Medical Information

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Constipation and Laxative Abuse

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"A good reliable set of bowels is worth more to a man than any quantity of brains."[1]

—HENRY WHEELER SHAW

ELIMINATION OF DIETARY WASTE is a fundamental process in man. Learned behavior, with regard to bowel function, begins early in child development, and by adulthood one has acquired his bowel habit or elimination pattern. This is based not only on physiologic mechanisms within the gastrointestinal tract, but also "the time of day, the nature of the preceding meal, a particular toilet, and often a cigarette or other added factor . . . "[2]

There is no obvious public consensus as to what constitutes a normal bowel pattern. Thus, prompted by early experiences with toilet training, later advice from friends, and a constant barrage of advertising from the communications media, many adults feel their elimination process is somehow not normal, and thereby become "constipated." A wide variety of subjective symptoms are then ascribed to constipation, and with the easy availability of proprietary cathartics, one begins the slide to laxative abuse.

The amount of money expended for laxatives in this country has been estimated at 200 million dollars annually.[3] Thirty to 50 different drugs that aid defecation are available to the interested buyer in Palo Alto drug stores (personal survey). Lader found that 30 percent of women and 40 percent of men surveyed from a greater London population were self-medicating with laxatives.[4] In another study, 3 percent of those studied who were not and 14 percent of those who were in hospital took laxatives more than once a week.[5]

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Normal Physiology

The amount and nature of fecal material depends on physiologic mechanisms within the gut. This has been extensively reviewed in various texts[2,6] and need not be covered here. Suffice it to say that after absorption and propulsion of food have occurred in the small bowel, the resulting residue is presented to the colon where further absorption of water and electrolytes takes place. Waste material is moved toward the rectum by several types of propulsive or motor activity involving single or multiple segments of the colon.[7] Net forward movement approximates 5 cm per hour, and after meals this may increase to 10 cm an hour.

As the rectum fills and becomes distended, the defecation reflex is set off, and the desire to defecate occurs. The internal anal sphincter (smooth muscle) relaxes, abdominal and chest wall muscles contract, intra-abdominal pressure increases, and the rectum is evacuated. The external anal sphincter (striated muscle) is under voluntary control, and normally in a contracted state. If one desires to defecate, the external sphincter relaxes; however, the act may also be voluntarily inhibited, and the external sphincter remains contracted, pressure in the rectum decreases and stool is returned to the descending colon.[2,6]

Constipation

Definitions of constipation vary. Almy describes it as the passage of stools that are either excessively dry, infrequent, or of insufficient size.[2] Heffernan refers mainly to hard stools.[8] Patients seem to pick one or all of these characteristics in describing what they mean by "constipation."

One might then ask what constitutes a normal bowel pattern. Connell et al,[5] after interviewing over 1,500 Londoners of all ages, found 99 percent to have from three movements a day to three a week. Healthy male prisoners were noted to have considerable variability in the time between evacuations, the average interval being about 27 hours.[9]

Various methods have been used in defining the physiologic abnormalities present in "constipated" patients. Motility studies of the colon, using balloon catheters,[10] have shown less pressure activity

in the upper rectum of constipated than in normal persons. Recordings in the sigmoid area revealed longer activity time intervals in constipated persons under the age of 40. Waller et al[11] found that persons with constipation had little or no increase in pressure waves during meals. Wangel and Deller[12] noted one constipated patient to have a pressure gradient directed away from the anal canal, in contrast to those with diarrhea. Hinton and Lennard-Jones[13] measured gut transit time, using radiopaque insoluble pellets. Normal subjects passed the first marker within 72 hours of swallowing it, and virtually all had been passed within five days. Constipated subjects took longer by both measurements. Using colonic perfusion techniques, Devroede and Soffie[3] also found delayed transit in patients with idiopathic constipation (defined as less than 3 stools per week).

Causes of Constipation

Successful treatment of a patient with constipation depends on careful identification of the cause (obstructing lesions, systemic disorders, drugs, environment, psychogenic). A thorough history and physical examination, appropriate laboratory tests, sigmoidoscopy, and barium examination of the bowel are usually required.

Following such studies, obstructing lesions such as cancer, diverticulitis, and inflammatory bowel disease are apparent and can be dealt with. British investigators[14,15] have expressed belief that obstinate constipation can be secondary to elongated or redundant segments of colon. Parks et al[16] have described "the syndrome of the descending perineum" as yet another anatomic cause of constipation. In these patients, after years of excessive abdominal straining the perineal and pelvic muscle tone is poor, with subsequent downward bulging of the rectal mucosa and a sensation of incomplete evacuation and "constipation." The diagnosis of Hirschsprung's disease, which previously depended on biopsy and radiologic findings, has been aided by anorectal manometric studies. By using rectal balloons, it has been shown that rectal distension causes relaxation of the internal anal sphincter in normal persons and contraction in patients with Hirschsprung's disease.[17,18]

Systemic disorders that lead to chronic illness and debility may cause hard or infrequent stools owing to prolonged bed rest, poor dietary habits and weak musculature.[19] Patients with endocrine disorders such as hypothyroidism[20] and hypercalcemia[21] may complain bitterly of constipation secondary to decreased motility.

