

AGA Technical Review on Constipation

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The symptom of constipation is very common.^{1,2} The aisles of any drugstore confirm the impact of this problem. The goal of this technical review is to identify a rational, efficacious, and ideally cost-effective approach to the patient presenting with constipation. The perspective will be that of the practicing gastroenterologist. Constipation in children will not be specifically addressed, nor will special populations such as patients with spinal cord injury.

The background for this technical review, especially the subthemes comprising the clinical syndromes, their epidemiology, diagnosis, treatment, and their socioeconomic impacts have been subjects of recent reviews and monographs.^{1,3,4} These were supplemented by selected and focused literature searches. Our discussion of the epidemiology of constipation is based on peer-reviewed, published surveys. Estimates of the economic impact to society have been published; however, formal cost-effectiveness analyses for specific diagnostic and therapeutic algorithms have not been performed. Comparisons of diagnostic approaches, with precise estimates of specificity and sensitivities, also have not been published. Indeed, in many instances, individual diagnostic techniques have not been even standardized. Moreover, most reports of treatment have not separated clearly patients with slow-transit constipation (STC) from those with disorders of the pelvic floor. There are few well-designed clinical trials of therapy, and only one meta-analysis of

comparable studies has been published. Most evidence must, therefore, be based on clinical experience, descriptive studies, and reports of expert committees. Where possible, we indicate those studies that have tested for transit defects and pelvic floor dysfunctions.

Epidemiology of Constipation

Before addressing the question “how common is constipation?,” one must first define it, although even this fundamental issue is answerable only imperfectly. The typical medical definition of constipation emphasizes infrequent or difficult evacuation of feces,⁵ and physicians often define constipation as a bowel movement every 3 to 4 days or less.⁶ This opinion is likely based on a study of otherwise healthy people in Great Britain that found that 99% of the population had between 3 bowel movements a week and 3 bowel movements a day.⁷ However, patients often have different opinions. In a survey of young adults not seeking medical care, Sandler and Drossman⁸ found that 52% defined constipation as straining to pass fecal material, 44% felt it was the process of passing hard stools, only 32%

Abbreviations used in this paper: IBS, irritable bowel syndrome; STC, slow-transit constipation.

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Table 1. Definitions of Constipation

Diagnostic criteria for constipation
At least 12 weeks, which need not be consecutive, in the preceding 12 months of 2 or more of:
Straining in $>1/4$ defecations
Lumpy or hard stools in $>1/4$ defecations
Sensation of incomplete evacuation in $>1/4$ defecations
Sensation of anorectal obstruction/blockade in $>1/4$ defecations
Manual maneuvers to facilitate $>1/4$ defecations (e.g., digital evacuation, support of the pelvic floor) and/or <3 defecations/week
Loose stools are not present, and there are insufficient criteria for IBS

Criteria from Thompson et al.¹¹

thought it merely to be the infrequent passage of stools, and 34% thought the term referred to an inability to defecate at will. Thus, it must be recognized that self-reported constipation is just as likely to refer to straining or hard stools as it is to focus on infrequent stooling.

In an effort to introduce uniform standards to clinical research, an international panel of experts developed a consensus definition of constipation.^{9,10} Several components were included, consisting of straining, hard stools, feelings of incomplete evacuation, or 2 or fewer bowel movements per week. Debate continues as to whether patients with 2 or fewer bowel movements per week should be considered to have constipation irrespective of their response to the first 3 questions. These criteria, initially published in 1992, were recently revised (Table 1).¹¹

Recognizing that differences in the definition of constipation preclude firm conclusions, how common is it? Sonnenberg and Koch¹² reviewed the data from several nationwide surveys and estimated the prevalence at 2% of the U.S. population (approximately 4 million people). In this analysis, constipation was the most commonly reported digestive complaint. A similar result (3%) was obtained in the U.S. householder study of Drossman et al.² Everhart et al.¹³ used a different set of federal data and reported a much higher prevalence rate: 21% in women and 8% in men. Stewart et al.¹⁴ surveyed more than 10,000 subjects and estimated a prevalence of 28%, which when extrapolated to the U.S. population equals 55 million people. Other surveys have arrived at estimates varying throughout this range, 2%–28%.^{1,15–17}

What can we conclude? Constipation is common; any disorder that affects $>1\%$ of the population can surely be so designated. Exactly how common depends greatly on the definition used. Finally, it should be emphasized that all published studies have addressed only the prevalence of constipation (the proportion of the population with constipation at any point in time); data on incidence, i.e.,

the rate of development of new symptoms of constipation, are lacking.

Risk Factors for Constipation

Although absolute prevalence estimated from these studies differs widely, there is good agreement as to the risk factors for constipation.^{12,13,18–22} Most studies find that self-reported constipation is more common in women than in men and that the prevalence increases with age. In one study, although self-reported constipation and laxative use increased with age, the proportion of subjects with 2 or fewer bowel movements per week was not associated with age.²³ Constipation is associated with inactivity, low calorie intake, the number of medications being taken (which is actually independent of the profiles of their side effects), low income, and a low education level.^{12,13,18–22} Constipation has not been reported to be associated with a low intake of fiber in any study to date. However, interpretation of this point is not simple, because these data come from cross sectional studies and thus do not take into account the number of persons who increased their fiber intake as treatment for constipation. Constipation is associated with depression as well as physical and sexual abuse.²⁴ Each of these increases the risk of constipation. However, one should not assume these are causative, nor should one assume that treatments directed toward the modification of risk factors will result in improved bowel function. For example, although inactivity is associated with constipation, exercise has not clearly been shown to be an effective treatment. Clinicians may try to modify these risk factors, but need to recognize that data from clinical trials are lacking.

Economic Impact

Given the number of people who in questionnaires report constipation, it comes as no surprise that this symptom contributes significantly to the costs of health care. Sonnenberg and Koch²⁵ estimated that the condition accounted for 2.5 million physician visits per year; indeed, 1.2% of the U.S. population presented to a physician with constipation in any one year. Consultation was more common among women and increased with age. This rate of visits was stable from 1958 to 1986. Of these patients, 31% were evaluated by general or family practitioners, 20% were seen by general internists, and only 4% were referred to gastroenterologists. Nevertheless, this equals 100,000 patients referred to gastroenterologists for constipation per year. To place the 2.5 million physician visits into perspective, 150,000 people develop colon cancer and 25,000 people develop pancreatic cancer in the United States each year.²⁶ These

relative numbers highlight the problems of effectively identifying patients with colon cancer from among the multitude of patients with constipation. Moreover, they point out the potential benefits to society of a clearer approach to this symptom, such as when it does or does not warrant more extensive investigation.

Almost all (85%) physician visits for constipation result in a prescription for laxatives or cathartics.²⁵ Population-based data are lacking as to the number of tests and procedures performed for constipation in the United States. In a study of 51 patients seen in a surgical referral clinic (tertiary care), the average cost of the diagnostic evaluation was \$2752.²⁷ The largest line item was the colonoscopy, which was responsible for more than one third of the total expenditures. These investigators calculated the cost per patient who benefited by the evaluation to be \$11,697.²⁷ Certainly, society cannot afford to pay for 2.5 million people to undergo such an evaluation, because this would cost \$6.9 billion, plus the costs of any treatment. To put this figure in perspective, the Medicare program has budgeted \$500 million per year for the new screening program for colorectal cancer!

