



# DIARRHEA AND CONSTIPATION

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- prevalence rates for **chronic diarrhea at 2–7%** and for **chronic constipation at 12–19%**, with **women** being affected twice as often as men.
- Although diarrhea and constipation may present as mere nuisance symptoms at one extreme, they can be **severe or life threatening** at the other. Even mild symptoms may signal a serious underlying gastrointestinal (GI) lesion, such as colorectal cancer, or systemic disorder, such as thyroid disease.

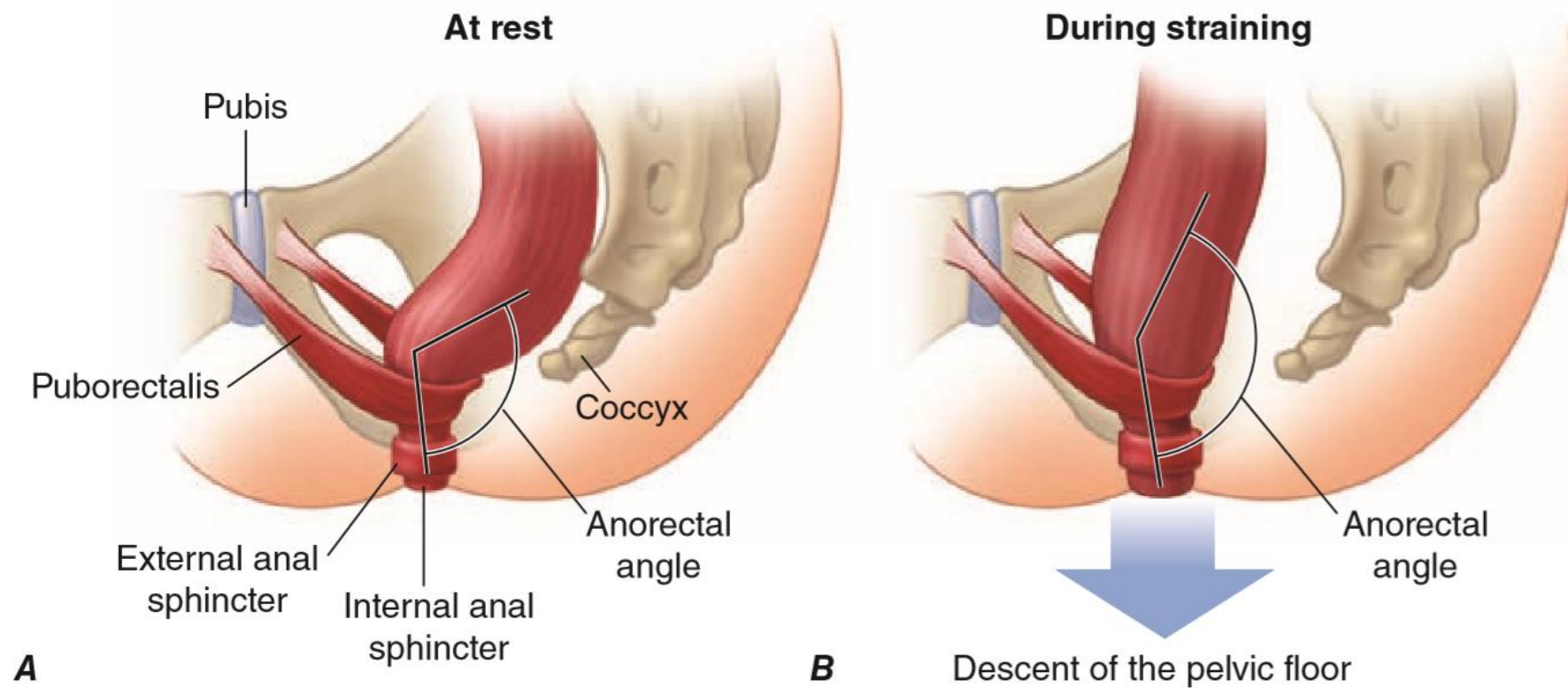
- On an average day, 9 L of fluid enter the GI tract, ~1 L of residual fluid reaches the colon, and the stool excretion of fluid constitutes about 0.2 L/d.
- The colon has a large capacitance and functional reserve and may recover up to four times its usual volume of 0.8 L/d, provided the rate of flow permits reabsorption to occur. Thus, the colon can partially compensate for excess fluid delivery to the colon that may result from intestinal absorptive or secretory disorders.

