

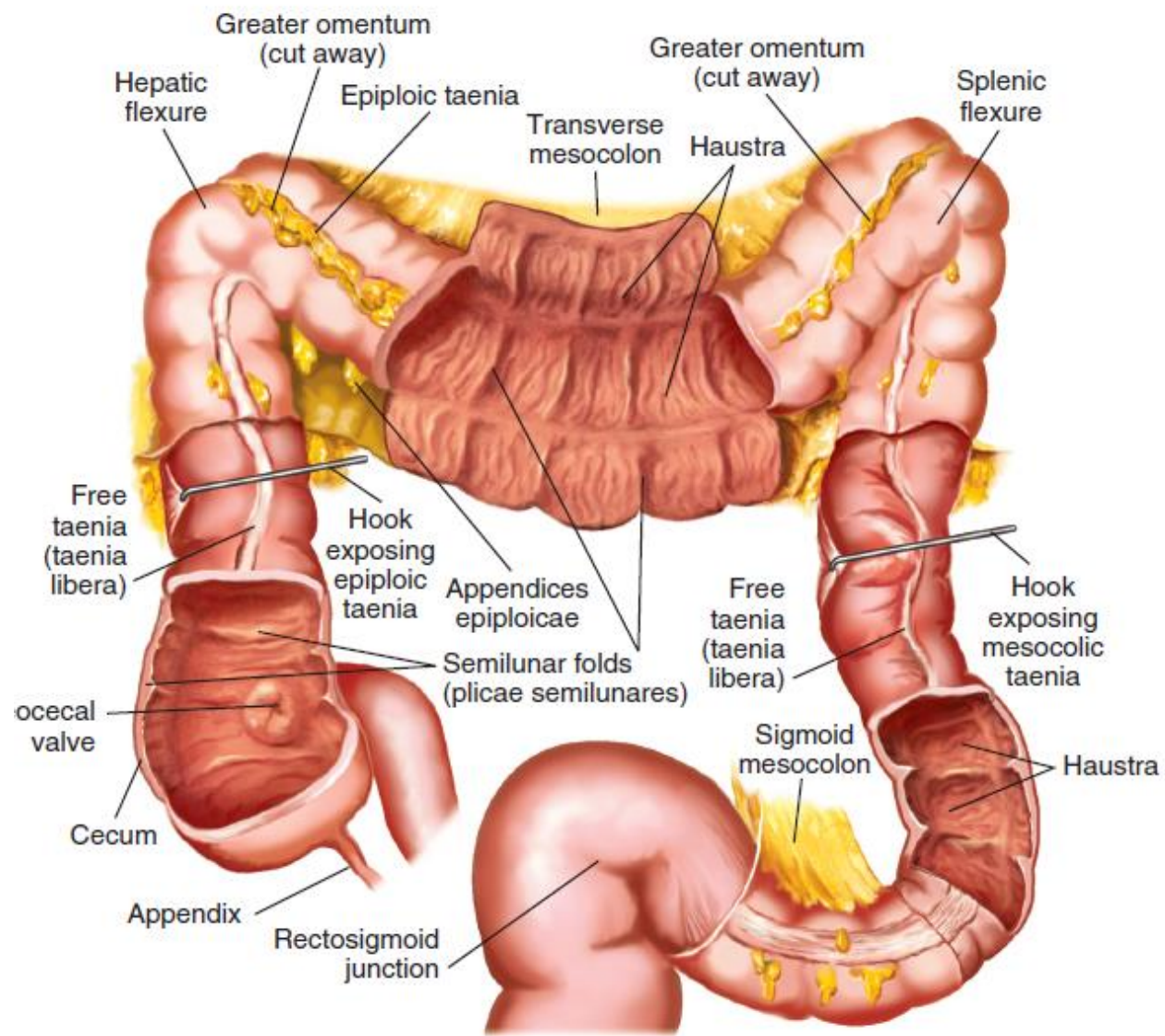
Constipation: An Overview

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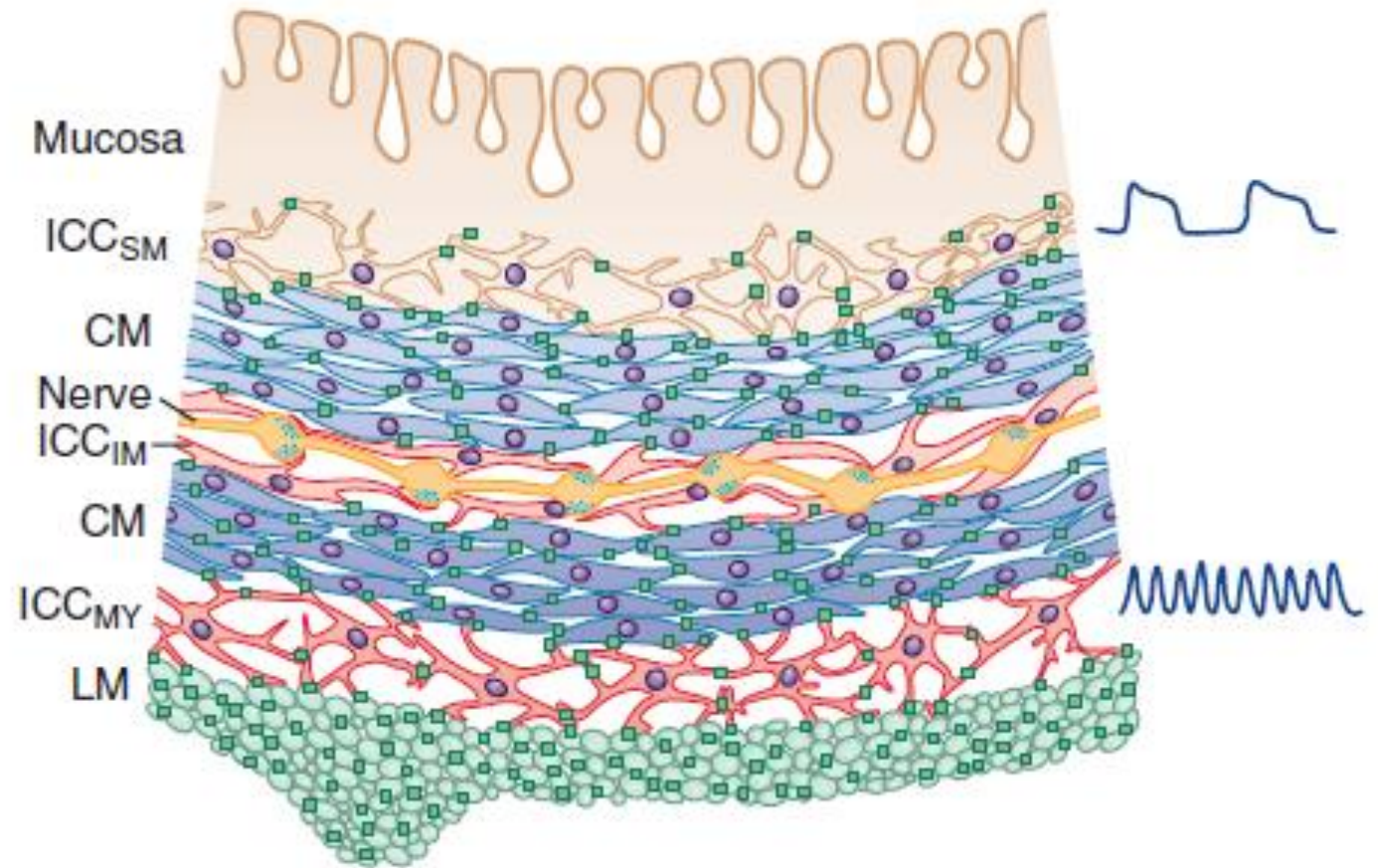
Back to the Basics- Function of the Colon

