REVIEW ARTICLE

CURRENT CONCEPTS

Chronic Constipation

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CONSTIPATION IS A COMMON SYMPTOM AFFECTING BETWEEN 2 PERcent[1,2] and 27 percent[3] of the population in Western countries. In the United States, it results in more than 2.5 million visits to physicians, 92,000 hospitalizations,[2] and laxative sales of several hundred million dollars a year. Constipation is more prevalent in women than in men,[4] in nonwhites than in white persons,[5] in children than in adults,[6] and in elderly than in younger adults.[5] Severe constipation (e.g., bowel movements only twice a month) is seen almost exclusively in women.[4] Physical inactivity, low income, limited education, a history of sexual abuse, and symptoms of depression are all risk factors for constipation.[7]

Though the symptoms associated with constipation are often intermittent and mild, they may be chronic, difficult to treat, and debilitating. This review focuses on the evaluation and treatment of patients whose constipation is chronic and severe and does not resolve with the use of simple dietary or therapeutic measures. An understanding of the physiological processes involved in colonic transit and defecation is important for the effective management of constipation.

DEFINITION

There is no single definition of constipation. Most patients define constipation by one or more symptoms: hard stools, infrequent stools (typically fewer than three per week), the need for excessive straining, a sense of incomplete bowel evacuation, and excessive time spent on the toilet or in unsuccessful defecation.[8,9] An epidemiologic study of constipation in the United States identified it as an inability to evacuate stool completely and spontaneously three or more times per week.[1] A consensus definition of constipation (the Rome II criteria), used in current research, is shown in Table 1.[10]

CAUSES AND PATHOPHYSIOLOGY

Constipation is frequently multifactorial and can result from systemic or neurologic disorders or medications. Constipation can be classified into three broad categories: normal-transit constipation, slow-transit constipation, and disorders of defecatory or rectal evacuation. More than one mechanism may contribute to constipation in a patient. In a study of more than 1000 patients with chronic constipation, normal transit through the colon was the most prevalent form (occurring in 59 percent of the patients), followed by defecatory disorders (25 percent), slow transit (13 percent), and a combination of defecatory disorders and slow transit (3 percent).[6]

Normal-Transit Constipation

Normal-transit constipation (or "functional" constipation) is the most common form of constipation that clinicians see. In patients with this disorder, stool traverses at a normal rate through the colon and the stool frequency is normal, yet patients believe they are constipated.[11] In this group of patients, constipation is likely to be due to a perceived difficul-

ty with evacuation or the presence of hard stools. The patients may experience bloating and abdominal pain or discomfort, and they may exhibit increased psychosocial distress[11]; some may have increased rectal compliance, reduced rectal sensation, or both.[12] Symptoms of constipation typically respond to therapy with dietary fiber alone or with the addition of an osmotic laxative.[13] Lack of a response to these therapies may reflect a disturbance of evacuation or transit that requires further management.

Defecatory Disorders

Defecatory disorders are most commonly due to dysfunction of the pelvic floor or anal sphincter. Other terms used to describe defecatory disorders include anismus, pelvic-floor dyssynergia, paradoxical pelvic-floor contraction, obstructed constipation, functional rectosigmoid obstruction, the spastic pelvic-floor syndrome, and functional fecal retention in childhood. Functional fecal retention in children may result in secondary encopresis[14] due to leakage of liquid stool around impacted stool, which can lead to an initial misdiagnosis of diarrhea.

Prolonged avoidance of the pain associated with either the passage of a large, hard stool or an anal fissure or hemorrhoid may result in defecatory disorders.[15] Structural abnormalities, such as rectal intussusception, rectocele, obstructing sigmoidocele, and excessive perineal descent, are less common causes of defecatory disorders. Some patients have a history of sexual or physical abuse or an eating disorder. Failure of the rectum to empty effectively may be due to an inability to coordinate the abdominal, rectoanal, and pelvic-floor muscles during defecation.[16,17] These dysfunctions can be identified clinically and with the use of defecography as reduced descent of the perineum (less than 1 cm) and a reduced change in the anorectal angle (usually less than 15 degrees) during simulation of straining to defecate (Fig. 1). Ignoring or suppressing the urge to defecate may contribute to the development of mild constipation[18] before the evacuation disorder becomes severe.