- During the fasting period, the motility of the small intestine is characterized by a cyclical event called the migrating motor complex (MMC), which serves to clear nondigestible residue from the small intestine.
- This organized, propagated series of contractions lasts, on average, 4 min, occurs every 60–90 min, and usually involves the entire small intestine.
- After food ingestion, the small intestine produces irregular, mixing contractions of relatively low amplitude, except in the distal ileum where more powerful contractions occur intermittently and empty the ileum by bolus transfers.

- The distal ileum acts as a reservoir, emptying intermittently by bolus movements. This action allows time for salvage of fluids, electrolytes, and nutrients
- Segmentation by haustra compartmentalizes the colon and facilitates mixing, retention of residue, and formation of solid stools.
- The resident microorganisms, predominantly anaerobic bacteria, in the colon are necessary for the digestion of unabsorbed carbohydrates that reach the colon

- In health, the ascending and transverse regions of colon function as reservoirs (average transit time, 15 h), and the descending colon acts as a conduit (average transit time, 3 h).

- Tonic contraction of the puborectalis muscle, which forms a sling around the rectoanal junction, is important to maintain continence; during defecation, sacral parasympathetic nerves relax this muscle, facilitating the straightening of the rectoanal angle



**FIGURE 42-1** Sagittal view of the anorectum (A) at rest and (B) during straining to defecate. Continence is



# Diarrhea

- Diarrhea is loosely defined as passage of abnormally liquid or unformed stools at an increased frequency. For adults on a typical Western diet, stool weight >200 g/d can generally be considered diarrheal.
- Diarrhea may be further defined as acute if <2 weeks, persistent if 2–4 weeks, and chronic if >4 weeks in duration.
- More than 90% of cases of acute diarrhea are caused by infectious agents; these cases are often accompanied by vomiting, fever, and abdominal pain. The remaining 10% or so are caused by medications, toxic ingestions, ischemia,

- **Pseudodiarrhea**, or the frequent passage of small volumes of stool, is often associated with rectal urgency, tenesmus, or a feeling of incomplete evacuation, and accompanies IBS or proctitis.
- **Fecal incontinence** is the involuntary discharge of rectal contents and is most often caused by neuromuscular disorders or structural anorectal problems.

**TABLE 42-2 Association Between Pathobiology of Causative Agents and Clinical Features in Acute Infectious Diarrhea**

<b>PATHOBIOLOGY/AGENTS</b>	<b>INCUBATION PERIOD</b>	<b>VOMITING</b>	<b>ABDOMINAL PAIN</b>	<b>FEVER</b>	<b>DIARRHEA</b>
Toxin producers Preformed toxin <i>Bacillus cereus</i> , <i>Staphylococcus aureus</i> , <i>Clostridium perfringens</i>	1–8 h 8–24 h	3–4+	1–2+	0–1+	3–4+, watery
Enterotoxin <i>Vibrio cholerae</i> , enterotoxigenic <i>Escherichia coli</i> , <i>Klebsiella pneumoniae</i> , <i>Aeromonas</i> species	8–72 h	2–4+	1–2+	0–1+	3–4+, watery
Enteroadherent Enteropathogenic and enteroadherent <i>E. coli</i> , <i>Giardia</i> organisms, cryptosporidiosis, helminths	1–8 d	0–1+	1–3+	0–2+	1–2+, watery, mushy
Cytotoxin producers <i>Clostridium difficile</i> Hemorrhagic <i>E. coli</i>	1–3 d 12–72 h	0–1+ 0–1+	3–4+ 3–4+	1–2+ 1–2+	1–3+, usually watery, occasionally bloody 1–3+, initially watery, quickly bloody
Invasive organisms Minimal inflammation Rotavirus and norovirus	1–3 d	1–3+	2–3+	3–4+	1–3+, watery
Variable inflammation <i>Salmonella</i> , <i>Campylobacter</i> , and <i>Aeromonas</i> species, <i>Vibrio parahaemolyticus</i> , <i>Yersinia</i>	12 h–11 d	0–3+	2–4+	3–4+	1–4+, watery or bloody
Severe inflammation <i>Shigella</i> species, enteroinvasive <i>E. coli</i> , <i>Entamoeba histolytica</i>	12 h–8 d	0–1+	3–4+	3–4+	1–2+, bloody

Source: Adapted from DW Powell, in T Yamada (ed): *Textbook of Gastroenterology and Hepatology*, 4th ed. Philadelphia, Lippincott Williams & Wilkins, 2003.

