intestinal adaptation.
Jeppesen PB and Mortensen PB. 2000 [12]	**Intestinal failure** may be defined by the **minimum energy and wet weight absorption required to avoid home parenteral nutrition** Patients with **intestinal insufficiency** who maintained intestinal autonomy and did not depend on parenteral supplements Involuntary ingestion below the minimal amount necessary to maintain nutrient and fluid balance, frequently termed **oral failure**, is seen in patients with pseudoobstruction and dysmotility syndromes.
Nightingale J. 2001 [13]	Intestinal failure occurs 'when there is **reduced intestinal absorption so that macronutrient and/or water and electrolyte supplements** are needed to maintain health and/or growth'.
Shaffer J. 2002 [14]	**A novel classification of intestinal failure** was recently devised to reflect this: **Type I** intestinal failure is classified as self-limiting intestinal failure as occurs following abdominal surgery; **Type II** is intestinal failure in severely ill patients with major resections of the bowel and septic, metabolic and nutritional complications requiring multidisciplinary intervention with metabolic and nutritional support to permit recovery; **Type III** is chronic intestinal failure requiring long-term nutritional support.
Buchman AL et al., 2003 [15]	It has been suggested that intestinal failure is better defined in terms of fecal energy loss rather than residual bowel length. However, fecal energy loss is a function of both energy intake and energy absorption. **Patients who are unable to increase their oral intake sufficiently or are unable to absorb sufficient energy despite significantly increased intake**, are defined as patients with intestinal failure and require parenteral nutrition support.
Ding LA and Li jS. 2004 [16]	**Staging** of intestinal failure: Acute intestinal failure, Chronic intestinal failure **Grading** of intestinal failure: severe, moderate, mild
Goulet O et al., 2004 [17]	Intestinal failure can be defined as the reduction of functional gut mass below the minimum needed for **digestion** and absorption of nutrient and fluids required for maintenance in adults or growth in children. It has been suggested that IF is better defined in terms of fecal energy loss rather than residual bowel length in patients with short bowel syndrome. Another approach is **to define the degree of IF according to the amount of PN required** for maintenance in adults and growth in children
Kocoshis SA, 2004 [18]	Although intestinal failure can be defined by excessive fecal energy loss, a more widely accepted definition is "**the inability of the gastrointestinal tract to sustain life autonomously**".
Jeejeebhoy KN. 2005 [19]	Gastrointestinal function is inadequate to maintain the nutrition and hydration of the individual without supplements given **orally** or intravenously
O'Keefe SJD. 2006 [3]	Intestinal failure '**results from obstruction, dysmotility, surgical resection, congenital defect or disease–associated loss of absorption and is characterized by the inability** to maintain protein-energy, fluid, electrolyte **or micronutrient balance**'
Lal S. (2006) [20]	Causes of intestinal failure are varied, with self-limiting or '**Type 1**' intestinal failure occurring relatively commonly following abdominal surgery, necessitating short-term fluid or nutritional support. The rarer, '**Type 2**' intestinal failure, is associated with septic, metabolic and complex nutritional complications, usually following surgical resection in

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Intestinal failure

- Most accepted / comprehensive definition of Intestinal Failure (ESPEN 2015)

  Reduced gut function below the minimum necessary for the ***absorption*** of macronutrients and/or water and electrolytes

  Such that ***intravenous*** supplementation is required to ***maintain health and/or growth***