- Luminal contents include water, electrolytes, bacteria, gas, food residue
- Absorption of water and sodium: 1L-1.5L of fluid crosses IC valve → 100 to 200 mL of fecal water daily
- Patients with constipation have intact absorptive mechanisms, volume of stool water and quantity of stool solids is reduced proportionally
- Slow transit constipation- decreased peristaltic movement may lead to more bacterial degradation of stool → more NaCl and water reabsorption → stool weight/frequency
- Diameter/length of the colon → wide or long colon may be a reason for slow colonic transit

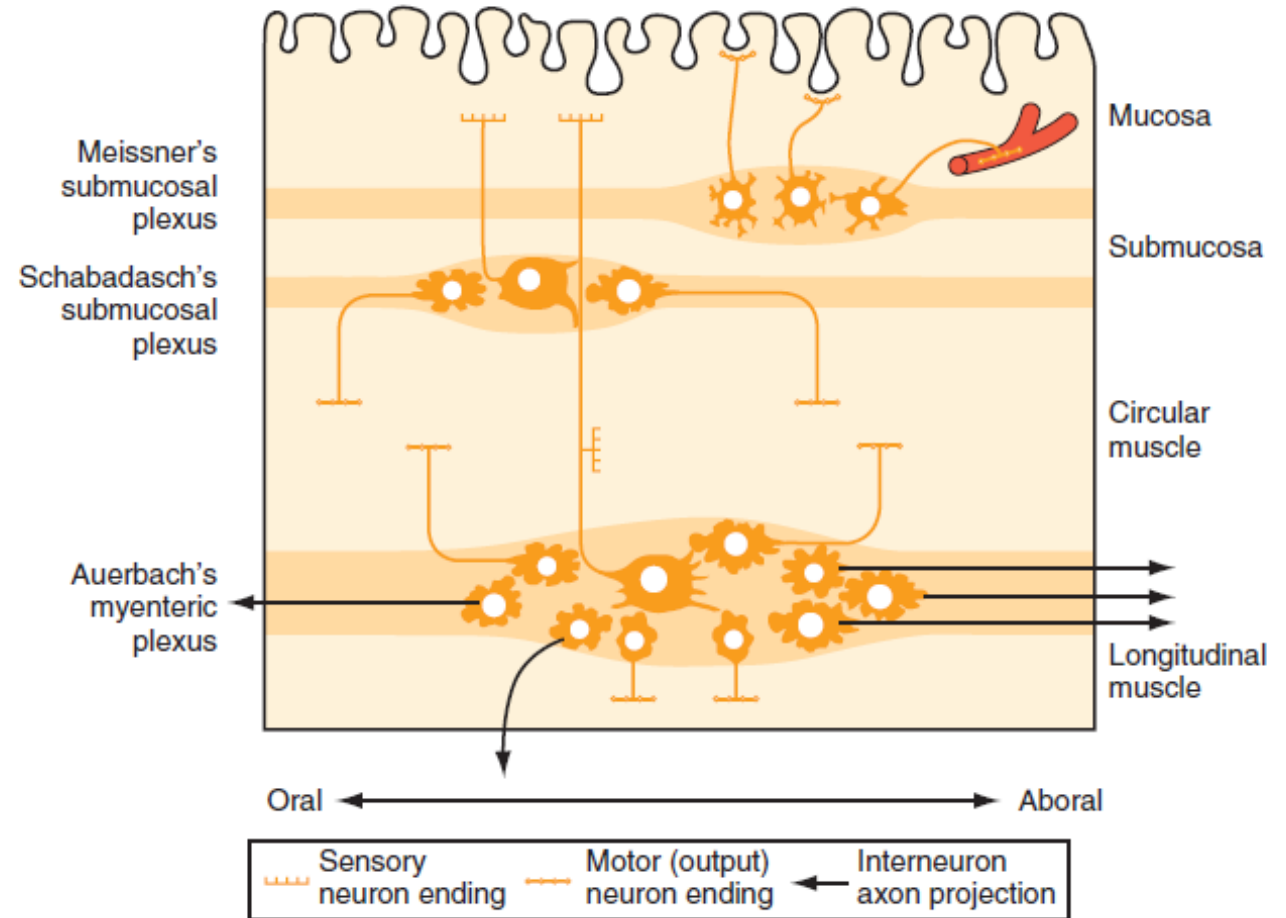
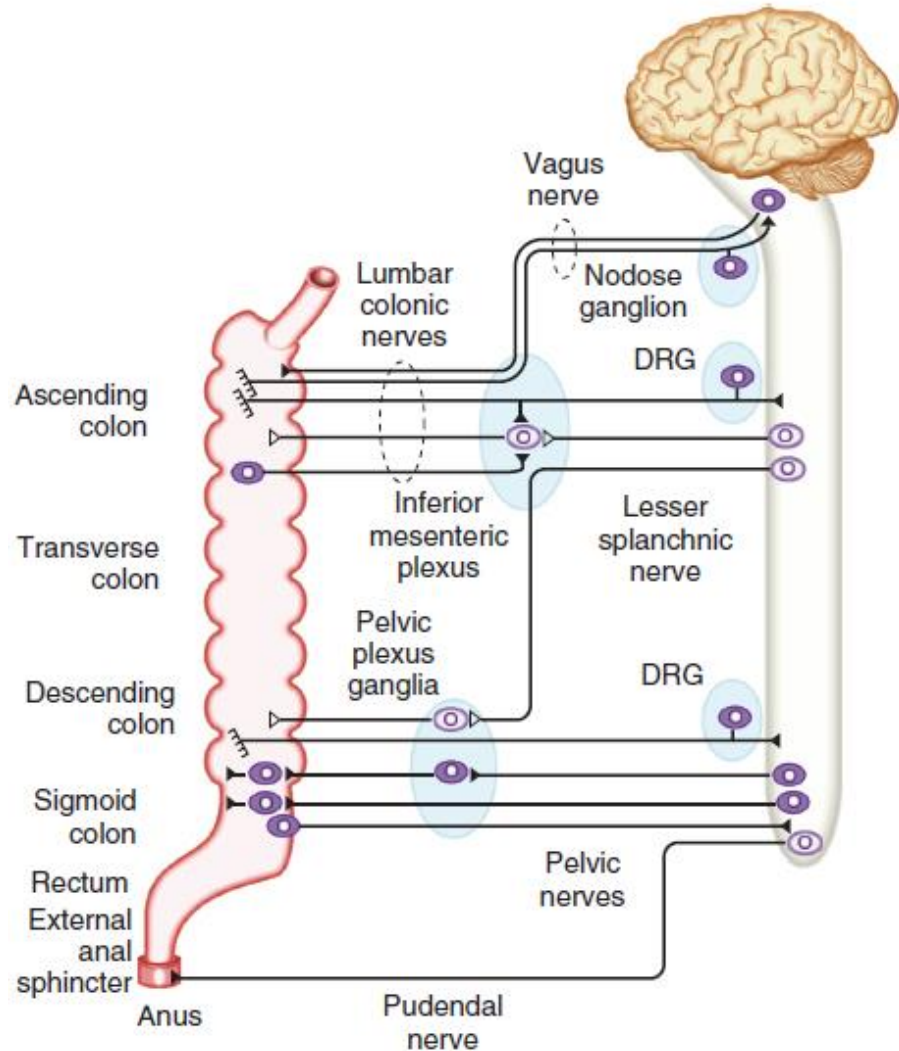
Back to the Basics- Motor Function of the Colonic Muscle