Economic analyses have suggested that screening for colon cancer is cost-effective.²⁸ Formal economic analyses of the evaluation of constipation have not been performed. Because it is unlikely that patients with constipation are at lower risk for cancer, the performance of an anatomic evaluation of the colon in constipated patients is thus likely to also be cost-effective. Constipation may, in fact, indicate a higher risk of colorectal malignancy^{28,29}; thus, exclusion of malignancy perhaps is the most cost-effective first step in approaching a patient with constipation.

To summarize these general aspects, constipation is common in the community, with prevalence estimates as high as 28%. A minority of those with constipation seek medical care, but this still accounts for 2.5 million annual visits in the United States. Most people see primary care providers and receive a prescription for laxatives. They may undergo an anatomic evaluation of the colon. The role of the gastroenterologist is to assist in identifying selected patients with constipation who might benefit from additional testing or more specific treatments. By doing this, scarce health care resources may be used most efficiently.

Clinical Features and Pathophysiology

Although physicians often focus mainly on the infrequency of bowel movements in the definition of constipation, patients have a broader set of complaints. The lower limit of normal stool frequency usually quoted

is 3 per week,⁷ and 2 or fewer stools weekly was included as one of the Rome criteria (Table 1). In this definition, frequency was only 1 of 6 prime features (including straining, hard stools, and a feeling of incomplete evacuation). It has been estimated that the symptoms encompassed by the patients' definitions are (in decreasing importance) straining, stools that are excessively hard, unproductive urges, infrequency, and a feeling of incomplete evacuation.³⁰ An adequate evaluation of the symptom must, therefore, include an informed and directed history of the specific features. What is it that constitutes "constipation," in the view of this patient? The interview must also elicit a complete list of prescription and over-the-counter drugs. Constipating side effects are widespread among common medications (Table 2); moreover, most patients who feel they are constipated will be trying to relieve symptoms, often with self-prescribed over-the-counter agents. Thus, the pattern of laxative use, and sometimes abuse, must be established if the sequence of alternating constipation and diarrhea, so common in irritable bowel syndrome (IBS),^{10,15-17} is to be recognized. In population studies, laxative use and abuse are present in 7% and 4%, respectively.^{31,32}

In addition to the usual definitions of constipation, Table 1 includes the symptoms of "evacuatory failure," and herein is a significant conceptual advance in the understanding of constipation.^{4,33-36} Two major pathophysiologies can now be identified, with a third being the coexistence of both in the same patient. STC ("colonic inertia") is thought to have as a primary defect slower than normal movement of contents from the proximal to the distal colon and rectum.^{4,36-39} In some individuals, the basis for slow transit may be dietary or

Table 2. Medications Associated With Constipation

Class	Examples
Prescription drugs	
Opiates	Morphine
Anticholinergic agents	Librax, belladonna
Tricyclic antidepressants	Amitriptyline > nortriptyline
Calcium channel blockers	Verapamil hydrochloride
Antiparkinsonian drugs	Amantadine hydrochloride
Sympathomimetics	Ephedrine, terbutaline
Antipsychotics	Chlorpromazine
Diuretics	Furosemide
Antihistamines	Diphenhydramine
Nonprescription drugs	
Antacids, especially calcium-containing	Tums
Calcium supplements	
Iron supplements	
Antidiarrheal agents	Loperamide, attapulgite
Nonsteroidal anti-inflammatory agents	Ibuprofen

even cultural.^{40–43} In others, slow colonic transit probably has a true pathophysiologic basis in abnormal colonic motility. It has been suggested that there are 2 subtypes of STC⁴⁴: (1) colonic inertia, possibly related to decreased numbers^{45,46} of high-amplitude propagated contractions. These peristaltic sequences are thought to be the mechanism for the mass movement of contents, and their absence is expressed as prolonged residence times of fecal residues in the right colon,³⁹ and (2) increased, uncoordinated motor activity in the distal colon that offers a functional barrier or resistance to normal transit.⁴⁴ This distinction requires colonic manometry for its definition, these techniques are not generally available, and are not appropriate for the bulk of patients, except in research settings.

The other major pathophysiology, pelvic floor dysfunction, features normal or slightly slowed colonic transit overall but a preferential storage of residue for prolonged periods in the rectum.^{4,33–36} In this instance, the primary failure is one of an inability to evacuate adequately contents from the rectum. This functional defect has received numerous names (“outlet obstruction,” “obstructed defecation,” “dyschezia,” “anismus,” “pelvic floor dyssynergia”); this plethora of pseudonyms for a heterogenous syndrome has complicated, and perhaps confused, what otherwise is an important conceptual step. Less well understood at this time are the putative pathophysiologies that lead to this end point, i.e., the inability to empty stools from the rectum. The simplest possible classification would subdivide evacuatory failure into the following: (1) examples of muscular hypertonicity (failure to relax or “anismus”); incomplete relaxation³⁵ or paradoxical contraction of the pelvic floor and external anal sphincters during attempted defecation does occur, although this phenomenon may be less frequent than originally proposed^{47–49}; and (2) muscular hypotonicity, sometimes with megarectum and excessive pelvic floor descent.^{50,51} These syndromes are multifactorial,⁴⁹ and some are not yet well understood. The role of excessive straining, leading to or associated with excessive perineal descent, obstetrical damage to the perineal nerve, constipation, rectal intussusception, solitary rectal ulcer syndrome, and fecal incontinence is not entirely clear.^{4,48–54}

When evaluated carefully, a proportion of patients seen at tertiary referral centers have some features of both sets of disorders.^{36,55,56} Indeed, separation of STC from disorders of evacuation as the major cause of constipation is extremely important because the primary therapeutic approaches differ significantly. Surgical series have pointed out clearly that evacuatory failure needs to be

sought and, if present, treated before any decision is made about surgical therapy for intractable constipation.^{36,55,56}

Insight into the pathogenic mechanisms of intractable constipation can be gained from referral series.^{27,36,55–57} In the largest series,³⁶ of 1000 patients referred to a tertiary center for the medical and surgical evaluation of intractable constipation, 59% had normal colonic transit (or slightly delayed only). These were likely examples of IBS with constipation; 28% had pelvic floor dysfunction (with or without slow transit), and 13% had slow transit only. An earlier examination of a similar cohort draws attention to the point that patients with significant complaints of abdominal pain were more likely to have normal or slightly delayed colonic transit, and perhaps to be more representative of IBS.⁵⁸ On the other hand, chronic constipation may represent an, as yet, unrecognized expression of a neuropathy of the colon’s enteric nervous system.⁵⁹

Clinical Evaluation

Historical features are key, and the questioning must be specific. What feature does the patient rate as most distressing? Is it infrequency per se, straining, hard stools, unsatisfied defecation, or symptoms that occur *between* infrequent bowel movements (bloating, pain, malaise)? Strong emphasis on these last characteristics suggests an underlying IBS.^{10,57}

Pelvic floor dysfunction should be suspected strongly on the basis of a careful history and physical examination. Prolonged and excessive straining before elimination are suggestive; when evacuatory defects are pronounced, soft stools and even enema fluid may be difficult to pass. The need for perineal or vaginal pressure to allow stools to be passed or direct digital evacuation of stools are even stronger clues. It is important to raise this question early, because evacuatory disorders do not respond well to standard laxative programs, and failure to recognize such a component is a frequent reason for therapeutic failure. However, although evacuatory disorders may be overrepresented in referral series, they are also common in population surveys.³¹