Pironi et al. Clinical Nutrition 2015

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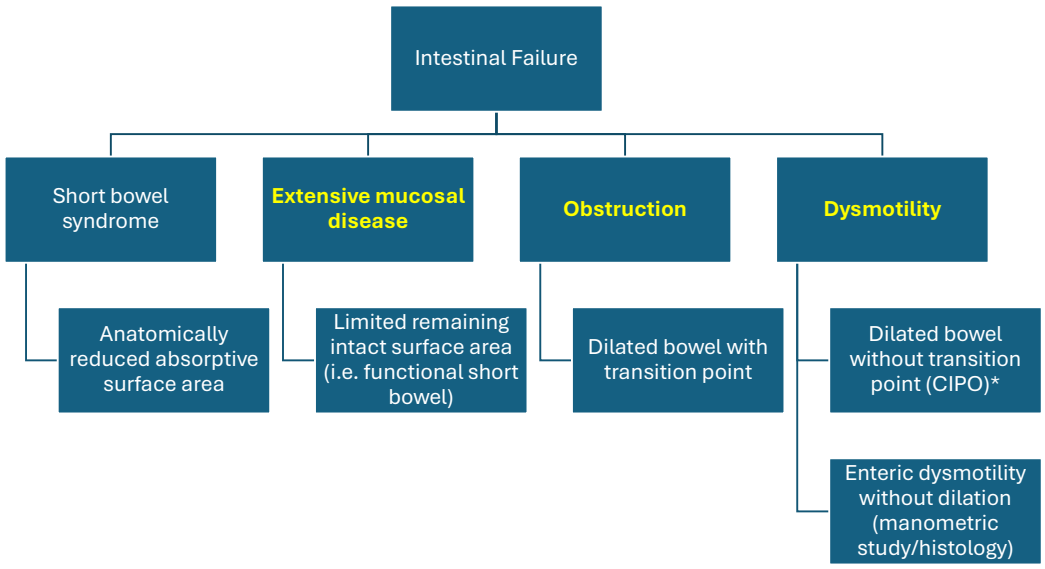

CIPO: Chronic Intestinal Pseuo-Obstruction

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Shift in Home PN indications

Table 1. Demography and Indication for HPN in Patients Entering the Registry in 2005–2008 and 2011–2014.[a]

Characteristic	2005–2008 (n = 182)	2011–2014 (n = 187)	*P* Value
Age, y	52 (42, 62)[b]	57 (46.5, 63.5)[b]	.047
Sex			
Female	107 (58.8)	114 (61)	
Male	75 (41.2)	73 (39)	
Indication for HPN[c]	168	169	
Short bowel syndrome	110 (65.5)	54 (32)	<.001
Mucosal dysfunction	15 (8.9)	4 (2.4)	.010
Motility disorder	29 (17.3)	17 (10.1)	.058
Tumor/cancer	28 (16.7)	64 (37.9)	<.001
Surgical complication	28 (16.7)	51 (30.0)	.005
Pancreatic disorders	1 (0.6)	2 (1.2)	1.000

Hortencio et al. JPEN 2017

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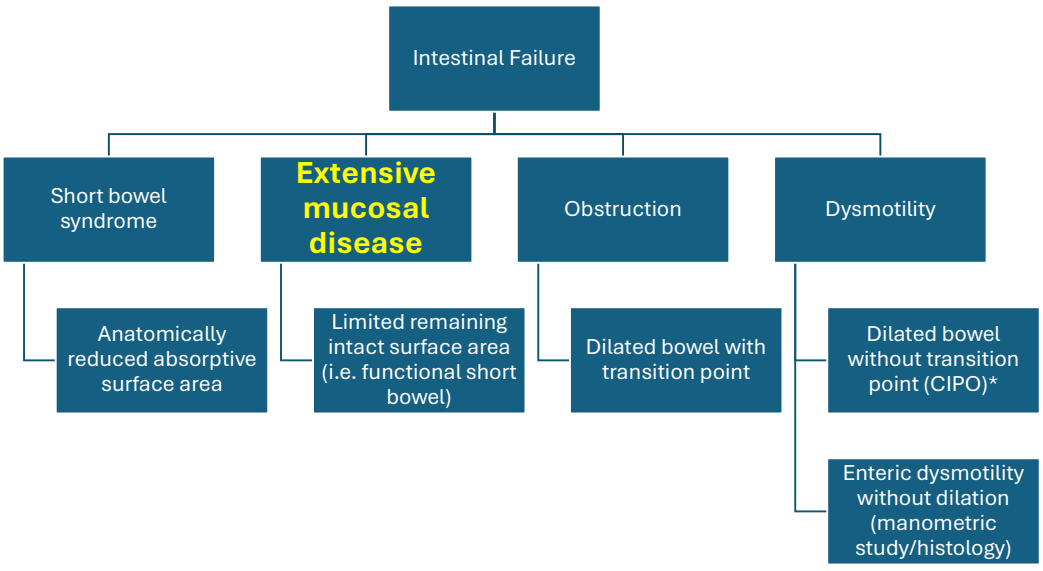