- Delay passage of luminal contents to allow for water absorption
- Mixes content and allows for contact with mucosa
- Storage of feces between defecation
- Propulsion of contents towards the anus
- Transit through the colon takes longer than elsewhere through the body → mean is about 34-35 hours, 72 hours is upper limit of normal
- Low-amplitude propagated contractions
- High-amplitude propagated contractions (HAPCs)
 - Frequency and duration reduced in some patients with constipation

Innervation of the colon



Innervation of the Colon



Innervation changes in constipation

- Slow transit constipation may be related to autonomic dysfunction
- Abnormal numbers of myenteric plexus neurons
 - Decreased excitatory substance P
 - Increased Inhibitory VIP or NO
- Reduced numbers of Interstitial Cells of Cajal
- Abnormal morphology of ICCs

Puborectalis muscle

Internal hemorrhoidal plexus

Internal anal sphincter

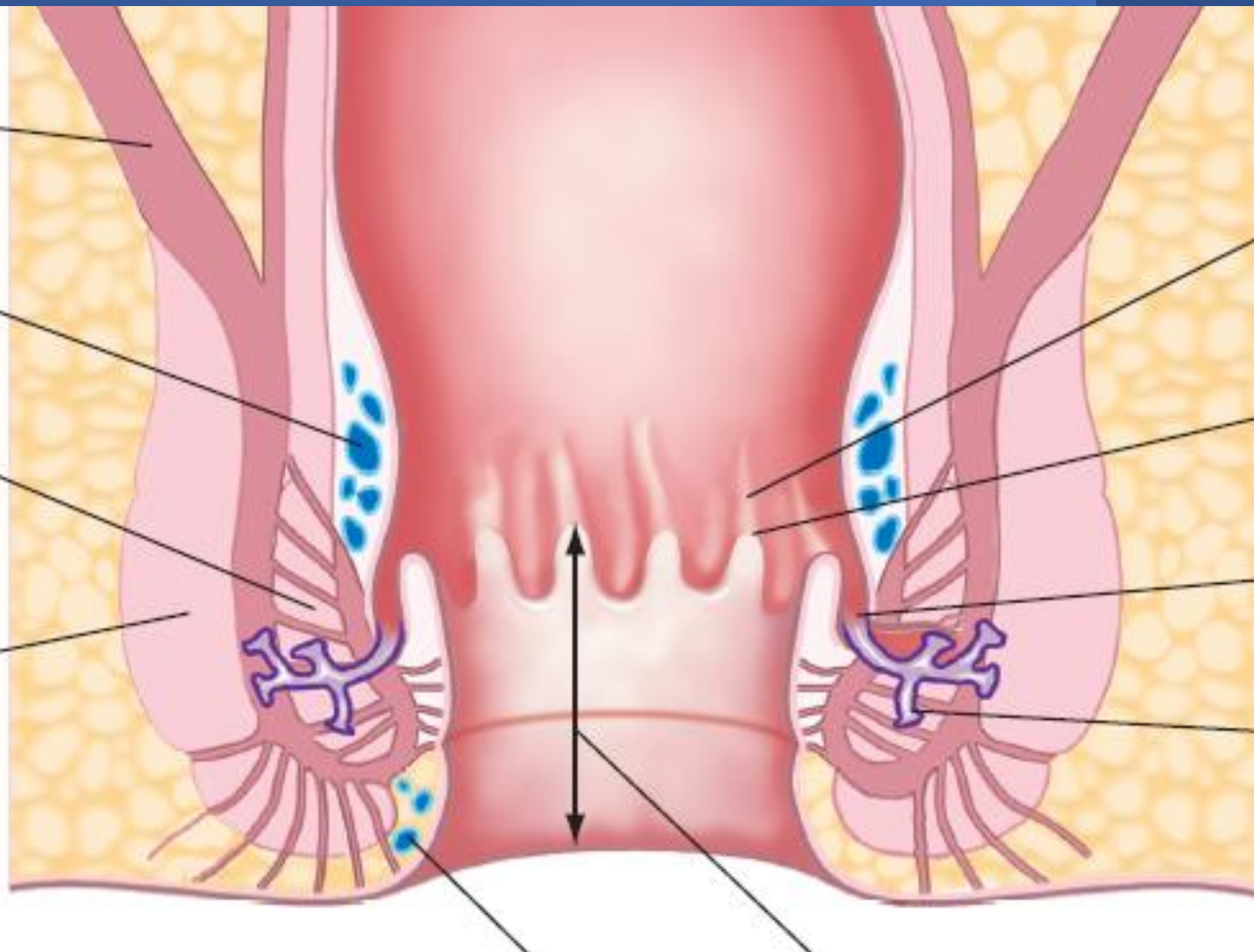
External anal sphincter

Column of Morgagni

Dentate line

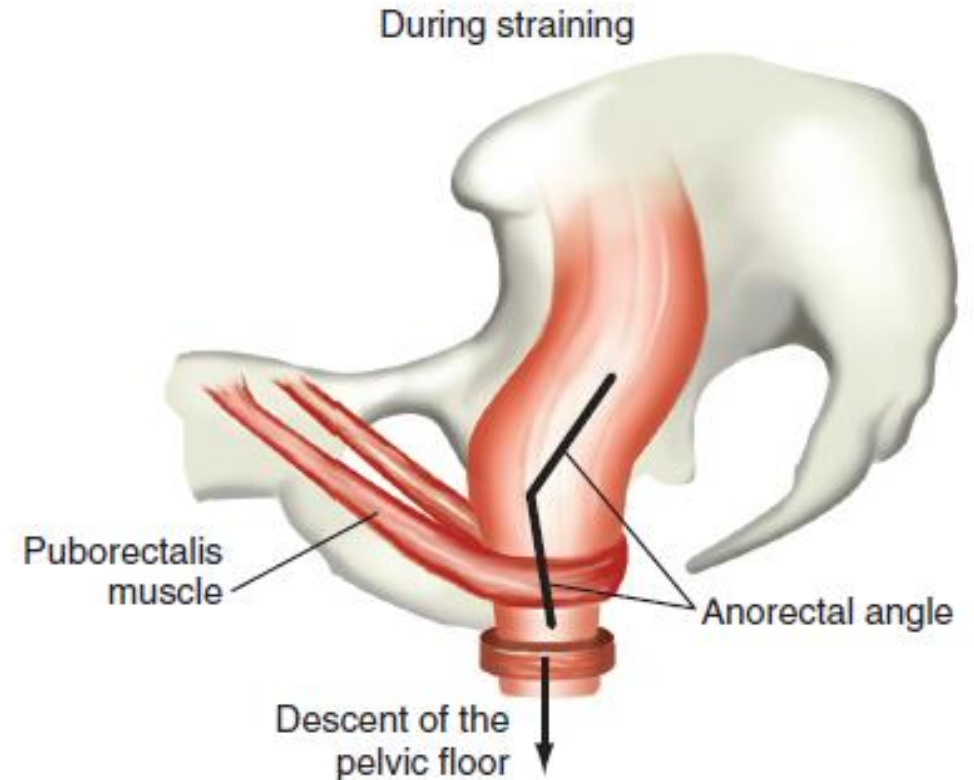
Anal crypt

Anal gland



Defecatory Function

- Predefecatory period
 - Increased amplitude and frequency of propagating sequences
 - Gastroileal reflex is a stimulus
- Urge to defecate
 - Stool comes into contact with receptors in upper anal canal
- . Coordinated relaxation of the puborectalis muscle and the external anal sphincter at a time when pressure is increasing in the rectum → expulsion of stool