The current regime and bowel pattern should be recorded. How often is a “call to stool” noted? Is the call always answered? What laxatives are being used, how often, and at what dosage? Are suppositories or enemas used in addition? How often are the bowels moved, and what is the nature of the stools? Physicians and patients need to be aware that, after a complete purge, it will take several days for residue to accumulate such that a normal fecal mass will be formed. Many commonly used medications have constipation as a notable side effect (anti-

Table 3. Common Medical Conditions Associated With Constipation

Drug effects
See Table 2
Mechanical obstruction
Colon cancer
External compression from malignant lesion
Strictures: diverticular or postischemic
Rectocele (if large)
Postsurgical abnormalities
Megacolon
Anal fissure
Metabolic conditions
Diabetes mellitus
Hypothyroidism
Hypercalcemia
Hypokalemia
Hypomagnesemia
Uremia
Heavy metal poisoning
Myopathies
Amyloidosis
Scleroderma
Neuropathies
Parkinson's disease
Spinal cord injury or tumor
Cerebrovascular disease
Multiple sclerosis
Other conditions
Depression
Degenerative joint disease
Autonomic neuropathy
Cognitive impairment
Immobility
Cardiac disease

cholinergics, calcium channel blockers; see Table 2). A full record of prescription and over-the-counter medications must be obtained.

The physical examination and screening tests, if deemed appropriate, should also eliminate diseases to which constipation is secondary (Table 3). Physical findings of more direct importance are confined to the perineal/rectal examination, but these may be key:

1. In the left lateral position, with the buttocks separated, observe the descent/elevation of the perineum during simulated evacuation and retention squeeze. The perianal skin can be observed for evidence of fecal soiling and the anal reflex tested by a light pinprick or scratch.
2. During simulated defecation, the anal verge should be observed for any patulous opening (suspect neurogenic incontinence) or prolapse of anorectal mucosa.
3. The digital examination should evaluate resting tone of the sphincter segment, and its augmentation by a squeezing effort. The voluntary external sphincter will be tightened by squeezing; the

internal sphincter will not. The puborectalis muscle should be palpated and compressed between the rectal forefinger and external thumb; acute localized pain along the border of the muscle is a feature of the puborectalis spasm syndrome. Finally, the patient should be instructed to integrate the expulsory forces by requesting that she/he "expel my finger."

4. An examination should then be made to look for a rectocele, or consideration be given to gynecologic consultation.

At the conclusion of the initial clinical evaluation, it should be possible to classify tentatively the patient complaining of constipation into one (or possibly more) of the following categories:

1. IBS with constipation,^{10,57} when pain, bloating, and incomplete defecation predominate.
2. STC when pelvic floor function appears to be normal, and there is evidence of slow transit.
3. Rectal outlet obstruction (anismus/dyssynergia-failure of relaxation; or descending perineal syndrome and other flaccid disorders).
4. Combination of 2 and 3, often in conjunction with the features of IBS.
5. Organic constipation (mechanic obstruction or drug side effect; Table 3).
6. Secondary constipation (metabolic disorders; Table 3).

The degree to which some or all of the possibilities listed in Table 3 need to be sought will vary greatly. Most patients will require structural studies (flexible sigmoidoscopy plus barium enema or colonoscopy) and blood chemistries to exclude metabolic disorders. In some instances, treatment will be available for the primary disorder (hypothyroidism, hypercalcemia, rectal stricture, etc.). When not available or inadequate (e.g., scleroderma, amyloid, neurologic disease), the challenge of adequate symptomatic treatment remains (see later). In most instances at the level of the primary consultation, it will be sufficient to exclude organic and secondary constipation on clinical grounds, supplemented by selected diagnostic studies, and to treat symptomatically.

Secondary Encounters and Referral Consultations

Patients are referred for specialty consultation usually because their complaints continue despite the use of fiber supplementation and simple laxatives, and after conditions to which constipation may be secondary have

been evaluated. The gastroenterologist will then need to consider the following major issues:

1. Given the variability of patient recall, a symptom diary may be instituted.
2. Has an underlying metabolic, structural, neurologic, or iatrogenic cause been overlooked? The checklist of conditions (Table 3) can usually be completed by obtaining a focused history and performing specific aspects of the physical examination. Further laboratory and imaging studies may need to be selectively completed or repeated.
3. Constipation may be the initial manifestation of diffuse intestinal pseudo-obstruction,⁵⁹ although much more common will be an association with IBS, as "constipation-predominant IBS."^{10,57} What is it that disturbs the patient's lifestyle the most? Those with primary complaints of abdominal pain, bloating, or feelings of incomplete evacuation are most likely to fit ultimately in an "IBS subgroup." For example, patients with significant pain are more likely to have normal gastrointestinal transit than those with painless constipation.⁵⁸
4. The other major cohort to identify is those in whom the contribution of defects of expulsion is clinically significant. Indeed, the inability to adequately evacuate stools softened or liquefied by laxatives is pathognomonic of an abnormality of pelvic floor/sphincteric function.

Diagnostic Tests

These can be summarized most simply as an algorithm (Algorithm 1; see preceding Medical Position Statement). The sensitivities of these investigations *has not been* established; indeed, the details of their performances have not been well specified. Although there is general agreement as to the preferred approach,^{50,51,55,56,60-63} our recommendations represent, at this time, the views of the authors. The issue of the best diagnostic approach is compounded further, because interpretation of any single test must be guarded. It should be recognized that patient cooperation is a key voluntary component of most tests of anorectal function (e.g., expulsionary efforts, squeeze pressures). Patients may be restricted by feelings of inadequate privacy, and these voluntary components will, of necessity, vary among patients, and even for the same person at different times. Thus, the tests should be in a setting as private as possible, to reduce embarrassment and facilitate cooperation, but ideal conditions are rarely possible. We list in order of simplicity, cost, and general use, the studies referred to in

the algorithm. However, none of these has been subjected to strict evaluation of specificity and sensitivity.

Balloon Expulsion Test

This simple procedure, first described by Preston and Lennard-Jones,⁵⁵ quantifies the ability of a patient to evacuate a water-filled (usually 50 mL) balloon. It can be performed easily in conjunction with anorectal manometry and can be quantified by noting the magnitude of additional passive forces needed to expel the balloon if spontaneous evacuation is not possible.⁶⁰ Although never evaluated systematically, it is a simple, useful screening test for major dysfunctions of evacuation, and can also serve as a functional marker for biofeedback programs of pelvic floor retraining.