TABLE 1.—*Laxatives*

Type	*Example*
1. Stimulants	Cascara, phenolphthalein, castor oil
2. Lubricants	Mineral oil, dioctyl sodium sulfosuccinate
3. Bulk formers	Methylcellulose, psyllium seed
4. Saline cathartics ...	Magnesium sulfate, milk of magnesia

Drugs such as anticholinergics, antacids, analgesics or sedatives may cause a sluggish bowel pattern.[22]

Environmental factors can be very important relative to usual bowel function. Travel, the changing of habits previously associated with defecation, admission to the hospital and so on can lead to extreme constipation. The patient should be aided in keeping his usual pattern no matter what the circumstances. In the hospital, privacy, avoidance of strict nursing routine, exercises, and the use of a toilet or bedside commode help to avoid hardness or irregularity of stools.[19]

In my experience, the commonest cause of constipation is psychogenic. Usually, the patient has developed poor bowel habits at an early age or has neglected the defecation reflex. Laxative abuse soon follows, and finally "nothing works." Treating such patients requires considerable patience and time. One must carefully explain normal colonic and anal physiology, the need to develop a set routine for bowel function, the importance of diet,[23] and above all, the need to avoid laxative or enema abuse. The patient must be allowed to discuss his own notions of "constipation" fully so that erroneous ideas can be logically dispelled. Other causes of constipation must be ruled out. Finally, as one is weaned from a pattern of laxative abuse, various regimens using enemas, suppositories, or bulk laxatives, or combinations of these measures, may be *temporarily* required.[2,8]

Laxatives

Before discussing the clinical features of laxative abuse, a brief classification of the various laxatives should be remembered (Table 1). Detailed descriptions of the pharmacology of each type can be found in various reviews.[24-27]

In brief, the stimulants basically increase motor activity of the small or large bowel, or of both. Phillips et al,[28] using rabbit ileal loops, have shown

TABLE 2.—*Clinical Features of Laxative Abuse*

1. Factitious diarrhea	3. Osteomalacia
2. Electrolyte imbalance	4. Protein losing enteropathy
hypokalemia	5. Steatorrhea
hypocalcemia	6. Cathartic colon
hypermagnesemia	7. Liver disease

that the irritant laxatives may also work by inhibiting active sodium transport from lumen to plasma and thus lead to an osmotic cathartic effect. Laxatives of the lubricant class soften the stool and thereby promote easier evacuation. Bulk formers, by increasing residue and stool volume, stimulate bowel motility and may have an additional lubricating effect. Saline cathartics, because of poor absorption, exert an osmotic effect in the gut lumen. The resulting increased volume of water promotes intestinal motility.

Articles describing the use of cholic acid[29] and lactulose syrup[30] as laxatives have recently appeared, but the role of these agents is not yet clear.

Laxative Abuse

The chronic use of laxatives (Table 2) ultimately leads to increasing constipation and the need for even stronger purges. The need to reeducate chronic users has been discussed above. There remains, however, a much smaller group of patients who abuse laxatives to the point that serious illness ensues (Table 2). These patients may go to great lengths to hide their laxative ingestion,[30-33] despite numerous admissions to hospitals, serious symptoms and even surgical operation. Often they are subconsciously very hostile, and thus have guilt feelings which are alleviated by self punishment. Anger and confrontation tactics by the physician are of little benefit, and such patients desperately need psychiatric help.[31]

Patients with severe laxative abuse and diarrhea may present with serious electrolyte imbalance. Schwartz and Relman[34] first described this in 1953, reporting upon two female patients with hypokalemia secondary "to the overuse of laxatives." Since then, numerous other cases have been recognized and thoroughly investigated.[33-37] Fatigue, motor paralysis, cramps, and poor renal function are prominent symptoms. Low blood volume and loss of sodium in the diarrheal stools may also lead to secondary hyperaldosteronism.[33,37] Fletcher et al[38] described a patient with profound hypokalemia from the surreptitious ingestion of Ex-Lax.® At first she was believed to have Bartter's syndrome because of elevated plasma renin activity, increased aldosterone, and juxtaglomerular cell hyperplasia on renal biopsy. Profound hypocalcemia has been described both in adults[39,40] and infants[41,42] with overdoses of laxatives containing phosphate. In patients with chronic renal failure, laxatives containing magnesium may cause hypermagnesemia and clinical toxicity.[43]

Frame et al[43] reported the case of a 51-year-old woman with hypokalemia, hypocalcemia and osteomalacia after 20 years of phenolphthalein abuse. She improved after the drug was stopped. In 1968 investigators[45] at the National Institutes of Health described two patients with chronic diarrhea from laxative abuse. Both had undergone exploratory laparotomy. Clinical features were electrolyte depletion, poor renal function, steatorrhea, hypoproteinemia, and well documented protein losing enteropathy.

Another feature of the prolonged abuse of irritant cathartics may be the "cathartic colon."[46] Findings on barium study of the colon may mimic inflammatory bowel disease very closely. One may visualize constricted areas, loss of haustral markings and even changes in the terminal ileum. British pathologists have reported thickening of the muscularis mucosae, excessive submucosal fat[47] and damage to the myenteric plexus in the colonic specimens from such patients.[48]

It is now clear that the use of laxatives containing oxyphenisatin can lead to serious chronic liver disease.[49-54] The patients are indistinguishable from those with chronic active hepatitis and may have several immunologic abnormalities including a positive lupus erythematosus clot test, smooth muscle and antinuclear antibodies. When the laxative is stopped, most improve, although one death has been reported.[53]

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