CIPO: Chronic Intestinal Pseuo-Obstruction

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Extensive mucosal disease

- Inflammatory conditions affecting small bowel
  - ↓ surface area = ↓ digestion and absorption = *Functionally* short bowel
  - Protein-losing enteropathy
- Cues: GI symptoms and weight loss despite adequate intake.
- **Autoimmune**: SB Crohn's, Refractory Celiac, Eosinophilic GI disease, vasculitis, Graft-vs-host disease, Combined variable immunodeficiency disorder
- **Iatrogenic**: Immunotherapy, radiation

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Extensive mucosal disease - diagnosis

- **Clinical & medical history. Screen for EIM of GI disorders.**
  - E.g. Fatigue, brain fog, rash, sores, arthralgia, fracture, nephrolithiasis
- Biochemical testing (CBC, CMP, anti-tTG, CRP, fecal cal, fecal fat, Ig's)
- Endoscopic evaluation (EGD, Capsule, colonoscopy)
- Imaging (CT / MR enterography)

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Extensive mucosal disease - therapy

- **Treat the underlying primary disorder**
  - Collaboration with other disciplines (e.g. heme/onc, rheum, allergy/immunology, surgery)
- **Determine nutrition status & need for nutrition therapy**
  - Nutrition assessment tools: GLIM, SGA
  - Weight loss, intake, p/e (function, muscle/fat, edema)
  - Ongoing assessment…

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GLIM (Global Leadership Initiative on Malnutrition)

Phenotypic criteria			Etiologic criteria	
Weight loss	Low body mass index	Reduced muscle mass	Reduced food intake or assimilation	Inflammation
>5% within past 6 months (>10% = severe) >10% beyond 6 months (>20% = severe)	<20 if < 70 years (<18.5 = severe) <22 if > 70 years (<20 = severe) Asian: <18.5 if < 70 years <20 if > 70 years	Reduced by validated body composition measuring techniques*	≤ 50% of energy requirement > 1 week Any reduction for > 2 weeks Any GI condition that adversely impacts food assimilation or absorption	Acute disease/injury **Chronic disease-related**

*DXA, Bioelectrical impedance, CT, MRI (or mid-arm muscle, calf circumference)

Cederhom et al. Clinical Nutrition 2018

30

GLIM vs SGA, or both?

- GLIM may pick up more malnutrition in IBD due to presence of inflammation as etiologic criteria (w/o necessarily reduced PO)
- SGA relies on reduce PO, weight loss, physical exam, function

A - Well-nourished no decrease in food/nutrient intake; < 5% weight loss; no/minimal symptoms affecting food intake; no deficit in function; no deficit in fat or muscle mass **OR** *an individual with criteria for SGA B or C but with recent adequate food intake; non-fluid weight gain; significant recent improvement in symptoms allowing adequate oral intake; significant recent improvement in function; and chronic deficit in fat and muscle mass but with recent clinical improvement in function.

B - Mildly/moderately malnourished definite decrease in food/nutrient intake; 5% - 10% weight loss without stabilization or gain; mild/some symptoms affecting food intake; moderate functional deficit or recent deterioration; mild/moderate loss of fat and/or muscle mass **OR** *an individual meeting criteria for SGA C but with improvement (but not adequate) of oral intake, recent stabilization of weight, decrease in symptoms affecting oral intake, and stabilization of functional status.