Defecography

Defecatory function can be measured either scintigraphically or radiographically. The scintigraphic method evaluates anorectal angulation and pelvic floor descent during evacuation, and can also quantify the evacuation of artificial stools.⁶⁰ Its advantage is simplicity and minimal radiation exposure; the disadvantage is that anatomic defects may not be as well seen as with barium defecography.^{50,61} Barium defecography can be performed in conjunction with a standard barium enema (for structural evaluation of the whole colon), and thus an anatomic/functional evaluation of defecation can be performed at the same time. Of the observations possible with these techniques, the most relevant are (1) the failure of the anorectal angle to open (i.e., become more obtuse) during defecation and (2) the degree of pelvic floor descent during defecation. Decreased descent is a component of impaired pelvic floor relaxation ("anismus"), and, conversely, excessive descent ("descending perineum syndrome") can also be a pathophysiologic mechanism of constipation. In this instance, excessive straining, internal intussusception, solitary rectal ulcers, and prolapse may also occur.⁵¹⁻⁵⁴

Colonic Transit

Rates at which fecal residue moves through the colon are important determinants of fecal form, which can be categorized from liquid, to semiformal, to pelleted stools.^{63,64} The method most commonly used to measure transit is that of radiopaque markers, first introduced by Hinton et al.⁶⁵ and subsequently refined and simplified.⁶⁶ These are inexpensive tests that are possible at any medical center (markers are available from Sitz-Mark, Konsyl Pharmaceuticals, Fort Worth, TX). The test is reproducible⁶⁷ and can be recommended for any patient in whom constipation is a major symptom.

Less widely used are radionuclide gamma scintigraphic techniques.^{64,67} Radiographic and scintigraphic methods correlate well,^{64,67} with the major advantage of scintigraphy being that only 24 or 48 hours of scanning are needed, whereas the radiopaque techniques require 5–7 days for completion.⁶⁶ Whitehead's laboratory⁶⁸ lengthened the radiopaque marker test to 5 days and reported a more precise evaluation of severely constipated patients. This may be the preferred approach. It should be remembered that rectal distention by retained stools can slow colonic transit,⁶⁹ and severely constipated patients should have laxatives and/or enemas to empty the colon before a study of transit.⁷⁰

Anorectal Manometry

The subject has been reviewed extensively by Diamant and colleagues in a technical review and medical position statement for this series of Practice Guidelines.^{62,63} Precise methodologies vary between laboratories, and local normal values need to be developed and recognized. Until a standardized methodology can be accepted, data from center to center cannot be generalized. The procedure has greatest value in (1) excluding Hirschsprung's disease by the presence of a normal rectoanal inhibitory reflex and (2) providing supportive data for clinical or physiologic suggestions of pelvic floor dysfunction. For example, high basal sphincter pressures with relatively little voluntary augmentation,⁵⁶ suggest spastic pelvic floor/sphincter dysfunction (anismus).

This review will not consider tests that are used in clinical research or that are generally not applicable to practice. These include (1) specific tests of rectal perception of distention or electrical stimuli, (2) electromyography of the external sphincter or puborectalis, and (3) pudendal nerve terminal motor latency. We agree with Diamant et al.⁶² that these studies, although of value in highly selected instances, or for research purposes, are not part of the standard armamentarium. These investigators also point out the potential role of surface electromyograms in the therapeutic mode of biofeedback. Additional details can be found in specific reviews.^{4,62,71}

Medical Management

Treatment algorithms as included in the Medical Position Statement encapsulate our suggestions, and Table 4 is an extensive listing of common laxative agents including dosages and costs.

As a beginning approach, we suggest a gradual increase in fiber intake. This can be incorporated into the diet (Table 5) or used as standardized fiber supplements (Table 4). Patients need to be instructed as to how to best

use fiber supplements. They should not expect an immediate response (as can be expected with a purgative), but should embark upon a program of several weeks' duration, decreasing or increasing the daily dose of fiber after a 7–10-day period. They should begin with 2 daily doses (AM and PM), with fluids and/or meals. They should be warned that fiber supplements usually increase gaseousness, but that the symptoms often decrease after several days.

If more treatment is needed, the next simplest program should begin with an inexpensive saline agent, such as milk of magnesia. Patients can often titrate the dose such that soft, but not liquid stools, are achieved. Only later should stimulant agents (Dulcolax) or more expensive agents such as lactulose and polyethylene glycol (PEG) be considered. In general, simple or STC should be able to be controlled by one or other of these regimes. The saline laxatives all have the same mechanism of action, osmotic retention of fluid in the gut lumen, and the choice of agent (magnesium hydroxide, magnesium sulfate, sodium phosphate, sodium sulfate, etc.) is largely arbitrary. Variations on the saline/osmotic theme with PEG-electrolyte solutions (e.g., Golytely) have no conceptual advantage, and nonabsorbable carbohydrates (lactulose, sorbitol) are often limited by their extreme potential to produce gas, by bacterial metabolism of unabsorbed carbohydrate.

In the only meta-analysis of therapeutic trials, Tramonte et al.⁷² excluded 85% of 733 reports (not controlled), 11% for other reasons, and were able to evaluate 25 different treatments in 36 randomized trials. They concluded, "Both fiber and laxatives modestly improved bowel movement frequency in adults with chronic constipation. There was inadequate evidence to establish whether fiber was superior to laxatives or one laxative class was superior to another."⁷²

Stimulant laxatives (senna, bisacodyl) have traditionally been discouraged based on the silver staining results of Barbara Smith,^{73,74} which suggested that their long-term use damaged the enteric nervous system, perhaps irreversibly. However, the silver staining method is technically quite tricky, and subsequent observations using electron microscopy and immunohistochemistry have not confirmed her conclusions.^{75,76} Neurologic damage might just as readily be the cause, not the result,⁵⁹ and reticence to condone long-term stimulants is now much less.

Cisapride is a benzodiazepine that was developed as a prokinetic directed primarily to the upper gut. It has been used extensively for the treatment of constipation also; the results are quite equivocal.^{77–80} Concerns over its safety caused it to be withdrawn from the market in

Table 4. Summary of Medications Commonly Used for Constipation

Type	Generic name	Trade name	Dosage	Side effects	Time to onset of action (h)	Cost per use (\$)	Mechanism of action
Fiber	Bran	—	1 cup/day	Bloating, flatulence, iron and calcium malabsorption	—	—	Stool bulk ↑, colonic transit time ↓, GI motility ↑
	Psyllium	Metamucil, Perdiem with fiber	1 tsp up to tid	Bloating, flatulence	—	0.10–0.30	
	Methylcellulose	Citrucel	1 tsp up to tid	Less bloating	—	0.50–1.43	
	Calcium polycarbophil	FiberCon	2–4 tablets qd	Bloating, flatulence	—	0.44–0.88	
Stool softener	Docusate sodium	Colace	100 mg bid		12–72	0.14–0.80	
Hyperosmolar agents	Sorbitol	—	15–30 mL qd or bid	Sweet tasting, transient abdominal cramps, flatulence	24–48	0.12–0.48	Nonabsorbable disaccharides metabolized by colonic bacteria into acetic and other SCFAs
	Lactulose	Chronulac	15–30 mL qd or bid	Same as sorbitol	24–48	1.14–4.56	
	PEG	Golytely, Colyte Miralax	8–32 oz qd	Incontinence due to potency	0.5–1	20 per treatment	Osmotically ↑ intraluminal fluids
	Suppository	Glycerin	Up to daily	Rectal irritation	0.25–1	0.20	Evacuation induced by local rectal stimulation
Stimulants	Bisacodyl	Dulcolax	Up to daily	Irritation	0.25–1	0.85	
	Bisacodyl	Dulcolax	10 mg suppositories up to 3 times/wk	Incontinence, hyperkalemia, abdominal cramps, rectal burning with daily use of suppository form	0.25–1	0.26–1.50	Similar to senna (see anthraquinones)
	Anthraquinones (senna, cascara)	Senokot	2 tabs qd to 4 tabs bid	Degeneration of Meissner's and Auerbach's plexus (unproven), malabsorption	8–12	0.22–0.44	Electrolyte transport altered by ↑ intraluminal fluids; myenteric plexus stimulated; motility ↑
		Perdiem (plain)	1–2 tsp qd		8–12	0.40–0.80	
		Peri-Colace	1–2 tabs qd	Abdominal cramps, dehydration, melanosis coli	8–12	0.57–1.14	
Saline laxative	Magnesium	Milk of magnesia	15–30 mL qd or bid	Magnesium toxicity, dehydration, abdominal cramps, incontinence	1–3	0.11–0.44	Fluid osmotically drawn into small bowel lumen; CCK stimulated; colon transit time ↓
		Haley's M-O (with mineral oil)	15–30 mL qd or bid		1–3	0.20–0.60	
Lubricant	Mineral oil	—	15–45 mL	Lipid pneumonia, malabsorption of fat-soluble vitamins, dehydration, incontinence	6–8	1.50	Stool lubricated
Enemas	Mineral oil retention enema	—	100–250 mL qd/rectum	Incontinence, mechanical trauma	6–8	1.86	Stool softened and lubricated
	Tap water enema	—	500 mL/rectum	Mechanical trauma	5–15 min	Labor only	Evacuation induced by distended colon; mechanical lavage
	Phosphate enema	Fleet	1 U/rectum	Accumulated damage to rectal mucosa, hyperphosphatemia, mechanical trauma	5–15 min	1.30	
	Soapsuds enema	—	1500 mL per rectum	Accumulated damage to rectal mucosa, mechanical trauma	2–15 min	2.10	