# five high-risk groups are recognized

- Travelers:

enterotoxigenic or enteroaggregative **Escherichia coli** as well as to **Campylobacter**, **Shigella**, **Aeromonas**, **norovirus**, **Coronavirus**, and **Salmonella**.

Visitors to Russia (especially St. Petersburg) may have increased risk of Giardia-associated diarrhea;

visitors to Nepal may acquire Cyclospora.

Campers, backpackers, and swimmers in wilderness areas may become infected with Giardia.

Cruise ships may be affected by outbreaks of gastroenteritis caused by agents such as norovirus.

- Consumers of certain foods:

Diarrhea closely following food consumption at a picnic, or restaurant may suggest infection with Salmonella, Campylobacter, or Shigella from chicken;

enterohemorrhagic E. coli (O157:H7) from undercooked hamburger;

Bacillus cereus from fried rice or other reheated food;

Staphylococcus aureus or Salmonella from mayonnaise or creams;

Salmonella from eggs;

Listeria from fresh or frozen uncooked foods or soft cheeses;

Vibrio species, Salmonella, or acute hepatitis A from seafood, especially if raw.

- Immunodeficient persons:

primary immunodeficiency (e.g., IgA deficiency, common variable hypogammaglobulinemia, chronic granulomatous disease) or the much more common secondary immunodeficiency states (e.g., AIDS, pharmacologic suppression).

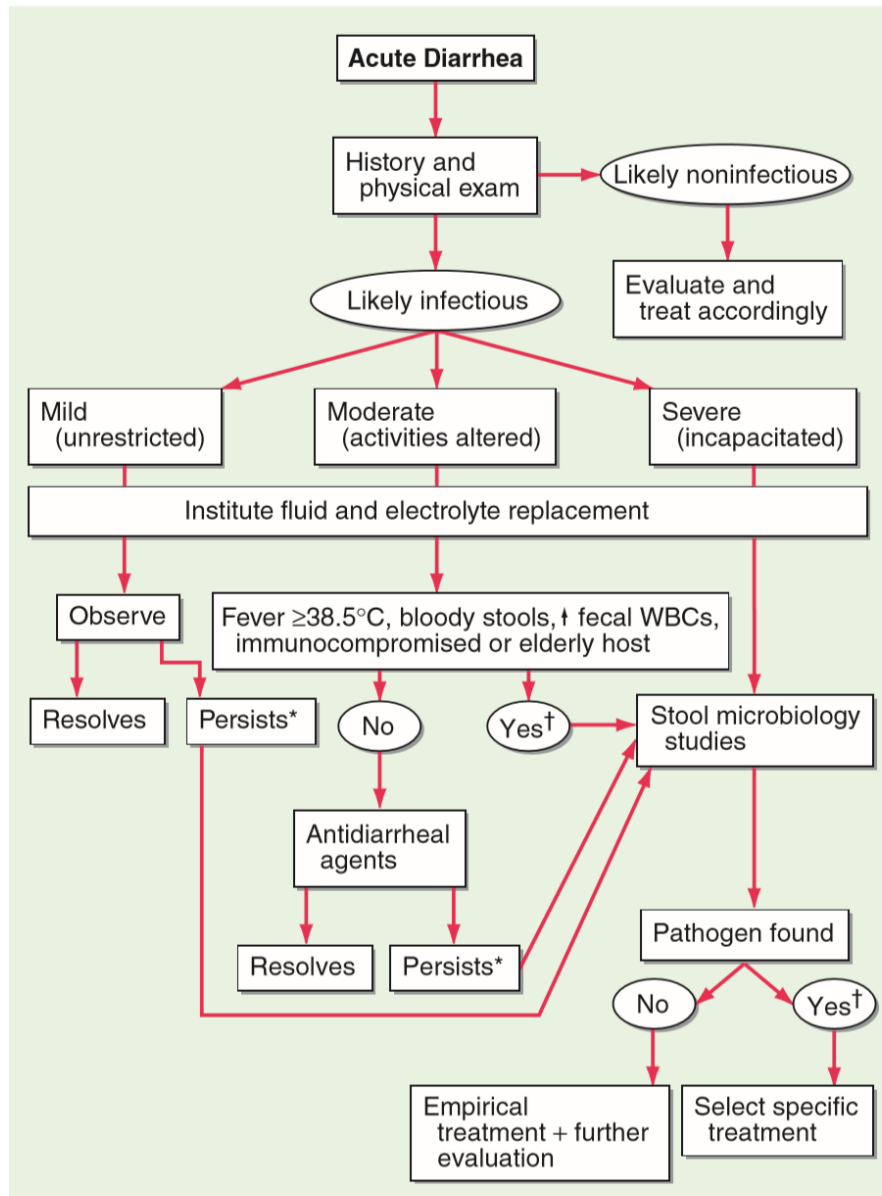
Persons with **hemochromatosis** are especially prone to invasive, even fatal, enteric infections with **Vibrio** species and **Yersinia** infections and should avoid raw fish.