C - Severely malnourished severe deficit in food/nutrient intake; > 10% weight loss which is ongoing; significant symptoms affecting food/ nutrient intake;severe functional deficit **OR** *recent significant deterioration obvious signs of fat and/or muscle loss.

https://nutritioncareincanada.ca/sites/default/uploads/files/SGA%20Tool%20EN%20colour_2017(1).pdf

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Fallacy of the protein markers

- **Be careful of albumin and protein levels** in inflammatory states!
- Normal albumin level can be maintained by muscle breakdown or redistribution from extra-vascular space (60%) to intra-vascular space (40%)
- Negative phase reactant
  - Loss from intravascular space / catabolism
  - Reduced synthesis in liver

Cameron et al. JGH 2024

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Malnutrition suspected / identified

- Refer to dietitian for more in-depth review and diet counselling
- Complex nutrition care also involves nurse, pharmacist, patient
- Modes of medical nutrition therapy:
    1. Oral diet modification / Oral nutrition supplement
    2. **Enteral nutrition** (polymeric vs pre-digested)
    3. **Parenteral support** (IV fluid, lytes, nutrient)
    4. Combinations of any of the above

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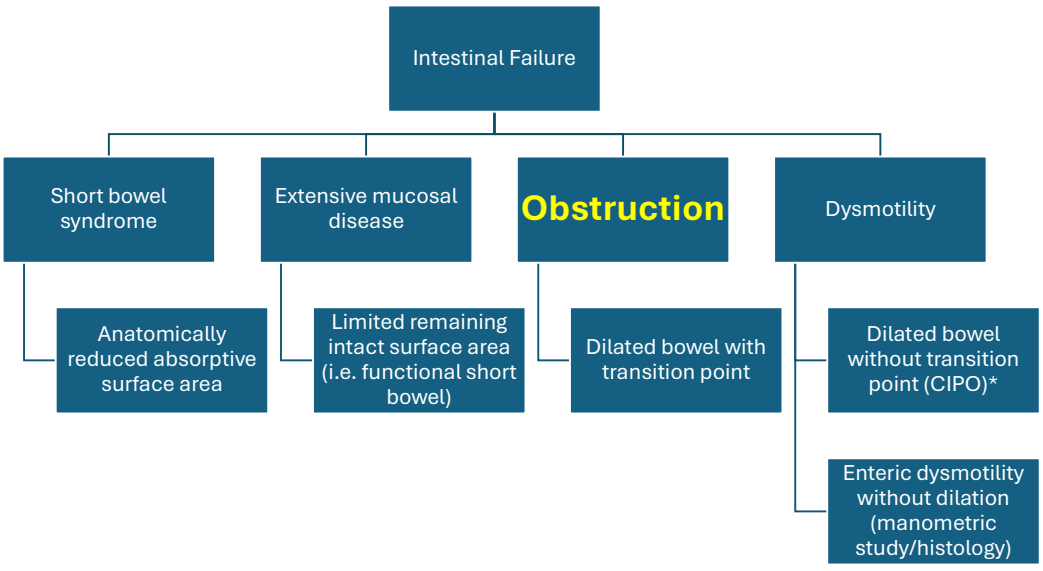

CIPO: Chronic Intestinal Pseuo-Obstruction

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Obstruction

- Mechanical obstruction of digestive tract
  - acute vs chronic/persistent vs recurrent
- Cues: Obstipation/constipation with intolerance to any oral intake.
- **Luminal**: Inflammatory stricture (e.g. Crohn's, radiation, NSAIDs), anastomotic stricture, intussusception, mass
- **Extraluminal**: adhesions, mass, volvulus