Slow-Transit Constipation

Slow-transit constipation occurs most commonly[19] in young women who have infrequent bowel movements (once a week or fewer). The condition often starts at puberty. Associated symptoms are an infrequent urge to defecate, bloating, and abdominal pain or discomfort.

In patients with a minimal delay in colonic trans-

Table 1. Rome II Criteria for Constipation.
Adults
Two or more of the following for at least 12 weeks (not necessarily consecutive) in the preceding 12 months:
Straining during >25% of bowel movements
Lumpy or hard stools for >25% of bowel movements
Sensation of incomplete evacuation for >25% of bowel movements
Sensation of anorectal blockage for >25% of bowel movements
Manual maneuvers to facilitate >25% of bowel movements (e.g., digital evacuation or support of the pelvic floor)
<3 Bowel movements per week
Loose stools not present, and insufficient criteria for irritable bowel syndrome met[10]
Infants and children
Pebble-like, hard stools for a majority of bowel movements for at least 2 weeks
Firm stools ≤2 times per week for at least 2 weeks
No evidence of structural, endocrine, or metabolic disease

it, dietary and cultural factors contribute to symptoms. In these patients, a high-fiber diet may increase stool weight, decrease colon-transit time, and relieve constipation. Patients with more severe slow-transit constipation have a poor response to dietary fiber[13,19] and laxatives. Such patients have more delayed emptying of the proximal colon[20] and fewer high-amplitude peristaltic contractions after meals, which normally induce movement of content through the colon. Colonic inertia, a related condition, is characterized by slow colonic transit and the lack of an increase in motor activity after meals or after the administration of bisacodyl,[21] cholinergic agents, or anticholinesterases such as neostigmine.[22]

Histopathological studies in patients with slow-transit constipation have shown alterations in the number of myenteric plexus neurons expressing the excitatory neurotransmitter substance P,[23] abnormalities in the inhibitory transmitters vasoactive intestinal peptide and nitric oxide,[24] and a reduction in the number of interstitial cells of Cajal,[25] which are thought to regulate gastrointestinal motility.

Hirschsprung's disease is an extreme form of slow-transit constipation with similar enteric neuropathological features. In Hirschsprung's disease, ganglion cells in the distal bowel are absent, a result of an arrest in the caudal migration of neural-crest cells through the gut during embryonic development; the bowel narrows at the area that lacks ganglion cells. Though most patients with this disorder
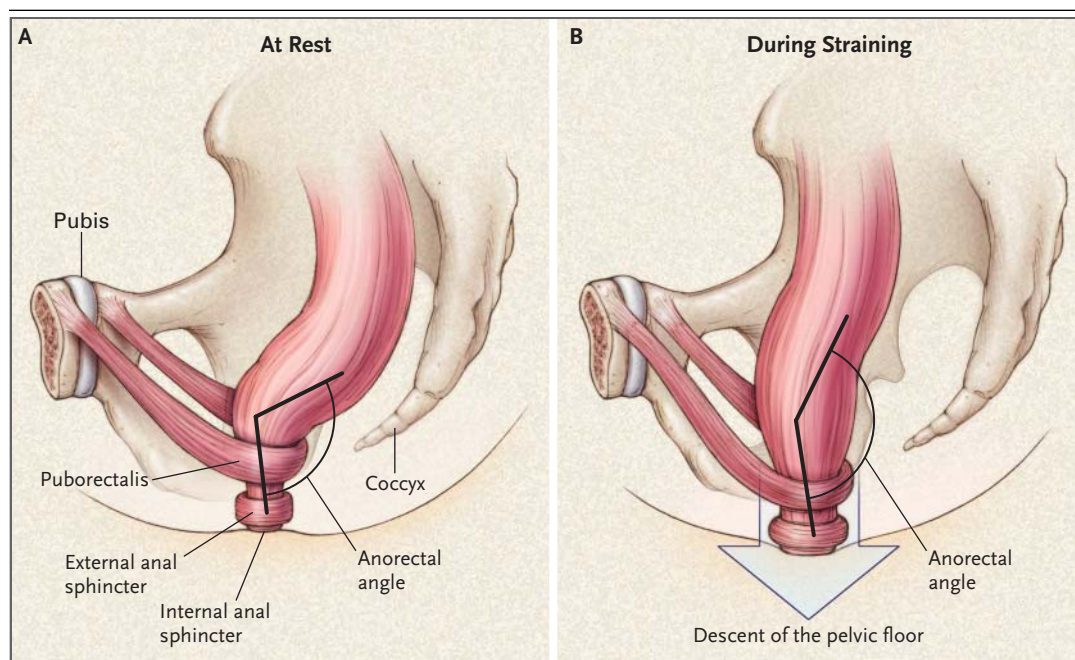
Figure 1. Sagittal View of the Anorectum at Rest (Panel A) and during Straining to Defecate (Panel B).