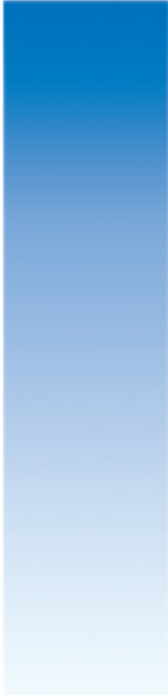








Effects of Age

- Age effect on Enteric Nervous System
 - Mouse models-
 - loss of cholinergic neurons in myenteric and submucosal plexus is noticeable in the distal GI tract
 - Accumulation of dystrophic axonal swellings in the innervation of the gut
 - Electrical stimulation in older patients more likely to cause muscle relaxation rather than contraction in a study on ex vivo colonic muscle
- Effect of Aging on Colonic and Rectoanal Sensorimotor Functions
 - Increased stiffness, reduced sensation in colon and rectum
 - Reduced resting pressures and increased perineal laxity
 - May lead to more fecal incontinence
 - Other factors that can contribute include reduced caloric intake, medications, microbiome disturbances, anatomical issues

The Basics of Constipation

- Mean prevalence among adults 14%
- Three or fewer bowel movements per week
- Stool frequency reports can be inaccurate

Whole-gut transit time	Type of stool	Description	Pictorial representation
Long transit (e.g., 100 hours) 	Type 1	Separate hard lumps, like nuts, hard to pass	
	Type 2	Sausage shaped but lumpy	
	Type 3	Like sausage but with cracks on its surface	
	Type 4	Like sausage or snake, smooth and soft	
	Type 5	Soft blobs with clear-cut edges (passed easily)	
	Type 6	Fluffy pieces with ragged edges, a mushy stool	
	Type 7	Watery, no solid pieces	Entirely liquid
Short transit (e.g., 10 hours)			

Types of Constipation



Opioid induced
Constipation

Irritable Bowel
Syndrome with
Constipation
predominance (IBS-C)

Functional
Constipation

Defecatory Disorders



Rome Criteria

C2. FUNCTIONAL CONSTIPATION

*Diagnostic criteria**

1. Must include **two or more** of the following:**
2. Straining during more than ¼ (25%) of defecations
3. Lumpy or hard stools (Bristol Stool Form Scale 1-2) more than ¼ (25%) of defecations
4. Sensation of incomplete evacuation more than ¼ (25%) of defecations
5. Sensation of anorectal obstruction/blockage more than ¼ (25%) of defecations
6. Manual maneuvers to facilitate more than ¼ (25%) of defecations (e.g., digital evacuation, support of the pelvic floor)
7. Fewer than three SBM per week
8. Loose stools are rarely present without the use of laxatives
9. Insufficient criteria for irritable bowel syndrome

*Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

For research studies, patients meeting criteria for opioid-induced **constipation (OIC) should not be given a diagnosis of FC because it is difficult to distinguish between opioid side effects and other causes of **constipation**. However, clinicians recognize that these two conditions may overlap.

BOX 19-2 Risk Factors for Constipation

Advanced age

Female gender

Low level of education

Low level of physical activity

Low socioeconomic status

Nonwhite ethnicity

Use of certain medications (see [Box 19-3](#))

TABLE 19-1 Clinical Classification of Functional Constipation

Category	Features	Physiologic Test Results
Normal-transit constipation	Incomplete evacuation; abdominal pain may be present but not a predominant feature	Normal
Slow-transit constipation	Infrequent stools (e.g., $\leq 1/\text{wk}$), lack of urge to defecate, poor response to fiber and laxatives, generalized symptoms (e.g., malaise, fatigue); more prevalent in young women	Delay in colonic transit (e.g., retention in colon of $>20\%$ of radiopaque markers 5 days after ingestion)
Defecatory disorder*	Frequent straining, incomplete evacuation, need for manual maneuvers to facilitate defecation	Abnormal balloon expulsion test and/or anorectal manometry

Medications

Acetaminophen (>7 tablets weekly)

Antacids (aluminum containing)

Anticholinergic agents (e.g., antiparkinsonian drugs, antipsychotics, antispasmodics, tricyclic antidepressants)

Anticonvulsants (e.g., carbamazepine, phenobarbital, phenytoin)

Antineoplastic agents (e.g., vinca derivatives)

Calcium channel blockers (e.g., verapamil)

Calcium supplements

Diuretics (e.g., furosemide)

5-Hydroxytryptamine₃ antagonists (e.g., alosetron)

Iron supplements

NSAIDs (e.g., ibuprofen)

Mu-opioid agonists (e.g., fentanyl, loperamide, morphine)

BOX 19-3 Secondary Causes of Constipation

Mechanical Obstruction

Anal stenosis

Colorectal cancer

Extrinsic compression

Rectocele or sigmoidocele

Stricture

Secondary Causes of Constipation

Metabolic and Endocrinologic Disorders

Diabetes mellitus

Heavy metal poisoning (e.g., arsenic, lead, mercury)