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Obstruction - diagnosis

- **Clinical & medical/surgical history.**
  - Personal and family hx cancer (? Age-appropriate screening)
  - Personal hx of surgery (e.g. adhesions, anastomotic stricture)
- Diagnosis is usually based on cross-sectional imaging demonstrating **transition point** during acute episode.
- Tissue diagnosis helps with determining cause and treatment of malignant obstruction

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Obstruction - therapy

- Supportive vs surgical depending on etiology
  - Discussion between medical and surgical teams
  - **Somatostatin** analogue and **corticosteroid**
    - (Feuer and Broadley. Cochrane 2000; Obita et al. J Pain Symptom Manage 2016)
- Determine nutrition status & need for nutrition therapy
  - **Ensure nutrition therapy aligns with GOC** (e.g. palliative patient)
- Usually requires ***total*** parenteral nutrition as enteral route not feasible

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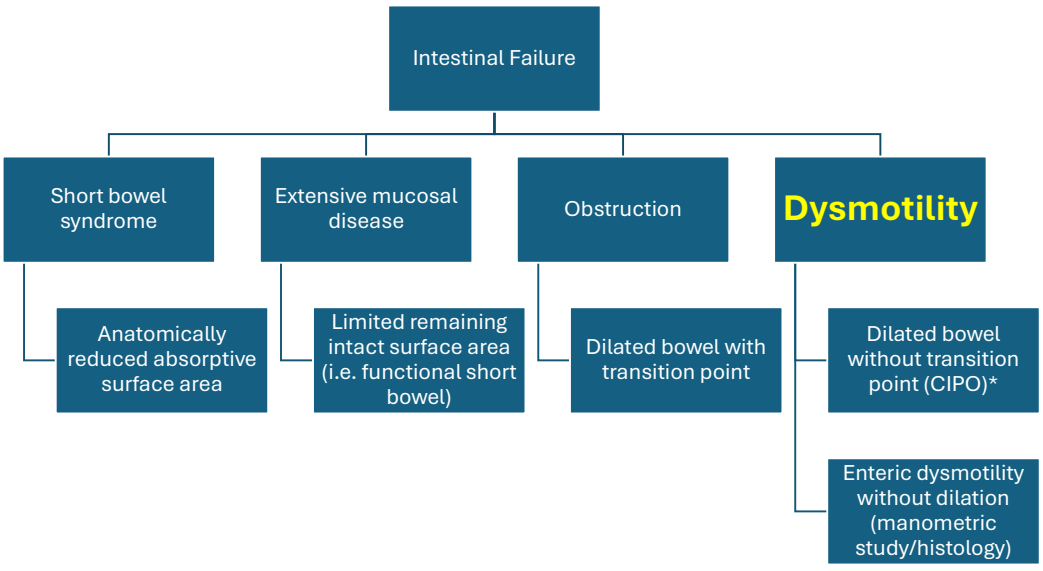

CIPO: Chronic Intestinal Pseuo-Obstruction

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Dysmotility

- Due to neurological or motor dysfunction
- Chronic intestinal pseudo-obstruction (CIPO)
  - **Dilated** bowel **without** transition point due to motor dysfunction
- Enteric dysmotility
  - Usually **non-dilated** bowel
  - Relies on manometric/transit studies and/or histology (n/a at most institutions) - Diagnosis not always possible

Nightingale et al. Gut 2020

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Dysmotility by etiology

- Myopathy
  - Primary: Hollow Visceral Myopathy
  - **Secondary**: Connective tissue disease (e.g. scleroderma), Radiation, Infiltrative (e.g. Amyloid), Muscular dystrophies
- Neuropathy
  - Primary: Mitochondrial dz (e.g. MNGIE), infections (e.g. viral, Chaga's)
  - Secondary: CNS disorders (e.g. MS, PD), diabetes, paraneoplastic syndrome, drugs (anti-cholinergic, opioids), *malnutrition*