Continence is maintained by normal rectal sensation and tonic contraction of the internal anal sphincter and the puborectalis muscle, which wraps around the anorectum, maintaining an anorectal angle between 80 and 110 degrees. During defecation, the pelvic-floor muscles (including the puborectalis) relax, allowing the anorectal angle to straighten by at least 15 degrees, and the perineum descends by 1.0 to 3.5 cm. The external anal sphincter also relaxes and reduces pressure on the anal canal.

present in infancy or early childhood, some patients with a relatively short segment of colon involved do not have symptoms until later in life.[26] Hirschsprung's disease is associated with mutations in the *RET* proto-oncogene or the gene for the endothelin-B receptor.[27]

CLINICAL EVALUATION

HISTORY AND PHYSICAL EXAMINATION

A thorough history taking and physical examination can rule out most secondary causes of constipation. An assessment of stool form can be used to estimate the extremes of colonic transit time, since very loose or hard stools are correlated with rapid or slow colonic transit, respectively.[28]

A careful rectal examination should be performed in every patient with constipation and is often the most revealing part of the clinical evaluation. First, the perianal area should be examined for scars, fistulas, fissures, and the presence of external hemorrhoids. Next, the clinician should observe the perineum with the patient at rest and then while the

patient is bearing down, in order to determine the extent of perineal descent, which is normally between 1.0 and 3.5 cm (Fig. 1). Reduced descent may indicate an inability to relax the pelvic-floor muscles during defecation. Excessive perineal descent (below the plane of the ischial tuberosities or exceeding 3.5 cm) may indicate laxity of the perineum, which usually results from childbirth or many years of excessive straining during defecation and may lead to incomplete evacuation. Eventually, the stretching of the pelvic floor associated with excessive descent may injure the sacral nerves, reducing rectal sensation and resulting in incontinence.[29]

Finally, a digital examination of the rectum should be performed to determine whether fecal impaction, anal stricture, or rectal masses are present. A patulous anal sphincter may suggest trauma or a neurologic disorder as the cause of impaired sphincter function. Other important functions that should be assessed during the digital rectal examination are summarized in Table 2. Difficulty inserting the finger into the anal canal or the inability to do so suggests elevated pressure of the anal sphincter at rest

or anal stricture. Tenderness of the posterior aspect of the rectum may suggest a spasm of the pelvic floor.

LABORATORY TESTS

Laboratory tests that may be useful in patients with constipation include thyroid-function tests; measurements of calcium, glucose, and electrolytes; a complete blood count; and urinalysis. A complete examination of the colon is required to exclude the possibility of structural diseases such as colon cancer when there are so-called alarm symptoms (e.g., a new onset or worsening of constipation, blood in the stools, weight loss, fevers, anorexia, nausea, vomiting, or a family history of inflammatory bowel disease or colon cancer). In patients without alarm symptoms who are younger than 50 years of age, sigmoidoscopy is sufficient. However, all adults older than 50 who present with constipation should undergo colonoscopy or both sigmoidoscopy and barium-enema examination to screen for colorectal cancer.[30]

PHYSIOLOGICAL EXAMINATION

Physiological testing is necessary only in patients with refractory symptoms who do not have a secondary cause of constipation or in whom a trial of a high-fiber diet and laxatives was not effective. In patients with physical findings or symptoms suggestive of a defecatory disorder, the initial physiological tests to consider are anorectal manometry and balloon expulsion. Defecography may be considered if the results of these tests are equivocal or if there is a clinical suspicion of a structural abnormality in the rectum that impedes defecation (e.g., rectal prolapse). Delayed colonic transit can result from a defecatory disorder, and measurement of the colonic transit time should therefore be considered after the underlying pelvic-floor dysfunction has been corrected.