Hypercalcemia

Hyperthyroidism

Hypokalemia

Hypothyroidism

Panhypopituitarism

Pheochromocytoma

Porphyria

Pregnancy

Secondary Causes of Constipation

Neurologic and Myopathic Disorders

Amyloidosis

Autonomic neuropathy

Chagas' disease

Dermatomyositis

Intestinal pseudo-obstruction

Multiple sclerosis

Parkinsonism

PSS

Shy-Drager syndrome

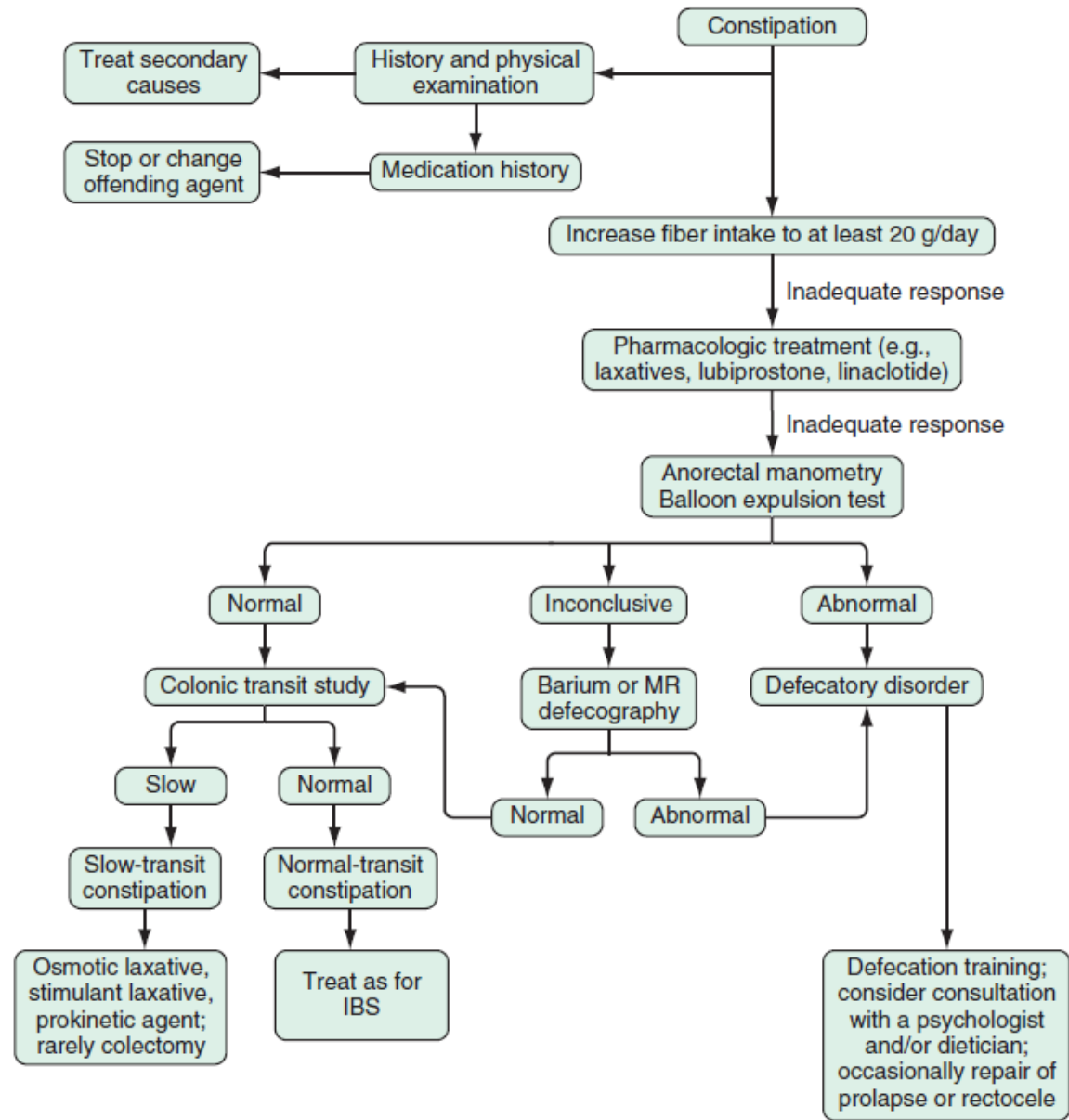
Spinal cord injury

Stroke

Constipation in Psychological Disorders

- Symptom of a psychiatric disorder
- Side effect of treatment
- Depression → constipation can be a somatic manifestation
- Patients with eating disorders may have prolonged whole-gut transit time and pelvic floor dysfunction
- Severe psychiatric disorders patients may not be able to report defecation accurately

General workup of Constipation



History

- Duration of symptoms, frequency of bowel movements and associated symptoms (abdominal discomfort, distension, incomplete evacuation)
- Assess stool size and degree (Bristol chart)
- **Red flag signs**
 - Unexplained weight loss, change in caliber of stool, severe abdominal pain, family history of cancer, hematochezia
- Assessment of prior surgical history
- Medication history

Physical Exam

- Palpate abdomen for distension/masses
- Rectal exam
 - Left lateral position
 - Evaluate the perineal area for any obvious scars, external hemorrhoids, fissures
 - Assess for tone
 - DRE
 - Evaluate for fecal impaction, anal stricture or any obvious masses

Palpation

High anal sphincter tone at rest precludes easy entry of the examining finger (in absence of a painful perianal condition [e.g., anal fissure])

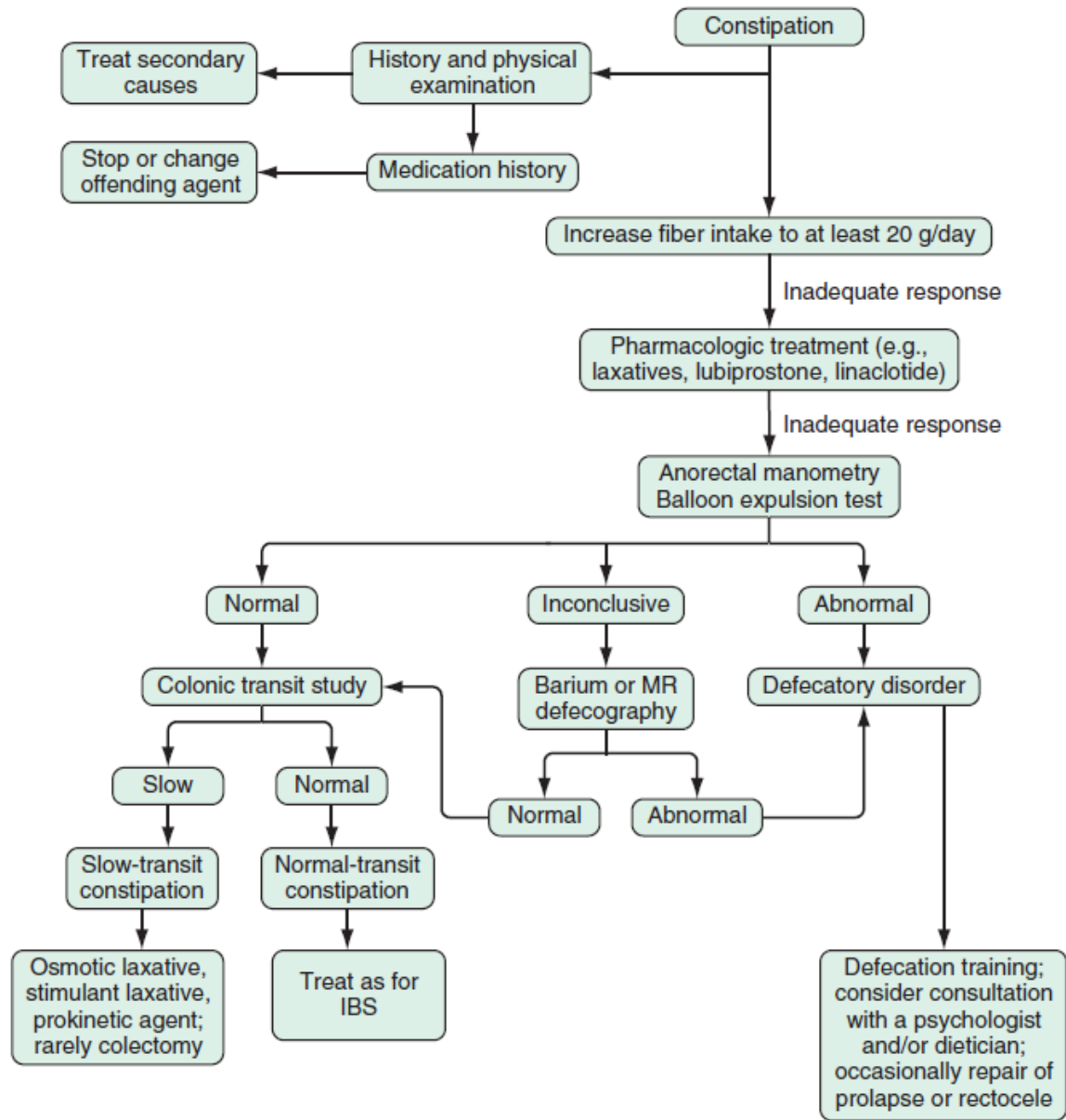
Anal sphincter pressure during voluntary squeeze only minimally higher than anal pressure at rest

The perineum and examining finger descend <1 cm or >4 cm during simulated straining at defecation

The puborectalis muscle is tender to palpation through the rectal wall posteriorly, or palpation reproduces pain