MNGIE: Mitochondrial neurogastrointestinal encephalomyopathy

Nightingale et al. Gut 2020

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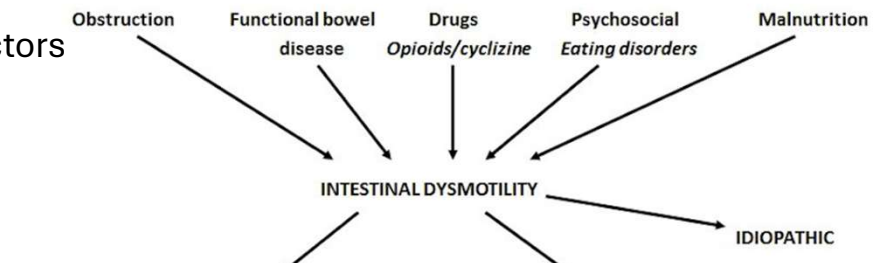
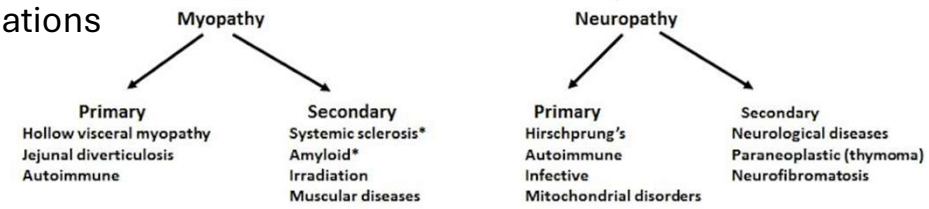
Contributing factors



Types / Classifications



*: Systemic sclerosis and amyloid can belong to both myopathy and neuropathy.

Nightingale et al. Gut 2020

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Dysmotility – diagnosis

- **Clinical & medical/surgical history.**
  - Co-existing congenital, neurological, psychological, autoimmune conditions
- Diagnosis conducted in a multi-disciplinary fashion.
  - Rule out obstruction
  - Assess motility (E.g. Manometry, transit studies/scintigraphy, gastric emptying study, defecography) while minimizing confounders (e.g. Opioids).

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Diagnostic momentum in the digital era

- "the tendency of a diagnosis to be accepted and passed on, with little examination of the underlying evidence for its validity"
- Be mindful of premature labeling, especially in non-dilated bowel - **"probable" or "suspected"** may be more accurate.
- Functional dyspepsia (DGBI) ↔Gastroparesis ≠ Enteric dysmotility
  - Disordered eating
  - Important clinical distinction but may be challenging to establish

Satiya-Murti and Lockhart. Neurol Clin Pract. 2015
Paine et al. Frontline Gastroenterology 2020

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ESPEN Endorsed Recommendation

Avoiding the use of long-term parenteral support in patients without intestinal failure: A position paper from the European Society of Clinical Nutrition & Metabolism, the European Society of Neurogastroenterology and Motility and the Rome Foundation for Disorders of Gut–Brain Interaction

S. Lal [a,*], P. Paine [b], J. Tack [c,d], Q. Aziz [e], R. Barazzoni [f], C. Cuerda [g], P. Jeppesen [h], F. Joly [i], G. Lamprecht [j], M. Mundi [k], S. Schneider [l], K. Szczepanek [m], A. Van Gossum [n], G. Wanten [o], T. Vanuytsel [c], L. Pironi [p,q]

"As there is also no established evidence that patients with HD/hEDS have small intestinal malabsorptive failure, **PN should be avoided other than again in life threatening extremis as a temporary bridge** to pain management and rehabilitative MDT therapies. This may be particularly important since anecdotal clinical experience suggests that HPN-related CRBSI are more frequent in this patient cohort."