GI, gastrointestinal; tsp, teaspoon; tid, three times daily; qd, every day; bid, twice a day; SCFAs, short-chain fatty acids; CCK, cholecystokinin; PEG, polyethylene glycol.

July 2000. Other prokinetics are being developed with more selective actions on the colon, and novel agents are under study as colonic prokinetics.^{80,81} Attention has also been directed toward the use of additional pharmacologic approaches with prostaglandins⁸² because diarrhea is a common side effect of their use for other purposes. This is expensive and perhaps illogical, because it is using the side effect of a potent

drug to treat constipation. Another example, colchicine,⁸³ may be even less defensible. Although inexpensive, it is a cytotoxin that has long been used to treat gout, with a known propensity to produce diarrhea. In patients with a chronic problem such as constipation, the danger of major neuromuscular complications needs to be appreciated, especially if renal function is impaired.⁸⁴

Table 5. Content of Dietary Fiber of Common Foods

	≥4 g/serving	2 or 3 g/serving	1 g/serving		
			Fruits	Vegetables	Whole-grain products
All bran (1/3 cup)	10	Beans, baked (canned, 1/4 cup)	Apricot	Asparagus	Granola
Blackberries (3/4 cup)	4	Boysenberries (1/3 cup)			
Bran Buds	8	Bran flakes, 40%	Apple	Beans (string)	Oatmeal
Bran Chex	4	Raisin Bran	Grapefruit	Broccoli	Pasta (from whole-wheat flour)
Corn Bran	4	Ry-Krisp (3 triple crackers)	Melon	Beets	Total
Fiber One	12	Bran muffin (1 average)	Orange	Carrots	Wheat Chex
100% Bran (1/3 cup)	7	Oat bran (cooked)	Peach	Cauliflower	
Raspberries (1 cup)	5	Peas, dried (cooked, 1/3 cup)	Pear	Greens	
Wheat bran (unprocessed or miller's, 1 Tbsp)	4	Popcorn, popped (3 cups)	Pineapple		
Wheatena, cooked	4	Pumpkin (3 cups) Whole-wheat bread, roll, or bun (1 piece)			

NOTE. One serving equals 1/2 cup unless noted.

However, before these therapeutic regimens should be initiated, major decisions need to be made relating to the contribution of pelvic floor dysfunction. It must be recognized that disordered evacuation will respond poorly to more and more oral laxatives.⁸⁵ Thus, the question must be asked, is the role of impaired evacuation sufficient to justify an intensive program of pelvic floor retraining and biofeedback? Formal evaluations of biofeedback training in constipation are sparse, and important practical details of individual programs are often not stated. The subject has been reviewed recently in this series.⁶² However, the motivation of the patient and therapist, together with the frequency and intensity of the retraining program, likely contribute importantly to the chances of success. In addition to biofeedback therapists, dietitians and behavioral psychologists should participate. The results of biofeedback in children have been disappointing,⁸⁶ but intensive programs in adults can have a 75% success rate or better.⁸⁷⁻⁹⁰

Role of Surgery

Surgical Treatment of STC

The treatment of colonic inertia, when well documented and assuming failure of an aggressive and prolonged trial of laxatives, fiber, and prokinetics, is total colectomy with ileorectal anastomosis.^{36,55,56} Patients need to be told that the procedure is designed to treat the symptom of constipation (difficult and infrequent evacuation) and that other symptoms (e.g., abdominal pain and bloating) that the patient associates with constipation may not necessarily be relieved by achieving regular defecation. Colectomy is performed to the level of the sacral promontory with an anastomosis between the ter-

minal ileum and upper rectum. The presacral space is entered with careful preservation of the sympathetic nerves.^{36,55}

Ileorectostomy is more successful than ileosigmoidostomy.⁹¹ If any part of the sigmoid colon is left in place, constipation may recur, whereas an anastomosis at a level below 7-10 cm from the anal verge may result in an unacceptably high frequency of bowel movements and sometimes fecal incontinence. As with segmental resection or partial colectomy, removal of the colon with preservation of the cecum and ileocecal valve has been shown to be associated with poor results.⁹² If the cecal reservoir is maintained, dilatation follows and constipation recurs. In patients in whom a thorough physiologic evaluation has been undertaken, with demonstration of convincing evidence of colonic inertia and no evidence of outlet obstruction, prompt and sustained relief of constipation can be expected.^{55,56} Patients who continue to be constipated after ileorectostomy are likely to have abnormal pelvic floor function.^{55,56}

Surgical Treatment of Defecation Abnormalities

It seems plausible that division of the posterior fibers of the puborectalis muscle may be beneficial in patients in whom the muscle contracts paradoxically at the time of defecation. However, this appears not to be so.^{93,94} Partial division of the puborectalis either in the posterior plane or laterally has been disappointing. Dividing the inner fibers of puborectalis on either side of the midline produced symptomatic improvement in only 1 of 7 patients, whereas lateral division of the muscle produced improvement in only 3 of 15.

Descending Perineum Syndrome

Constipation also occurs in patients with “descending perineum syndrome.”⁹² Such patients strain endlessly at stool but the rectum empties incompletely. The perineum is seen to bulge well below the plane of the ischial tuberosities. This abnormal perineal descent is probably secondary to injury to the sacral nerves from either childbirth or chronic straining at stool.^{51,52} Incomplete evacuation leads to more straining, more traction on the nerves, and progressive denervation of the external anal sphincter and puborectalis. In time, this scenario leads to fecal incontinence and thereby compounds the patient’s misery. Surgery cannot correct this problem, which is best treated with biofeedback, although success is only about 50%.