Palpable mucosal prolapse during straining

“Defect” in anterior wall of the rectum, suggestive of rectocele



Tests for Structural Disease and Physiologic Testing

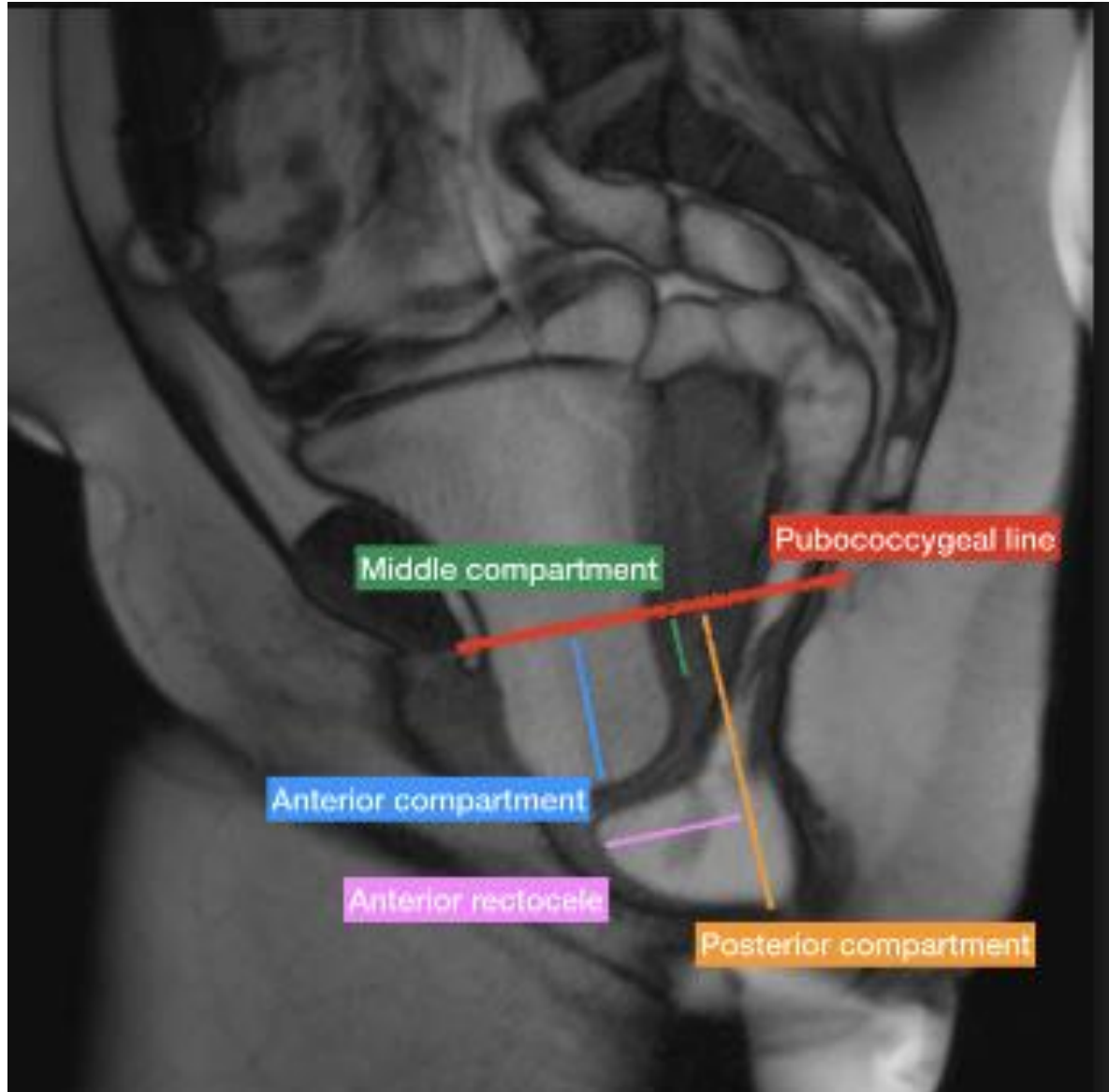
- CT, MRI, barium enema
- Colonoscopy if red flag signs
- Measurement of Colonic Transit Time
 - Radiopaque markers (Sitz marker study)
 - Wireless Motility Capsule
 - Colonic Scintigraphy

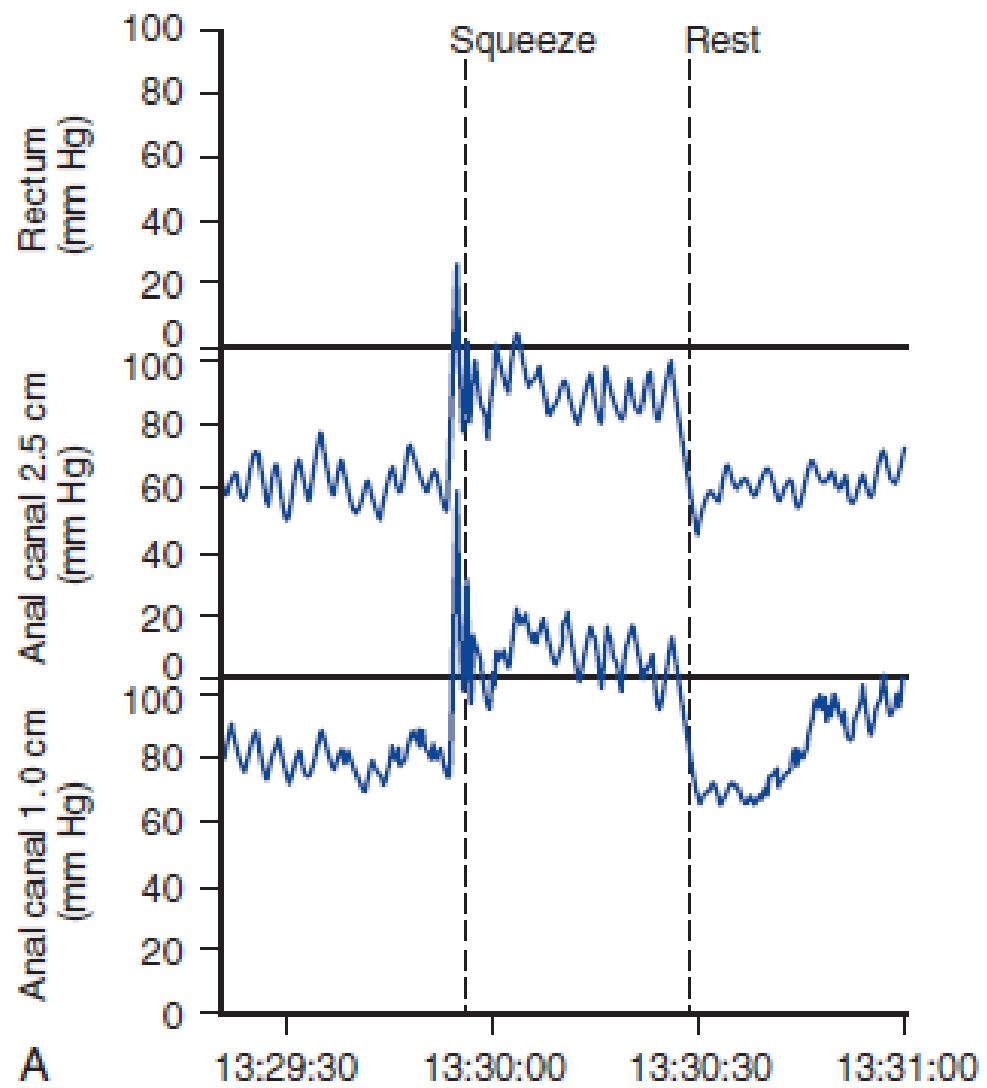
Dyssynergia

- Needs two out of three of the following
 - Impaired evacuation on balloon expulsion or defecography
 - Inappropriate contraction of the pelvic floor on imaging, EMG, or manometry
 - Inadequate propulsive forces from manometry or imaging

Studies for Physiology of Defecation

- Anorectal Manometry
- Balloon Expulsion Test
- Defecography
- Electromyography





Treatment Strategies

- Nonpharmacologic interventions
 - Reassurance
 - Lifestyle changes → always respond to a defecatory urge
- Psychological Support
- Discontinue any offending medications
- Fluid intake
- Biofeedback therapy if dyssynergia is diagnosed

squatty potty :)

35°



This **convenient, sturdy structure** is engineered to mirror the angle and efficiency of **natural squatting**, while allowing for the **luxury and comfort** of your own toilet.