In patients without clinical features that suggest a defecatory disorder, the initial physiological test to consider is measurement of the colonic transit time, to distinguish slow-transit constipation from normal-transit constipation. The anorectal manometry and balloon-expulsion tests should be considered for patients who do not have a response to treatment with fiber and laxatives.

Colonic-Transit Testing

The colonic transit time is normally less than 72 hours. It is measured by performing abdominal ra-

Table 2. Diagnostic Findings in Patients with Defecatory Disorders.
History
Prolonged straining to expel stool
Unusual postures on the toilet to facilitate stool expulsion
Support of the perineum, digitation of rectum, or posterior vaginal pressure to facilitate rectal emptying
Inability to expel enema fluid
Constipation after subtotal colectomy for constipation
Rectal examination (with patient in left lateral position)
Inspection
Anus pulled forward while the patient is bearing down
Anal verge descends <1.0 cm or >3.5 cm (or beyond the ischial tuberosities) while the patient is bearing down
Perineum balloons down while the patient is bearing down, and rectal mucosa partially prolapses through anal canal
Palpation
High anal-sphincter tone at rest
Anal-sphincter pressure during voluntary contraction is only slightly higher than tone at rest
Perineum and examining finger descend <1.0 cm or >3.5 cm while patient simulates straining during defecation
Puborectalis muscle is tender on 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
Anorectal manometry and balloon expulsion (with patient in left lateral position)
Average tone of anal sphincter at rest, >80 cm water (or >60 mm Hg)
Average pressure of anal sphincter during contraction, >240 cm water (or >180 mm Hg)
Failure to expel balloon

diography 120 hours after the patient has ingested radiopaque markers in a gelatin capsule (such as Sitz-Mark, Konsyl Pharmaceuticals).[31] Before the study, the patient should be on a high-fiber diet but should not take laxatives, enemas, or medications that may affect bowel function. Retention of more than 20 percent of the markers indicates prolonged transit. If the markers are retained exclusively in the lower left colon and rectum, the patient may have a defecatory disorder, although retention of markers throughout the colon does not rule out a defecatory disorder.

Anorectal Manometry

Anorectal manometry provides several important measurements: the pressure of the anal sphincter at rest (predominantly the internal anal sphincter) and the maximal voluntary contraction of the exter-

Table 3. Medications Commonly Used for Constipation.

Medication	Maximal Recommended Dose	Comments
Bulk laxative		Increases colonic residue, stimulating peristalsis
Psyllium (Metamucil, Perdiem, Fiberall)	Titrate up to ~20 g	Natural fiber that undergoes bacterial degradation, which may contribute to bloating and flatus; should be taken with plenty of water to avoid intestinal obstruction; allergic reactions such as anaphylaxis and asthma are rare
Methylcellulose (Citrucel)	Titrate up to ~20 g	Semisynthetic cellulose fiber that is relatively resistant to colonic bacterial degradation
Polycarbophil (Fibercon, Equalactin, Konsyl)	Titrate up to ~20 g	Synthetic fiber of polymer of acrylic acid, resistant to bacterial degradation
Osmotic laxative		Draws water into the intestines along osmotic gradient

Saline laxatives
  Magnesium hydroxide (Phillips' Milk of Magnesia)
  Magnesium citrate (Evac-Q-Mag)
  Sodium phosphate (Fleet Enema, Fleet Phospho-Soda, Visicol)
	15–30 ml once or twice a day
	150–300 ml as needed
	10–25 ml with 12 oz (360 ml) of water as needed
		A small percentage of magnesium is actively absorbed in the small intestines; hypermagnesemia can occur in patients with renal failure and in children
		Hyperphosphatemia can occur in patients with renal insufficiency; commonly used for bowel preparation before colonoscopy