Stoma

Patients sometimes request a stoma because of constipation. A stoma may be a good option, as it can be reversed. Again, careful selection of patients is essential. A colostomy allows the possibility of colonic irrigation, but a number of authors have reported unsatisfactory results because of persisting colonic inertia proximal to the site of the ostomy or a more generalized disorder of motility.

A recently described operation called a “continent colonic conduit” may be an answer for some patients.⁹⁵ The sigmoid colon can be used as a continent conduit by transection at its midpoint; then, by fashioning creation of a 3-cm longitudinal incision in the anterior wall of the distal colon 15 cm from the divided end, and by intussuscepting a 5-cm segment of colon commencing 5 cm from the transected end, a valve is created. The valve serves to prevent reflux of fecal material. The conduit is brought out onto the anterior abdominal wall, and intestinal continuity is re-established by anastomosing the proximal sigmoid colon to the upper rectum. Patients are taught to intubate and irrigate the conduit. The procedure is successful in reducing the time the patient spends evacuating rectal contents and increases the number of bowel movements. The procedure is also reversible but complex.

Thus, of the many patients complaining of constipation, only a small fraction³⁶ will benefit from surgical treatment, probably 5% of highly selected, referred population, and a minuscule proportion of the total cohort.

Conclusions

Based on the preceding review, an algorithmic approach to patients with constipation can be devised

(see Algorithms 1–3 in the preceding Medical Position Statement).

After the initial history and physical examination, it should be provisionally possible to classify patients into one of several subgroups. Standard blood tests (complete blood count, thyroid-stimulating hormone, calcium) and a colonic structural evaluation (flexible sigmoidoscopy and barium enema or colonoscopy) should be performed to rule out organic causes of the constipation. Patients with known neurologic conditions need these to be addressed. If the initial evaluation is normal or negative, an empiric trial of fiber (and/or dietary changes) can be followed by simple osmotic laxatives. Most patients will obtain symptom relief with these measures.⁴

Patients who fail to respond to this initial approach are appropriate candidates for more specialized testing. A simple, inexpensive radiopaque marker study will identify STC. Pelvic floor dysfunction needs to be excluded by performing anorectal manometry and a balloon expulsion study; if confirmed, defecography will solidify the diagnosis and evaluate anatomic defects. Patients with proven pelvic floor dysfunction, if the symptoms are severe enough, should be considered for biofeedback. However, this requires an extensive program of therapy. Rarely, the anorectal inhibitory reflex will be absent, and further evaluation for Hirschsprung’s disease is indicated.

Patients with colonic inertia should be treated with aggressive laxative programs (e.g., more saline laxatives, stimulant agents, lactulose, or PEG solutions). Truly refractory patients may be considered for surgery, although few will qualify after more extensive physiologic studies.

Many patients will have normal study results. Most will meet criteria for constipation-predominant IBS. The hope is that most of these people can be managed with laxatives and reassurance. As with other functional gastrointestinal disorders, psychological conditions need to be considered as contributing factors. Key to their adequate management is identification of the predominant symptom. Is this constipation or the associated symptoms (bloating, pain, nausea, etc.)?

Unfortunately, the clinical effectiveness and the cost-effectiveness of our algorithmic approach have not been assessed. The structural evaluation, at least in older patients, is likely cost effective on the basis of identifying colon cancer and adenomatous polyps. Laxatives, biofeedback, and surgery have all been shown to be effective in treating selected patients. Community-based physicians will likely do the evaluation sequentially, whereas tertiary centers, for patient convenience, may need to test

more simultaneously. Many of the specific points of our algorithms may be debated, and different algorithms certainly have not been compared for clinical or cost benefits. The goal of this review was to guide practicing gastroenterologists through rational and efficacious approaches to patients with constipation.