TABLE 19-2 Commercial Fiber Products

Agent	Starting Daily Dose (g)	Comments
Methylcellulose	4-6	Semisynthetic cellulose fiber that is relatively resistant to colonic bacterial degradation and tends to cause less bloating and flatus than psyllium
Psyllium	4-6	Made from ground seed husk of the ispaghula plant; forms a gel when mixed with water, so an ample amount of water should be taken with psyllium to avoid intestinal obstruction; undergoes bacterial degradation, which may contribute to side effects of bloating and flatus; allergic reactions (e.g., anaphylaxis, asthma) have been reported but are rare
Polycarbophil	4-6	Synthetic fiber made of polymer of acrylic acid, which is resistant to bacterial degradation
Guar gum	3-6	Soluble fiber extracted from seeds of the leguminous shrub <i>Cyamopsis tetragonoloba</i>

- Increase intake of dietary fiber in non constipated patients → increases stool weight and frequency
- May not be as effective alone in severely constipated patients who pass small stools, but is helpful in mild to moderate constipation
- Should still be tried as a first line
- 20-25 g/day is eventual target of dietary fiber

Type of Laxative	Generic Name(s)	Dose	Comments
Osmotic Laxatives <i>Poorly Absorbed Ions</i>			
Magnesium	Magnesium hydroxide	15-30 mL once or twice daily	Hypermagnesemia can occur in patients with renal failure and in children
Sulfate	Magnesium citrate Magnesium sulfate Sodium sulfate (Glauber's salt)	75-150 mL every day 10-15 g every day 5-10 g every day	Often used as part of a bowel preparation Sodium sulfate is generally not used by itself as a laxative agent
Phosphate	Sodium phosphate	0.5-10 mL with 12 oz of water	Hyperphosphatemia can occur, especially in patients with renal failure
<i>Poorly Absorbed Sugars</i>			
Disaccharides	Lactulose	15-30 mL once or twice daily	Gas and bloating are common side effects
Sugar alcohols	Sorbitol Mannitol	15-30 mL once or twice daily 15-30 mL once or twice daily	Sorbitol is commonly used as a sweetener in sugar-free products. In older adults, sorbitol has an effect similar to lactulose but costs less. Rarely used as a laxative
Polyethylene glycol	Polyethylene glycol electrolyte	17-34 g once or twice daily	Tends to cause less bloating and cramps than other agents; tasteless and odorless, can be mixed with noncarbonated beverages. Typically used to prepare the colon for diagnostic examinations and surgery; also available as a powder without electrolytes for regular use

Stimulant Laxatives			
Anthraquinones Ricinoleic acid Diphenylmethane derivatives	Cascara sagrada	325 mg (or 5 mL) at bedtime	Cause apoptosis of colonic epithelial cells that are phagocytosed by macrophages; result in a lipofuscin-like pigmented condition known as <i>pseudomelanosis coli</i> ; no definitive association has been established between anthraquinones and colon cancer or myenteric nerve damage (cathartic colon) Cramping is common Has effects in the small intestine and colon Removed from the U.S. market because of teratogenicity in animals Likely has effects only on the colon. Although widely used in Europe, it is only available in the USA as part of a colonoscopy preparation
	Senna	1-2 7.5-mg tablets daily	
	Castor oil	15-30 mL at bedtime	
	Bisacodyl Phenolphthalein	5-10 mg at bedtime 30-200 mg at bedtime	
	Sodium picosulfate	5-15 mg at bedtime	
Stool Softeners	Docusate sodium	100 mg twice daily	Efficacy in constipation is not well established

Stool Softeners and Emollients

- Docusate sodium
 - Stimulates fluid secretion but does not increase weight of stools
- Mineral oils
 - Emulsify stool mass and provide lubrication for stool passage
 - Long term use → malabsorption of fat soluble vitamins
- Enemas and suppositories
 - Stimulate contraction with distension/chemical actio

Enemas and Suppositories

- Phosphate Enemas
 - Distension and stimulation of the rectum
 - Can cause superficial damage to the rectum
 - Should be used in healthier patients, not those that cannot evacuate it promptly (risk of hyperphosphatemia/hypocalcemia)
- Saline, Tap Water and Soapsuds Enemas
 - Softens the feces
 - Saline does no damage to rectal mucosa
 - Large volume soap suds enema can cause hyperphosphatemia if retained for too long and can cause necrosis
- Stimulant Suppositories and Enemas

Chloride Channel Activator	Lubiprostone	8-24 μg twice daily	Increases secretion in the intestine. Its mechanism of action is presumed to be via the chloride 2 channel
Guanylate Cyclase C Agonist	Linaclotide	145 μg once daily	Increases secretion in the intestine through cyclic guanosine monophosphate

Other effective medications

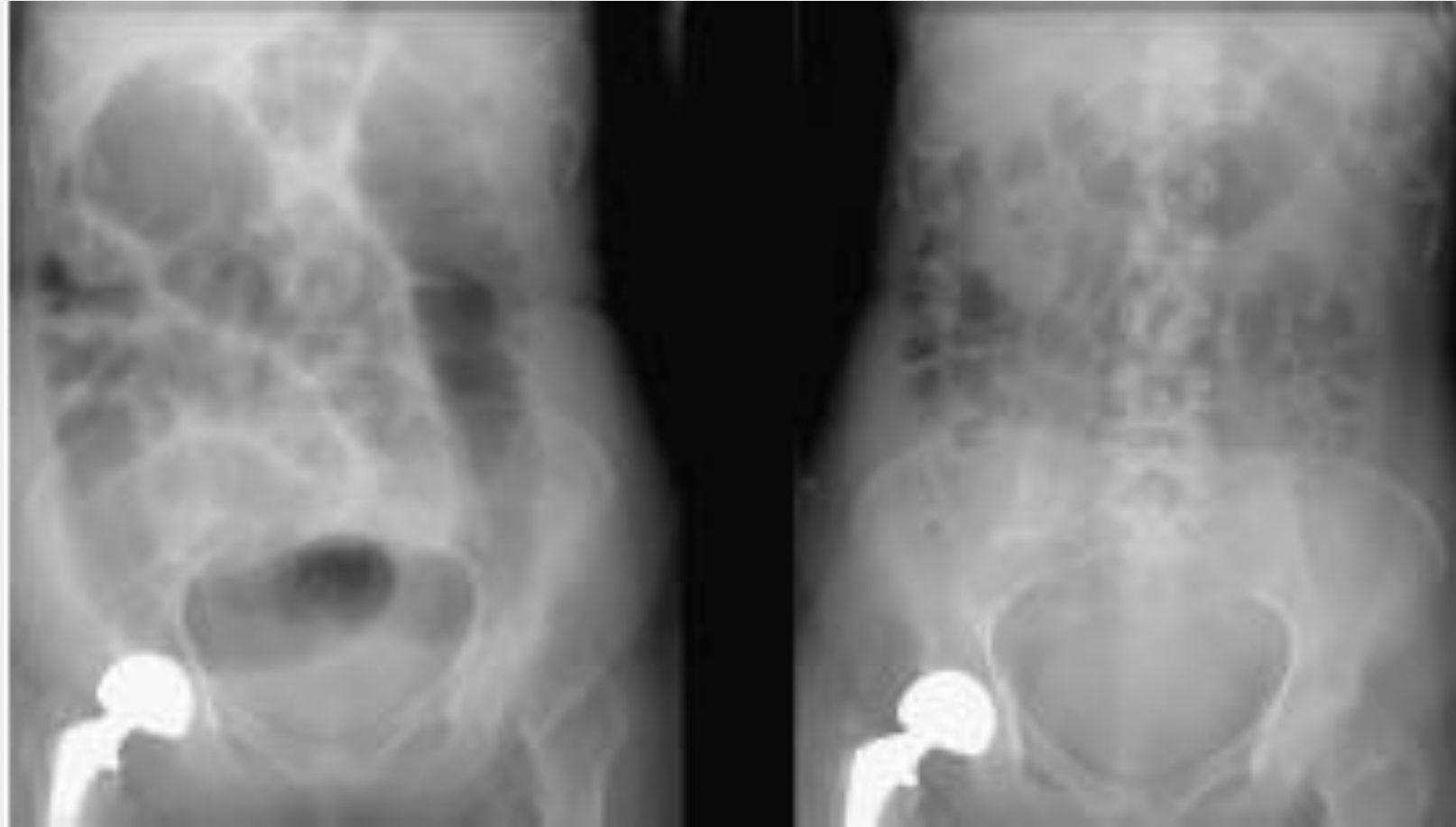
- Plecanatide (Trulance)
 - Similar mechanism to Guanylate Cyclase C
- Prucalopride (Motegrity)
 - Complete 5-HT₄ agonist
 - Accelerates colonic transit
- Methylnaltrexone (Naloxegol)
 - Mu-opioid receptor antagonist
 - Does not precipitate opioid withdrawal