- Daycare attendees and their family members:  
Shigella, Giardia, Cryptosporidium, rotavirus

- Institutionalized persons:

C. difficile can affect those with no history of antibiotic use and may be acquired in the community.





**FIGURE 42-2 Algorithm for the management of acute diarrhea.** Consider empirical treatment before evaluation with (\*) metronidazole and with (†) quinolone. WBCs, white blood cells.

**TABLE 42-3 Major Causes of Chronic Diarrhea According to Predominant Pathophysiologic Mechanism**

**Secretory Causes**

- Exogenous stimulant laxatives
- Chronic ethanol ingestion
- Other drugs and toxins
- Endogenous laxatives (dihydroxy bile acids)
- Idiopathic secretory diarrhea or bile acid diarrhea
- Certain bacterial infections
- Bowel resection, disease, or fistula (↓ absorption)
- Partial bowel obstruction or fecal impaction
- Hormone-producing tumors (carcinoid, VIPoma, medullary cancer of thyroid, mastocytosis, gastrinoma, colorectal villous adenoma)
- Addison’s disease
- Congenital electrolyte absorption defects

**Osmotic Causes**

- Osmotic laxatives ( $\text{Mg}^{2+}$ ,  $\text{PO}_4^{-3}$ ,  $\text{SO}_4^{-2}$ )
- Lactase and other disaccharide deficiencies
- Nonabsorbable carbohydrates (sorbitol, lactulose, polyethylene glycol)
- Gluten and FODMAP intolerance

**Steatorrheal Causes**

- Intraluminal maldigestion (pancreatic exocrine insufficiency, bacterial overgrowth, bariatric surgery, liver disease)
- Mucosal malabsorption (celiac sprue, Whipple’s disease, infections, abetalipoproteinemia, ischemia, drug-induced enteropathy)
- Postmucosal obstruction (1° or 2° lymphatic obstruction)

**Inflammatory Causes**

- Idiopathic inflammatory bowel disease (Crohn’s, chronic ulcerative colitis)
- Lymphocytic and collagenous colitis
- Immune-related mucosal disease (1° or 2° immunodeficiencies, food allergy, eosinophilic gastroenteritis, graft-versus-host disease)
- Infections (invasive bacteria, viruses, and parasites, Brainerd diarrhea)
- Radiation injury
- Gastrointestinal malignancies

**Dysmotile Causes**

- Irritable bowel syndrome (including postinfectious IBS)
- Visceral neuromyopathies
- Hyperthyroidism
- Drugs (prokinetic agents)
- Postvagotomy

**Factitial Causes**

- Munchausen
- Eating disorders

**Iatrogenic Causes**

- Cholecystectomy
- Ileal resection
- Bariatric surgery
- Vagotomy, fundoplication

*Abbreviation:* FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides, and polyols.

# Secretory Causes:

- Secretory diarrheas are due to derangements in fluid and electrolyte transport across the enterocolonic mucosa. They are characterized clinically by **watery, large-volume fecal outputs that are typically painless and persist with fasting**.
- Metastatic gastrointestinal **carcinoid** tumors or, rarely, primary bronchial carcinoids may produce watery diarrhea alone or as part of the carcinoid syndrome that comprises episodic flushing, wheezing, dyspnea, and right-sided valvular heart disease.
- Systemic **mastocytosis**, which may be associated with the skin lesion urticaria pigmentosa, may cause diarrhea that is either secretory and mediated by histamine or inflammatory due to intestinal infiltration by mast cells.