Poorly absorbed sugar		
Lactulose (Cephulac, Chronulac, Duphalac)	15–30 ml once or twice a day	Synthetic disaccharide consisting of galactose and fructose linked by bond resistant to disaccharidases; not absorbed by the small intestine; undergoes bacterial fermentation in the colon with formation of short-chain fatty acids; gas and bloating are common side effects

Sugar alcohols
  Sorbitol (Cystosol)
  Mannitol
	15–30 ml once or twice a day	Poorly absorbed by intestine; undergoes bacterial fermentation

Polyethylene glycol and electrolytes (Colyte, GoLYTELY, NuLYTELY)	17–36 g once or twice a day	Organic polymers that are poorly absorbed and not metabolized by colonic bacteria and may therefore cause less bloating and cramping than other poorly absorbed sugars[40]; can be mixed with noncarbonated beverages
Polyethylene glycol 3350 (Miralax)	17–36 g once or twice a day	Does not include electrolytes and is packaged for more regular use
Stimulant laxative		Stimulates intestinal motility or secretion

Anthraquinones
  Cascara sagrada (Colamin, Sagrada-lax)
  Senna (Senokot, Ex-Lax)
	325 mg (or 5 ml) daily
	187 mg daily
		Converted by colonic bacteria to their active form; may cause melanosis coli, a benign condition that is usually reversible within 12 months after the cessation of laxative use; no definitive association between anthraquinones and colon cancer or myenteric nerve damage has been established

Castor oil (Purge, Neoloid, Emulsoil)	15–30 ml daily	Hydrolyzed by lipase in the small intestine to ricinoleic acid, which inhibits intestinal water absorption, increases mucosal permeability, and stimulates motor function through the release of neurotransmitters from mucosal enterochromaffin cells; cramping and severe diarrhea are common
Diphenylmethane derivatives Bisacodyl (Dulcolax, Correctol)	5–10 mg every night	Hydrolyzed by endogenous esterases; stimulates secretion and motility of small intestine and colon
Sodium picosulfate (Lubrilax, Sur-lax)	5–15 mg every night	Hydrolized to its active form by colonic bacterial enzymes; affects only the colon
Stool softener Docusate sodium (Colace, Regulax SS, Surfak)	100 mg twice a day	Ionic detergents soften stool by allowing water to interact more effectively with solid stool; modest fluid secretion; efficacy for treatment is not well established
Mineral oil (Fleet Mineral Oil)	5–15 ml orally every night	An emollient providing lubrication for the passage of stool; long-term use can cause malabsorption of fat-soluble vitamins and anal seepage; lipoid pneumonia can occur in patients predisposed to aspiration of liquids

Table 3. (Continued.)

Medication	Maximal Recommended Dose	Comments
Rectal enema or suppository		
Phosphate enema (Fleet Enema)	120 ml daily	Initiates evacuation by distending the rectum, softening hard stool, and topically stimulating the colonic muscle to contract[41]; hyperphosphatemia and other electrolyte abnormalities can occur if the enema is retained
Mineral-oil–retention enema (Fleet Mineral Oil Enema)	100 ml daily	
Tap-water enema	500 ml daily	
Soapsuds enema	1500 ml daily	
Glycerin bisacodyl suppository	10 mg daily	
Cholinergic agent		
Bethanechol (Urecholine)	10 mg daily	Appears to be beneficial in patients with constipation due to tricyclic antidepressants[42]; intravenous neostigmine, a cholinesterase inhibitor, is effective in decompressing the colon in patients with acute colonic pseudo-obstruction[43] but has not been evaluated in patients with chronic constipation
Miscellaneous		
Colchicine (Colsalide)	0.6 mg 3 times a day	Data for both medications are limited,[44,45] and side effects are common; therefore, they are not recommended
Misoprostol (Cytotec)	600–2400 μg daily	
Prokinetic agent		
5-HT$_4$–receptor agonists*		
Cisapride (Propulsid)†	10–20 mg 4 times a day	Stimulation of 5-HT$_4$ receptors in the intestines induces peristalsis[46]; cisapride, a substituted benzamide, had variable results in treating constipation[47]; potentially lethal cardiac dysrhythmias led to its removal from the commercial market; tegaserod, a partial 5-HT$_4$ agonist, is an aminoguanidine indole derivative of serotonin that reduces pain and bloating, increases the frequency of bowel movements, and improves their consistency in women with constipation-predominant irritable bowel syndrome[48]
Tegaserod (Zelnorm)	6 mg twice a day	