Biofeedback and Pelvic Floor Therapy

- If dyssynergia is proved this can be very effective
- Defecation training can be offered first to try to reduce stigma around normal defecation process and decrease laxative burden
- Train patients to relax pelvic floor muscles while straining and possibly pair this with abdominal maneuvers to help advance stool

Colectomy

- Goal: Increase bowel frequency and ease of defecation
- Partial or total colectomy
- Construction of a stoma is occasionally performed v. ileostomy if there is a failure of initial colectomy
- Rule out small bowel dysmotility first



Acute Colonic Pseudo-Obstruction(Ogilvie's)

- Massive colonic dilation
- Unexplained by mechanical cause

TABLE 124-2 Conditions Associated with Acute Colonic Pseudo-obstruction (400 cases)

Health Condition (Most Common)	% of Cases
Trauma (nonoperative)	11.3
Infection (pneumonia, sepsis)	10.0
Cardiac (myocardial infarction, heart failure)	10.0
Gynecologic surgery	9.8
Abdominal/pelvic surgery	9.3
Neurologic (Parkinson's disease, spinal cord, multiple sclerosis, Alzheimer's disease)	9.3
Orthopedic surgery	7.3
Miscellaneous medical conditions (metabolic, cancer, respiratory failure, kidney failure)	32
Miscellaneous surgical conditions (urologic, thoracic, neurosurgical)	11.8

BOX 124-4 Proposed Mechanisms for Acute Colonic Pseudo-obstruction

Intestine Fails to Contract

Excess sympathetic motor input
Decreased parasympathetic motor input
Stimulation of peripheral μ -opioid receptors (endogenous or exogenous)
Reflex motor inhibition through splanchnic afferents

Intestine Fails to Relax

Excess parasympathetic motor input
Inhibition of nitric oxide release

Modified from Delgado-Aros S, Camillori M. Pseudo-obstruction in the critically ill. In: Scholmerich J, editor. Bailliere's best practice & research in clinical gastroenterology: Gastrointestinal disorders in the critically ill, vol. 17. London: Elsevier Science; 2003. pp. 427-44.

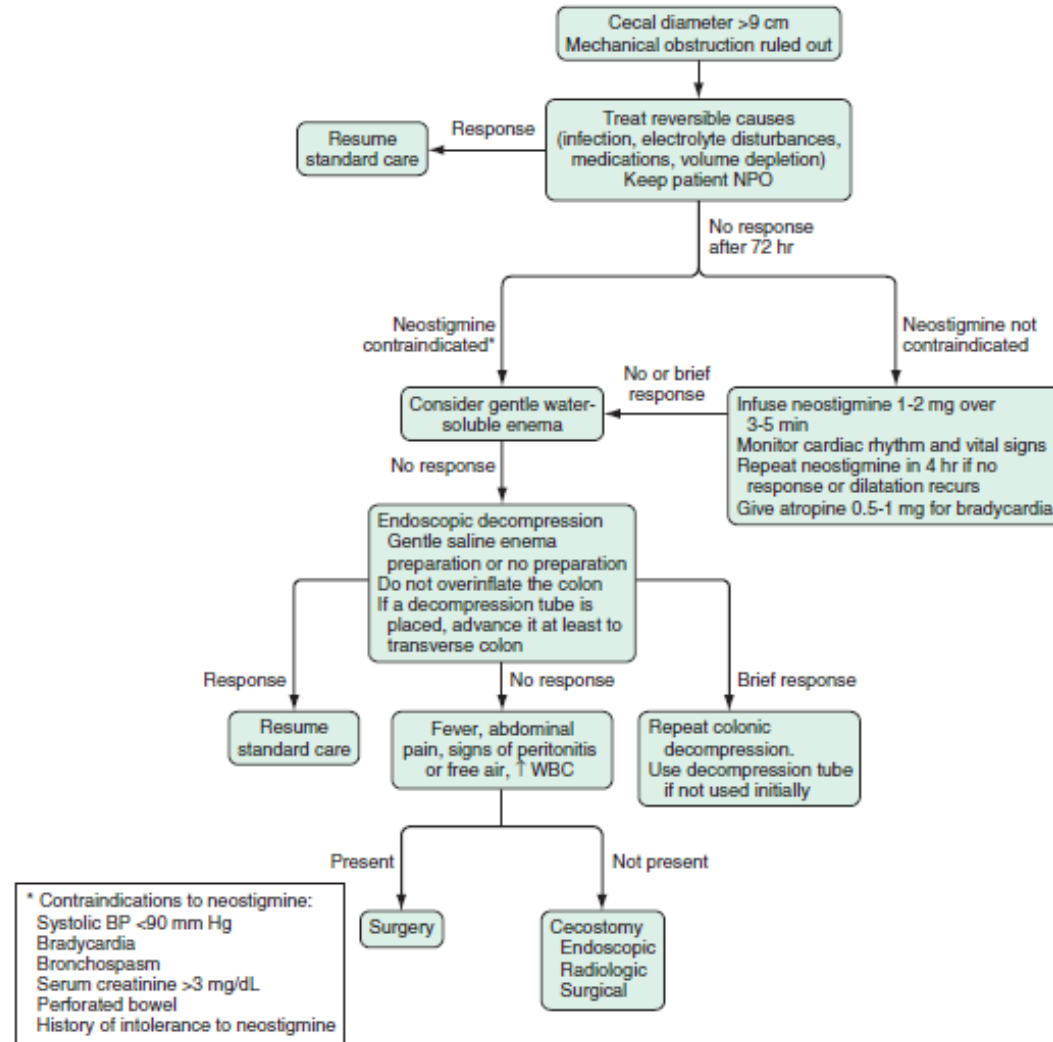


FIGURE 124-4. Algorithm for the treatment of acute colonic pseudo-obstruction.

Stercoral Ulcers of the Colon

- Pressure necrosis of the mucosa from a fecal mass (scybalum)
- Local ischemic necrosis and ulceration
- Multifactorial pathogenesis: chronic constipation
- Can lead to perforation or lower GI bleed
- Respond to antibiotics and aggressive constipation management unless there is evidence of perforation

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- American Gastroenterological Association and American College of Gastroenterology joint guidelines on Chronic Idiopathic Constipation
- *Mark Feldman, Lawrence S. Friedman, Marvin H. Sleisenger. (2002). Sleisenger & Fordtran's gastrointestinal and liver disease : pathophysiology, diagnosis, management. Philadelphia :Saunders,*



Thank you!!