# Osmotic Causes

- Ingestion of magnesium-containing antacids, health supplements, or laxatives may induce osmotic diarrhea typified by a stool osmotic gap ( $>50$  mosmol/L): serum osmolarity (typically 290 mosmol/kg)  $- (2 \times [\text{fecal sodium} + \text{potassium concentration}])$ .

# Steatorrheal Causes

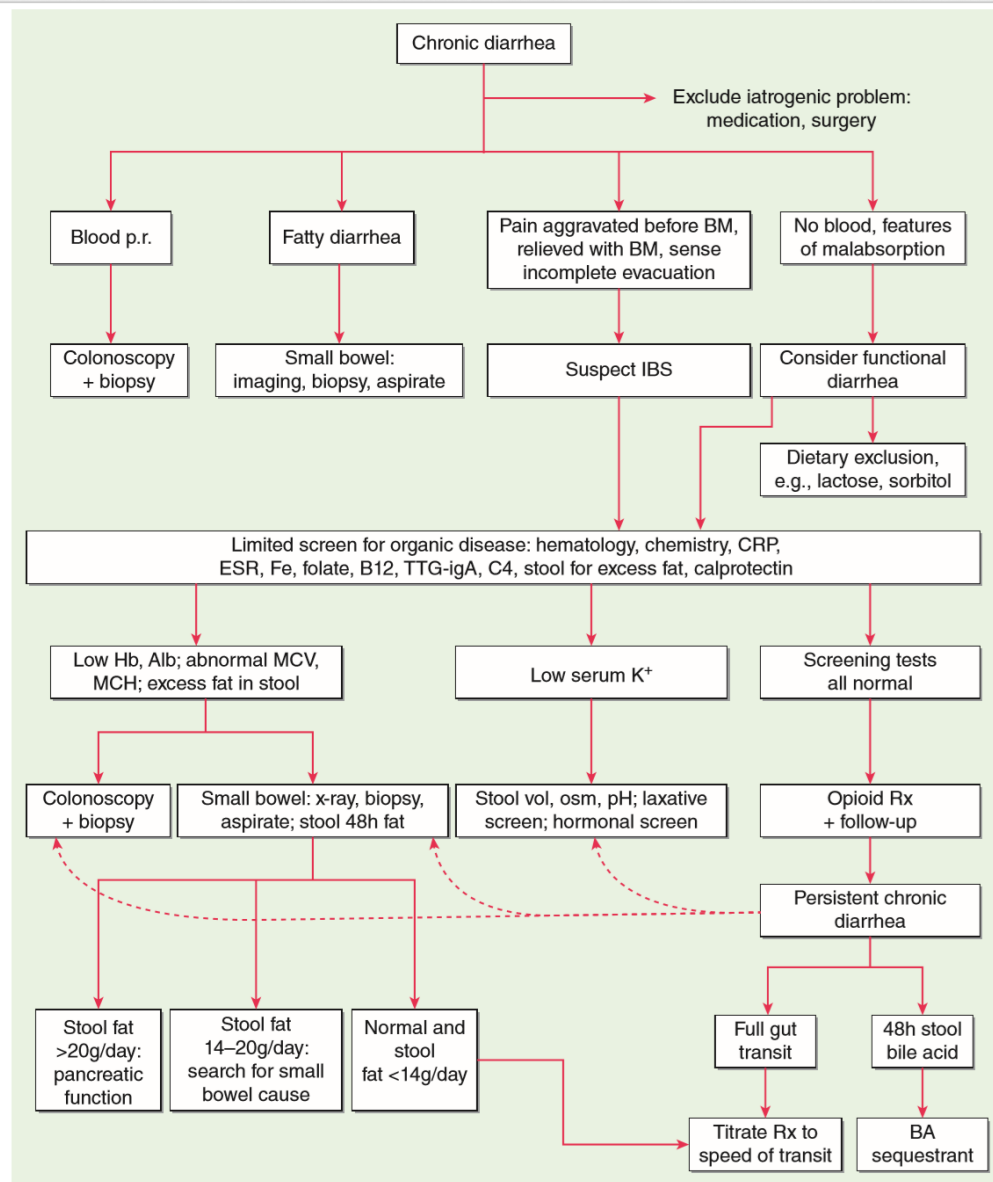
- greasy, foul-smelling, difficult-to-flush diarrhea often associated with weight loss and nutritional deficiencies
- Quantitatively, steatorrhea is defined as stool fat exceeding the normal 7 g/d; rapid-transit diarrhea may result in fecal fat up to 14 g/d; daily fecal fat averages 15–25 g with small-intestinal diseases and is often >32 g with pancreatic exocrine insufficiency. Intraluminal maldigestion, mucosal malabsorption, or lymphatic obstruction may produce steatorrhea.
- Whipple's disease, due to the bacillus *Tropheryma whipplei* and histiocytic infiltration of the small-bowel mucosa, is a less common cause of steatorrhea that most typically occurs in young or middle-aged men; it is frequently associated with arthralgias, fever, lymphadenopathy, and extreme fatigue, and it may affect the CNS and endocardium.