* 5-HT$_4$ denotes 5-hydroxytryptamine$_4$.
† This is available only through a limited-access program developed by Janssen Pharmaceutica and the Food and Drug Administration.

nal sphincter, the presence or absence of relaxation of the internal anal sphincter during balloon distention (the anorectal inhibitory reflex), rectal sensation, and the ability of the anal sphincters to relax during straining.[32,33] Patients with defecatory disorders commonly have inappropriate contraction of the anal sphincter at rest and while bearing down. The absence of the anorectal inhibitory reflex suggests the possibility of Hirschsprung's disease. However, in the majority of patients, lack of the reflex is due to enlargement of the rectum from retained stool and insufficient distention of the rectal wall by the distended balloon. A high anal pressure at rest and rectal pain suggest the presence of an anal fissure, or anismus, since a fissure causes a voluntary contraction by the external sphincter, which augments the resting pressure. Rectal hyposensitivity (suggested by an increase in the volume of balloon distention required to induce urgency) may suggest a neurologic disorder, but more frequently it results from increased rectal capacity due to prolonged retention of stool.

Balloon Expulsion

Balloon expulsion is a simple, office-based screening test for defecatory disorders. After insertion of the latex balloon into the rectum, 50 ml of water or air is instilled into the balloon, and the patient is asked to expel the balloon into a toilet. Inability to expel the balloon within two minutes suggests a defecatory disorder.[34] In some laboratories, balloon evacuation is performed in the left lateral position and weights are added to the rectal catheter over a pulley device to facilitate defecation. Normally, expulsion in the left lateral position can be achieved spontaneously or with the addition of less than 200 g of weight.[32]

Defecography

Defecography is performed by instilling thickened barium into the rectum. With the patient sitting on a radiolucent commode, radiographic films or videos are taken during fluoroscopy while the patient is resting, contracting the anal sphincter, and straining to defecate. This procedure is used to determine

whether complete emptying of the rectum has been achieved, to measure the anorectal angle and perineal descent, and to detect structural abnormalities that may impede defecation, such as a rectocele, internal mucosal prolapse, or intussusception.

A rectocele represents a herniation, usually of the anterior rectal wall toward the vagina, and it usually results from trauma during childbirth or episiotomy.[35] Paradoxical sphincter contraction is frequently encountered in patients with rectocele, suggesting that straining against a spastic pelvic floor may facilitate development of the rectocele. A rectocele is clinically significant only if it fills preferentially or if it fails to empty after simulated defecation.

MANAGEMENT

Increases in fluid intake and physical activity do not appear to relieve chronic constipation, except in patients who are dehydrated.[36,37] Patients with normal-transit or slow-transit constipation should increase their fiber intake to 20 to 25 g per day, either with changes in their diet or with commercial fiber supplements. Patients who do not have a response to fiber therapy should try an osmotic laxative such as milk of magnesia, sorbitol, lactulose, or polyethylene glycol. The dose of the osmotic laxative should be adjusted until soft stools are attained. Colonic stimulants, such as bisacodyl or senna derivatives, and prokinetic medications, such as tegaserod, a partial 5-hydroxytryptamine$_4$–receptor agonist, should be reserved for patients with severe constipation who do not have a response to fiber or osmotic laxatives. Surgery is rarely necessary.

Patients with defecatory disorders should undergo retraining of the evacuation process with the assistance of biofeedback.[38] Patients with severe defecatory disorders often do not have a good response to oral laxatives unless they are taken at relatively high doses, which may result in watery diarrhea and other adverse effects.