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References

- Locke GR III. The epidemiology of functional gastrointestinal disorders in north America. *Gastroenterol Clin North Am* 1996;25:1-19.
- Drossman DA, Li Z, Andruzzi E, Temple RD, Talley NJ, Thompson WG, Whitehead WE, Janssens J, Funch-Jensen P, Corazziari E, Richter JE, Koch GG. U.S. householder survey of functional gastrointestinal disorders. *Dig Dis Sci* 1993;38:1569-1580.
- Johanson JF. Constipation. In: Everhart JE, ed. *Digestive disease in the United States: epidemiology and impact*. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Institutes of Diabetes, Digestive and Kidney Disease. NIH Publication 94-1447. Washington, DC: U.S. Government Printing Office, 1994:567-593.
- Barbara L, Corinaldesi R, Gizza G, Stanghellini V, eds. *Chronic constipation*. Philadelphia: Saunders, 1996.
- Dorland's illustrated medical dictionary. Philadelphia: Saunders, 1988:375.
- Herz MJ, Kahan E, Zalevski S, Aframian R, Kuznitz D, Reichman S. Constipation: a different entity for patients and doctors. *Fam Pract* 1996;13:156-159.
- Connell AM, Hilton C, Irvine G, Lennard-Jones JE, Misiewicz JJ. Variation of bowel habit in two population samples. *BMJ* 1965;2:1095-1099.
- Sandler RS, Drossman DA. Bowel habits in young adults not seeking health care. *Dig Dis Sci* 1987;32:841-845.
- Thompson WG, Creed F, Drossman DA, Heaton KW, Mazzacca G. Functional bowel disease and functional abdominal pain. *Gastroenterol Int* 1992;5:75-91.
- Drossman DA, ed. *The functional gastrointestinal disorders*. Boston: Little, Brown, 1994:115-173.
- Thompson WG, Longstreth GF, Drossman DA, Heaton KW, Irvine EJ, Müller-Lissner SA. Functional bowel disorders and functional abdominal pain. *Gut* 1999;45(suppl 2):II43-II47.
- Sonnenberg A, Koch TR. Epidemiology of constipation in the United States. *Dis Colon Rectum* 1989;32:1-8.
- Everhart JE, Go VLW, Hohannes RS, Fitzsimmons SC, Roth HP, White LR. A longitudinal study of self-reported bowel habits in the United States. *Dig Dis Sci* 1989;34:1153-1162.
- Stewart WF, Liberman, Sandler RS, Woods MS, Sternhagen A, Chee E, Lipton RB, Farup CE. Epidemiology of constipation (EPOC) Study in the United States: Relation of clinical subtypes to socioeconomic features. *Am J Gastroenterol* 1999;94:3530-3539.
- Heaton KW, O'Donnell LJ, Braddon FE, Mountford RA, Hughes RO, Cripps PJ. Symptoms of irritable bowel syndrome in a British urban community: consulters and nonconsulters. *Gastroenterology* 1992;102:1962-1967.
- Jones R, Lydeard S. Irritable bowel syndrome in the general population. *BMJ* 1992;304:87-90.
- Talley NJ, Zinsmeister AR, Van Dyke C, Melton LJ III. Epidemiology of colonic symptoms and the irritable bowel syndrome. *Gastroenterology* 1991;101:927-934.
- Talley NJ, Fleming KC, Evans JM, O'Keefe EA, Weaver AL, Zinsmeister AR, Melton LJ. Constipation in an elderly community: a study of prevalence and potential risk factors. *Am J Gastroenterol* 1996;91:19-25.
- Campbell AJ, Busby WJ, Horwath CC. Factors associated with constipation in a community based sample of people aged 70 years and over. *J Epidemiol Community Health* 1993;47:23-26.
- Stewart RB, Moore MT, Marks RG, Hale WE. Correlates of constipation in an ambulatory elderly population. *Am J Gastroenterol* 1992;87:859-864.
- Sandler RS, Jordan MC, Shelton BJ. Demographic and dietary determinants of constipation in the US population. *Am J Public Health* 1990;80:185-189.
- Whitehead WE, Drinkwater D, Cheskin LJ, Heller BR, Schuster MM. Constipation in the elderly living at home. Definition, prevalence, and relationship to lifestyle and health status. *J Am Geriatr Soc* 1989;37:423-429.
- Harari D, Gurwitz JH, Avorn J, Bohn R, Minaker KL. Bowel habit in relation to age and gender. Findings from the National Health Interview Survey and clinical implications. *Arch Intern Med* 1996;156:315-320.
- Leroi AM, Bernier C, Watier A, Hemond M, Goupil G, Black R, Denis P, Devroede G. Prevalence of sexual abuse among patients with functional disorders of the lower gastrointestinal tract. *Int J Colorectal Dis* 1995;10:200-206.
- Sonnenberg A, Koch TR. Physician visits in the United States for constipation: 1958-1986. *Dig Dis Sci* 1989;34:606-611.
- Landis SH, Murray T, Bolden S, Wingo PA. Cancer statistics, 1998. *CA Cancer J Clin* 1998;48:6-29.
- Rantis PC Jr, Vernava AM III, Daniel GL, Longo WE. Chronic constipation—is the work-up worth the cost? *Dis Colon Rectum* 1997;40:280-286.
- Winawer SJ, Fletcher RH, Miller L, Godlee F, Stolar MH, Mulrow CD, Woolf SH, Glick SN, Ganiats TG, Bond JH, Rosen L, Zapka JG, Olsen SJ, Giardiello FM, Sisk JE, Van Antwerp R, Brown-Davis C, Marciniak DA, Mayer RJ. Colorectal cancer screening: clinical guidelines and rationale. *Gastroenterology* 1997;112:594-642.
- Ghadirian P, Maisonneuve P, Perret C, Lacroix A, Boyle P. Epidemiology of sociodemographic characteristics, lifestyle, medical history, and colon cancer: a case-control study among French Canadians in Montreal. *Cancer Detect Prev* 1998;22:396-404.
- Heaton KW, Radvan J, Cripps H, Mountford RA, Braddon FEM, Hughes AO. Defecation frequency and timing, and stool form in the general population—a prospective study. *Gut* 1992;33:818-824.
- Talley NJ, Weaver AL, Zinsmeister AR, Melton LJ. Functional constipation and outlet delay: a population-based study. *Gastroenterology* 1999;105:781-790.
- Neims DM, McNeill J, Giles TR, Todd F. Incidence of laxative abuse in community and bulimic populations: a descriptive review. *Int J Eat Disord* 1995;17:211-228.
- Sagar PM, Pemberton JH. Anorectal and pelvic floor function. Relevance of continence, incontinence, and constipation. *Gastroenterol Clin North Am* 1996;25:163-182.
- Mollen RM, Claassen At, Kuijpers JH. The evaluation and treatment of functional constipation. *Scand J Gastroenterol* 1997;223:8-17.
- Preston DM, Lennard-Jones JE. Anismus in chronic constipation. *Dig Dis Sci* 1985;30:413-418.
- Nyam DC, Pemberton JH, Ilstrup DM, Rath DM. Long-term results