# Inflammatory Causes

- Inflammatory diarrheas are generally accompanied by pain, fever, bleeding, or other manifestations of inflammation.

# Dysmotility Causes

- The IBS is characterized by disturbed intestinal and colonic motor and sensory responses to various stimuli. Symptoms of stool frequency typically cease at night, alternate with periods of constipation, are accompanied by abdominal pain relieved with defecation, and rarely result in weight loss.



**FIGURE 42-3 Algorithm for management of chronic diarrhea.** Patients undergo an initial evaluation based on different symptom presentations, leading to selection of patients for imaging, biopsy analysis, and limited screens for organic diseases. Alb, albumin; BA, bile acid; BM, bowel movement; C4, 7  $\alpha$ -hydroxy-4-cholesten-3-one; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; Hb, hemoglobin; Hx, history; IBS, irritable bowel syndrome; MCH, mean corpuscular hemoglobin; MCV, mean corpuscular volume; osm, osmolality; p.r., per rectum; Rx, treatment. (Reprinted from M Camilleri, JH Sellin, KE Barrett: Pathophysiology, evaluation, and management of chronic watery diarrhea. *Gastroenterology* 152:515, 2017.)



### **TABLE 42-4 Physical Examination in Patients with Chronic Diarrhea**

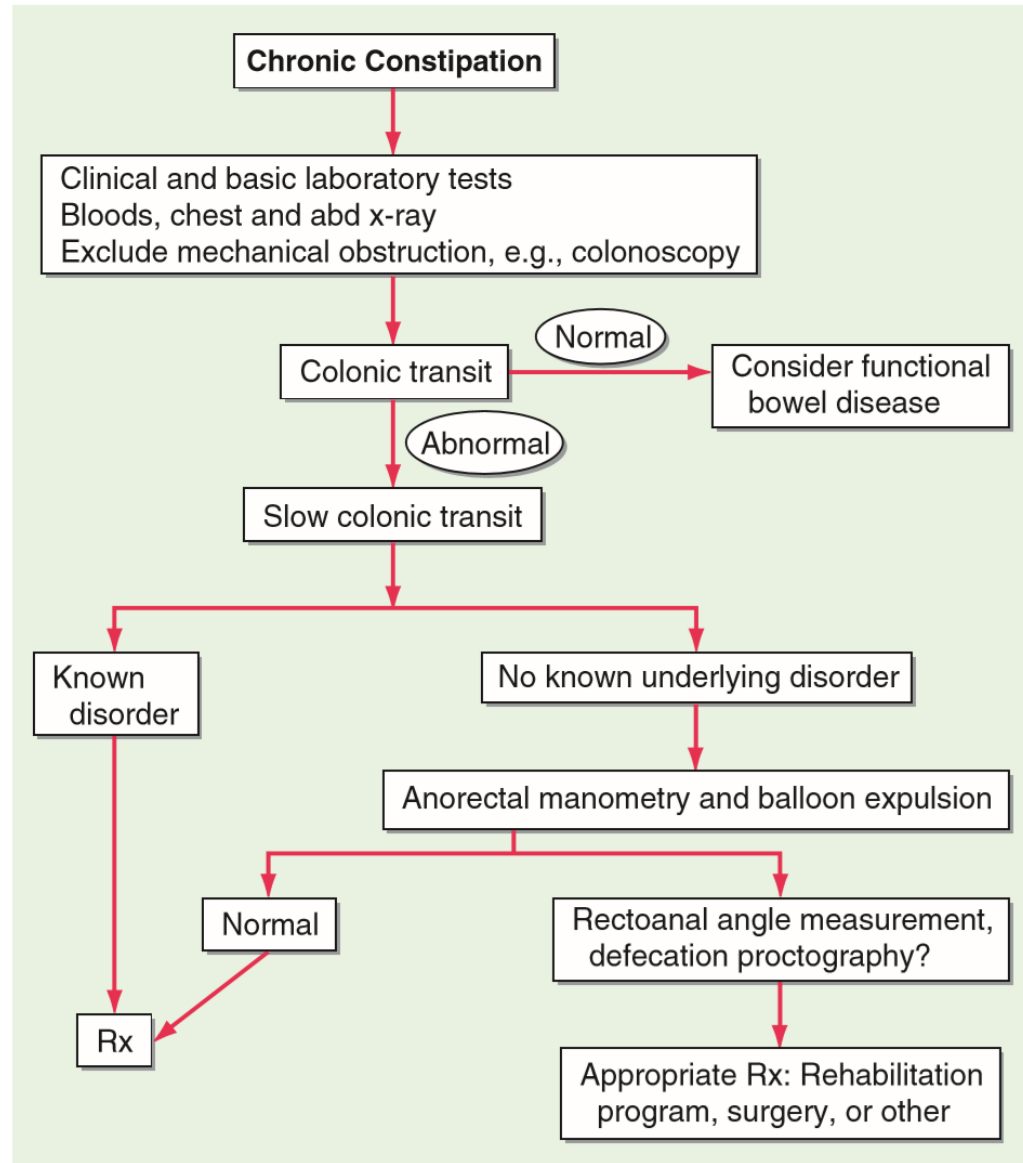
1. Are there general features to suggest malabsorption or inflammatory bowel disease (IBD) such as anemia, dermatitis herpetiformis, edema, or clubbing?
2. Are there features to suggest underlying autonomic neuropathy or collagen-vascular disease in the pupils, orthostasis, skin, hands, or joints?
3. Is there an abdominal mass or tenderness?
4. Are there any abnormalities of rectal mucosa, rectal defects, or altered anal sphincter functions?