Patients with fecal impaction should have the impacted feces removed manually or, if necessary, with enemas.[39] Further episodes of fecal impaction should be prevented with sufficient fiber intake and use of laxatives to induce regular bowel movements.

FIBER

Patients' compliance with the use of fiber supplements is poor because of their side effects, which include flatulence, distention, bloating, and unpleasant taste. To improve compliance, patients should be instructed to increase their dietary fiber intake gradually to 20 to 25 g per day over a period of one to two weeks. Initially, foods rich in dietary fiber (such as fruits and vegetables) should be increased. If this approach is not effective, commercially packaged fiber supplements should be tried (Table 3).

LAXATIVES

Osmotic laxatives are poorly absorbed or nonabsorbed substances that result in the secretion of water in the intestines to maintain isotonicity with plasma (Table 3). Most osmotic laxatives take several days to work. In patients with renal insufficiency or cardiac dysfunction, osmotic laxatives may cause electrolyte and volume overload from absorption of sodium, magnesium, or phosphorus. Dehydration can occur when osmotic laxatives are overused.

Stimulant laxatives increase intestinal motility and secretions. They work within hours and may cause abdominal cramps. Although stimulant laxatives have been reported to cause a so-called cathartic colon (often described as the loss of haustration and dilatation of the colon), data do not support this theory.[23] However, melanosis coli, a brown–black pigmentation of the colonic mucosa, may develop in patients who take stimulant laxatives containing anthraquinones.[49] The pigmentation is due to the accumulation of apoptotic epithelial cells in the colon that have been phagocytosed by macrophages. Melanosis coli does not lead to the development of colon cancer[50] or other abnormalities in the colon, and if the patient stops taking the stimulant laxative, the condition decreases over time.

PROKINETIC DRUGS

Tegaserod is a colonic prokinetic agent that improves stool consistency and frequency in women with irritable bowel syndrome characterized by constipation.[48]

BIOFEEDBACK THERAPY

During biofeedback for constipation due to defecatory disorders, patients receive visual and auditory feedback, or both, on the functioning of their anal-sphincter and pelvic-floor muscles. Biofeedback can be used to train patients to relax their pelvic-floor muscles during straining and to coordinate this relaxation with abdominal maneuvers to enhance the entry of stool into the rectum. Biofeedback can be performed with anorectal electromyography or a manometry catheter. Simulated evacuation with a

balloon or silicon-filled artificial stool, called "fecom,"[51] is commonly used to emphasize normal coordination for successful defecation. Patient education and the development of a rapport between the therapist and the patient are key to successful biofeedback.[52]

A systematic review of biofeedback studies revealed an overall success rate of 67 percent, although data from controlled studies are lacking.[38] The benefits of biofeedback appear to be long-lasting. Biofeedback may be less effective for patients with the descending perineum syndrome than for patients with other defecatory disorders.[29]

BOTULINUM TYPE A TOXIN

Preliminary data[53] suggest that injection of botulinum type A toxin into the puborectalis muscle may be effective in the treatment of defecatory disorders involving spastic pelvic-floor muscles. However, since controlled trials have not yet been performed, this approach is not recommended over biofeedback, for which there is more evidence of efficacy and more clinical experience.

SURGERY

For refractory constipation, total colonic resection and ileorectostomy should be considered only if the patient does not have a defecatory disorder and only after medical therapies have failed. Colonic resection is generally reserved for patients with slow-transit constipation.

A review of 32 studies showed that between 39 percent and 100 percent of patients were satisfied after colectomy.[54] The most common complications after surgery were obstruction of the small bowel, diarrhea, and incontinence. However, the diarrhea and incontinence improved after the first year.[20] Patients with upper-gut dysmotility (e.g., gastroparesis or pseudo-obstruction) or psychological disturbances do not fare as well.[55] Preliminary reports suggest that for those in whom colonic resection is deemed appropriate, laparoscopic subtotal colectomy is as effective as laparotomy.[56]

Rectal surgery should be considered only in patients with functionally significant rectoceles and in women whose constipation is relieved with application of digital vaginal pressure to facilitate defecation.[57]

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