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Dysmotility - therapy

- Treat the underlying primary condition (e.g. Scleroderma)
  - Multiple specialties (e.g. neurology, rheumatology, surgery, oncology)
- Supportive therapy, targeting the predominant symptom and abnormal results of testing (e.g. gastric emptying test).
- Avoid/Wean opioids – risk of narcotic bowel & confounds dx test
- Minimize medialization (over-investigation or treatment)

Nightingale et al. Gut 2020

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Dysmotility - therapy

- Determine nutrition status & need for nutrition therapy
  - Physical exam, measured weight trend Food diary
- Complex nutrition care also involves nurse, pharmacist, patient
- Modes of medical nutrition therapy:
  1. **Oral diet modification / Oral nutrition supplement**
  2. **Enteral nutrition** (+++ formulas)
  3. **Parenteral support** (IV fluid, lytes, nutrient)
  4. Combinations of any of the above

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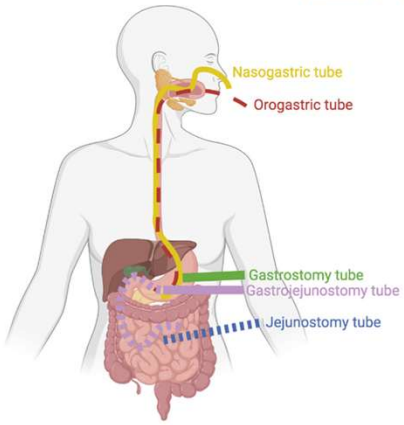
Dysmotility - nutrition therapy



- Enhanced ("effortful") oral feeding
  - Small volume, low fat, small particle diet
  - Oral nutrition supplements
- Enteral nutrition – gradual delivery
  - Nasogastric / Nasojejunal
  - Percutaneous gastrostomy / jejunostomy / gastroje
- Requires patience and ++coaching
- May require correction of severe malnutrition w/ parenteral support

Paine et al. Frontline Gastroenterology 2020
Ley et al. JPEN 2023

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Case #1 – 28yo with abdo pain and vomiting

- EGD and colonoscopies non-contributory.
- ++CT scans over time – dilated stomach, small bowel distention and sometimes potential transition proximal to IC valve
- Past surgeries = no adhesions or causes of obstruction. No full thickness Bx. **Diagnosed with CIPO** & recommended no further surgeries.
- Attempted G tube for feeding but removed due to pain at subxiphoid.
- Continues PN support at home + PO intake for pleasure (→ emesis).

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Case #2 – 37yo with malignant obstruction

- Due to indolent nature of metastatic disease and high functional capacity with QoL acceptable to the patient. Shared decision among team and patient for PN support at home and did well for > 1 year.
- Venting G tube allowed minimal oral intake for pleasure and social interactions.

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Case #3 – 20yo with oral intolerance

- Multiple disciplines involved: IM, neuro, rheum, complex pain, psych, urology, cardiology, GI, rad, surgery, outpatient RD, physio
- Various forms of EN tried (e.g. NG, ND, perc GJ, separate perc G + J, perc G). Various feeds.
- Inpatient PN for acute malnutrition management. CLABSI with candida sp. Weaned off PN.
- Addressing main bothersome symptom = Constipation.
  - Multiple meds for constipation: PEG3350, linaclotide, plecanatide, tenapanor, prucalopride
  - **Defecography** - generalized pelvic floor descent on straining; inappropriate sensation of incomplete evacuation → **Pelvic floor physio**
- Nutrition support: combination of PO, EN and outpatient IV fluids via a port for POTS symptoms

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Summary

- Non-short bowel causes of intestinal failure includes:
  - Extensive mucosal disease with reduced absorption*
  - Mechanical bowel obstruction
  - Intestinal dysmotility (dilated vs non-dilated bowel)*
- Objectively assess for malnutrition to determine nutrition support
- *EN/PO support may still be tried.
- Parenteral support may be indicated, failing PO/EN trials, or if in line with patient's GOC in the event of malignant obstruction.

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Questions

George Ou, MD

Omar Jamil, MD

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