5. Are there any mucocutaneous manifestations of systemic disease such as dermatitis herpetiformis (celiac disease), erythema nodosum (ulcerative colitis), flushing (carcinoid), or oral ulcers for IBD or celiac disease?

# constipation

- Constipation is a common complaint in clinical practice and usually refers to persistent, difficult, infrequent, or seemingly incomplete defecation.

**TABLE 42-5 Causes of Constipation in Adults**

<b>TYPES OF CONSTIPATION AND CAUSES</b>	<b>EXAMPLES</b>
<b>Recent Onset</b>	
Colonic obstruction	Neoplasm; stricture: ischemic, diverticular, inflammatory
Anal sphincter spasm	Anal fissure, painful hemorrhoids
Medications	
<b>Chronic</b>	
Irritable bowel syndrome	Constipation-predominant, alternating
Medications	Ca <sup>2+</sup> blockers, antidepressants
Colonic pseudoobstruction	Slow-transit constipation, megacolon (rare Hirschsprung's, Chagas' diseases)
Disorders of rectal evacuation	Pelvic floor dysfunction; anismus; descending perineum syndrome; rectal mucosal prolapse; rectocele
Endocrinopathies	Hypothyroidism, hypercalcemia, pregnancy
Psychiatric disorders	Depression, eating disorders, drugs
Neurologic disease	Parkinsonism, multiple sclerosis, spinal cord injury
Generalized muscle disease	Progressive systemic sclerosis



**FIGURE 42-4** Algorithm for the management of constipation. abd, abdominal.

- Radiopaque marker transit tests :

radiopaque markers are ingested; an abdominal flat film taken 5 days later should indicate passage of 80% of the markers out of the colon without the use of laxatives or enemas.

- Formal psychological evaluation may identify eating disorders, depression, or posttraumatic stress disorders that may respond to cognitive or other intervention and may be important in restoring quality of life to patients who might present with chronic constipation.