- of surgery for chronic constipation. *Dis Colon Rectum* 1997;40:273-279.
37. Chaussade S, Khyari A, Roche H, Garret M, Gaudric M, Couturier D, Guerre J. Determination of total and segmental colonic transit time in constipated patients. Results in 91 patients with a new simplified method. *Dig Dis Sci* 1989;34:1168-1172.
 38. van der Sijp JRM, Kamm MA, Nightingale JMD, Britton KE, Mather SJ, Morris GP, Akkermans LMA, Lennard-Jones JE. Radioisotope determination of regional colonic transit in severe constipation: comparison with radio-opaque markers. *Gut* 1993;34:402-408.
 39. Stivland T, Camilleri M, Vassallo M, Proano M, Rath D, Brown M, Thomforde G, Pemberton J, Phillips S. Scintigraphic measurements of regional gut transit in severe idiopathic constipation. *Gastroenterology* 1991;101:107-115.
 40. Burkitt D, Painter N. Gastrointestinal transit times; stool weights and consistency; intraluminal pressures. In: Burkitt DP, Trowell HC, eds. *Refined carbohydrate foods and disease: some implications of dietary fibre*. London: Academic, 1975:69-84.
 41. Cummings JH, Bingham SA, Heaton KW, Eastwood MA. Fecal weight, colon cancer risk, and dietary intake of non-starch polysaccharides (dietary fiber). *Gastroenterology* 1992;103:1783-1789.
 42. Wyman JB, Heaton KW, Manning AP, Wicks ACB. Variability of colonic function in healthy subjects. *Gut* 1978;19:146-150.
 43. Eastwood MA, Brydon WG, Baird JD, Elton RA, Helliwell S, Smith JH, Pritchard JL. Fecal weight and composition, serum lipids, and diet among subjects 18-80 years not seeking healthcare. *Am J Clin Nutr* 1984;40:628-634.
 44. Snape WJ Jr. Role of colonic motility in guiding therapy in patients with constipation. *Dig Dis* 1997;15(suppl 1):104-111.
 45. Narducci F, Bassotti G, Gaburri M, Morelli A. Twenty-four hour manometric recording of colonic motor activity in healthy man. *Gut* 1987;28:17-25.
 46. Bassotti G, Crowell MD, Whitehead WE. Contractile activity of the human colon: lessons from 24 hour studies. *Gut* 1993;34:129-133.
 47. Jones PN, Lubowski DZ, Swash M, Henry MM. Is paradoxical contraction of the puborectalis muscle of functional importance? *Dis Colon Rectum* 1987;30:667-670.
 48. Bartolo DC. Functional obstructed defecation. *Eur J Gastroenterol Hepatol* 1994;6:971-974.
 49. Papchrysostomov MC, Smith AN. Functional obstructive defecation: what is anismus? *Eur J Gastroenterol Hepatol* 1994;6:975-981.
 50. Bartram CI, Turnbull GK, Lennard-Jones JE. Evacuation proctography: an investigation of rectal expulsion in 20 subjects without defecatory disturbance. *Gastrointest Radiol* 1988;13:72-80.
 51. Henry MM, Parks AG, Swash M. The pelvic floor musculature in the descending perineum syndrome. *Br J Surg* 1982;69:470-472.
 52. Snooks SJ, Barnes PRH, Swash M, Henry MM. Damage to the innervation of the pelvic floor innervation. *Int J Colorectal Dis* 1986;1:20-25.
 53. Parks AG, Swash M, Urich H. Sphincter denervation in ano-rectal incontinence and rectal prolapse. *Gut* 1977;18:656-665.
 54. Schweiger M, Alexander-Williams J. Solitary rectal ulcer syndrome of the rectum: its association with occult rectal prolapse. *Lancet* 1977;1:1970-1971.
 55. Pemberton JH, Rath DM, Ilstrup DM. Evaluation and surgical treatment of severe chronic constipation. *Ann Surg* 1991;214:403-413.
 56. Wexner SD, Daniel N, Jagelman DG. Colectomy for constipation: physiologic investigations is the key to success. *Dis Colon Rectum* 1991;34:851-856.
 57. Mertz H, Naliboff B, Mayer E. Physiology of refractory chronic constipation. *Am J Gastroenterol* 1999;94:609-615.
 58. Lanfranchi GA, Bazzocchi G, Brignola C, Campieri M, Labo G. Different patterns of intestinal transit time and anorectal motility in painful and painless chronic constipation. *Gut* 1984;25:1352-1357.
 59. Leon SH, Krishnamurthy S, Schuffler MD. Subtotal colectomy for severe idiopathic constipation: a follow-up study of 13 patients. *Dig Dis Sci* 1987;32:1249-1254.
 60. Pezim ME, Pemberton JH, Levin KE, Litchy WJ, Phillips SF. Parameters of anorectal and colonic motility in health and in severe constipation. *Dis Colon Rectum* 1993;36:484-491.
 61. Shorvon PJ, McHugh S, Diamant NE, Somers S, Stevenson GW. Defecography in normal volunteers: results and implications. *Gut* 1989;30:1737-1749.
 62. Diamant NE, Kamm MA, Wald A, Whitehead WE. AGA technical review on anorectal testing techniques. *Gastroenterology* 1999;116:735-754.
 63. American Gastroenterological Association medical position statement on anorectal testing techniques. *Gastroenterology* 1999;116:732-735.
 64. Degen LP, Phillips SF. How well does stool consistency reflect colonic transit? *Gut* 1996;39:109-113.
 65. Hinton JM, Lennard-Jones JE, Young AC. A new method for studying gut transit times using radiopaque markers. *Gut* 1969;10:842-847.
 66. Metcalf AM, Phillips SF, Zinsmeister AR, MacCarty RL, Beart RW, Wolff BG. A simplified assessment of segmental colonic transit. *Gastroenterology* 1987;92:40-47.
 67. Degen LP, Phillips SF. Variability of gastrointestinal transit in healthy women and men. *Gut* 1996;39:299-305.
 68. Knowles JB, Whitehead WE, Meyer KB. Reliability of a modified sitz mark study of whole gut transit time (abstr). *Gastroenterology* 1998;114:A779.
 69. Klauser AG, Volderholzer W, Heinrich C, Schinolbeck N, Muller-Lissner S. Behavioral modification of colonic function. Can constipation be learned? *Dig Dis Sci* 1990;35:1271-1275.
 70. Bergen AJ, Read NW. The effect of preliminary bowel preparation on a simple test of colonic transit in constipated subjects. *Int J Colorectal Dis* 1993;8:75-77.
 71. Wald A. Anorectum. In: Schuster MM, ed. *Atlas of gastrointestinal motility in health and disease*. Baltimore, MD: Williams & Wilkins, 1993:229-249.
 72. Tramonte SM, Brand MB, Mulrow CD, Arnato MG, O'Keefe ME, Ramirez G. Treatment of chronic constipation: a systemic review. *J Gen Intern Med* 1997;12:15-24.
 73. Smith B. Effect of irritant purgatives on the myenteric plexus in man and the mouse. *Gut* 1968;9:139-143.
 74. Smith B. Pathologic changes in the colon produced by anthraquinone laxatives. *Dis Colon Rectum* 1973;16:455-458.
 75. Dufour P, Gendre P. Ultrastructure of mouse intestinal mucosa and changes observed after long term anthraquinone administration. *Gut* 1984;25:1358-1363.
 76. Kiernan JA, Heinicke EA. Sennosides do not kill myenteric neurons in the colon of the rat or mouse. *Neuroscience* 1989;30:837-842.
 77. Madsen JL. Effects of cisapride on gastrointestinal transit in healthy humans. *Dig Dis Sci* 1990;35:1500-1504.
 78. Krevsky B, Maurer AH, Malmud LS, Risher RS. Cisapride accelerates colonic transit in constipated patients with colonic inertia. *Am J Gastroenterol* 1989;84:882-887.
 79. Muller-Lissner SA. The Bavarian Constipation Study Group treatment of chronic constipation with cisapride and placebo. *Gut* 1987;28:1033-1038.
 80. Iwanaga Y, Wen J, Thollander MS, Kost LJ, Thomforde GM, Allen RG, Phillips SF. Scintigraphic measurement of regional gastrointestinal transit in the dog. *Am J Physiol* 1998;275:G904-G910.
 81. Briejer MR, Engelen M, Jacobs J, Vlamincck K, Schuurkes JAJ. R093877 enhances defecation frequency in conscious cats (abstr). *Gastroenterology* 1997;112:A705.

82. Soffer EE, Metcalf A, Launspach J. Misoprostol is effective treatment for patients with severe chronic constipation. *Dig Dis Sci* 1994;39:929-933.
 83. Verne GN, Eaker EY, David RH, Sninsky CA. Colchicine is an effective treatment for patients with chronic constipation: an open-label trial. *Dig Dis Sci* 1997;42:1959-1963.
 84. Kuncel RW, Duncan G, Watson D, Alderson K, Rogawski MA, Peper M. Colchicine myopathy and neuropathy. *N Engl J Med* 1987;316:1562-1568.
 85. Kamm MA. Constipation. In: Nichols RJ, Dozois RR, eds. *Surgery of the colon and rectum*. New York: Churchill Livingstone, 1997: 657-670.
 86. Loening-Bauke V. Biofeedback training in children with functional constipation. A critical review. *Dig Dis Sci* 1996;41:65-71.
 87. Bleijenberg G, Kuijpers HC. Treatment of spastic pelvic floor syndrome with biofeedback. *Dis Colon Rectum* 1987;30:108-111.
 88. Weber J, Ducrotte P, Touchais JY, Roussignol C, Denis P. Biofeedback training for constipation in adults and children. *Dis Colon Rectum* 1987;30:844-846.
 89. Turnbull GK, Ritvo PG. Anal sphincter biofeedback relaxation treatment for women with intractable constipation symptoms. *Dis Colon Rectum* 1992;35:530-536.
 90. Enck P. Biofeedback training in disordered defecation: a critical review. *Dig Dis Sci* 1993;38:1953-1960.
 91. Vasilevsky CA, Nemer FD, Balcos EG, Christenson CE, Goldberg SM. Is subtotal colectomy a viable option in the management of chronic constipation? *Dis Colon Rectum* 1988;31:679-681.
 92. Sagar PM, Pemberton JH. Surgery for constipation. In: Nicholls RJ, Dozois RR, eds. *Surgery of the colon and rectum*. New York: Churchill Livingstone, 1997:671-679.
 93. Keighley MRB, Shouler D. Outlet syndrome, is there a surgical option? *J R Soc Med* 1984;77:559-563.
 94. Kamm MA, Hawley PR, Lennard-Jones JE. Lateral diversion and the puborectalis in the management of severe constipation. *Br J Surg* 1988;75:661-663.
 95. William NS, Hughes SF, Stuchfield B. Continent colonic conduit for rectal evacuation in severe constipation. *Lancet* 1994;343: 1321-1324.
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