Slow Transit Constipation: A Review of a Colonic Functional Disorder

Jared C. Frattini, M.D.¹ and Juan J. Nogueras, M.D.¹

ABSTRACT

Constipation is a common gastrointestinal complaint that can cause significant physical and psychosocial problems. It has been categorized as slow transit constipation, normal transit constipation, and obstructed defecation. Both the definition and pathophysiology of constipation are unclear, but attempts to describe each of the three types have been made. Slow transit constipation, a functional colonic disorder represents ~15 to 30% of constipated patients. The theorized etiologies are disorders of the autonomic and enteric nervous system and/or a dysfunctional neuroendocrine system. Slow transit constipation can be diagnosed with a complete history, physical exam, and a battery of specific diagnostic studies. Once the diagnosis is affirmed and medical management has failed, there are several treatment options. Biofeedback, sacral nerve stimulation, segmental colectomy, and subtotal colectomy with various anastomoses have all been used. Of those treatment options, a subtotal colectomy with ileorectal anastomosis is the most efficacious with the data to support its use.

KEYWORDS: Constipation, slow transit constipation, colonic inertia, subtotal colectomy

Objectives: After completion of this article, the reader should be familiar with the pathophysiology and surgical as well as nonsurgical treatment options for colonic inertia.

DEFINITION

Arbuthnot Lane wrote the first description of the surgical treatment for slow transit constipation in 1908.¹ Since then, an accurate definition has been elusive. One of the original and most widely accepted definitions by Drossman et al, is two or fewer bowel movements per week or straining at stool more than 25% of the time.² The Rome criteria³ (Table 1) are an attempt at a consensus definition of constipation. These criteria describe the symptoms of constipation as straining at bowel movements, lumpy/hard stools, a sensation of incomplete evacuation, a sense of anorectal blockage, less than

three bowel movements per week, and the need for manual maneuvers to aid evacuation. These criteria do not aid in the differentiation of the three major types of constipation: normal transit, slow transit, and obstructed defecation.

The terminology, slow transit constipation, was first coined in 1986,⁴ in a group of women who all displayed slow total gut transit time with a normal caliber colon in addition to a variety of other systemic symptoms. A recent review demonstrated that slow transit constipation or colonic inertia has taken on several definitions according to radiographic, scintigraphic, manometric,

¹Department of Colorectal Surgery, Cleveland Clinic Florida, Weston, Florida.

Address for correspondence and reprint requests: Juan J. Nogueras, M.D., Department of Colorectal Surgery, Cleveland Clinic Florida, 2950 Cleveland Clinic Blvd., Weston, FL 33331 (e-mail: noguerj@ccf.org).

Volvulus, Prolapse, Intussusception, and Functional Disorders; Guest Editor, Dana R. Sands, M.D.

Clin Colon Rectal Surg 2008;21:146–152. Copyright © 2008 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New York, NY 10001, USA. Tel: +1(212) 584-4662.
DOI 10.1055/s-2008-1075864. ISSN 1531-0043.

Table 1 Rome II Criteria for Constipation³

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 (digital evacuation, support of the pelvic floor) to facilitate > 25% of bowel movements
Less than 3 bowel movements per week
Loose stools are not present, and there are insufficient criteria for irritable bowel syndrome

---

and miscellaneous studies and findings.⁵ In this review, Bassotti et al defined colonic inertia as a severe functional constipation in the absence of pelvic floor dysfunction accompanied by radiographic evidence of delayed transit with physiologic absence of colonic motor activity and no response to pharmacologic stimulation during colonic motility recording.

EPIDEMIOLOGY

It is difficult to determine the prevalence of slow transit constipation. Most patients with constipation do not have transit studies performed. A U.S. household survey estimated that constipation effected 3% of the population surveyed and an estimated 1.2% of visits to a physician were made by patients with complaints of constipation.^{6,7} One report estimates slow transit constipation represents 15 to 30% of constipated patients.⁵ A retrospective review on 70 patients formally evaluated for constipation found 27% had slow transit constipation.⁸ Another study of 731 woman using subjective definitions and assessments for constipation found 37% had delayed transit.⁹

PATHOPHYSIOLOGY

Dysmotility of the colon is the cause of slow transit constipation, but the etiology and explanation of this dysmotility is poorly understood. Many theories exist in an attempt to explain constipation such as lack of fiber, autonomic neuropathy, and disorders of both the enteric nervous system and neuroendocrine system.^{10–20} The interstitial cells of Cajal, the colonic pacemaker cells, are required for normal colonic motility and aid in the transfer of signals between nerves and muscles.^{21–24} Two studies have shown that patients with slow transit constipation have a decrease in the number of these cells.^{12,21} The etiology of this decrease is still a mystery, but the data points to a vital role of these cells in colonic

motility. In addition, abnormalities in the enteric nervous system in those with slow transit constipation have been postulated. Several studies have found impaired colonic contractile responses to both laxatives and neurotransmitters.^{10,11} An electrophysiologic study demonstrated significantly weaker or absent electrical activity in the colons of subjects with colonic inertia.²⁵ High-amplitude propagated contractions, responsible for colonic mass migration, have also been found to be decreased in number and duration.^{26,27} It has also been observed that patients with slow transit constipation have other associated motility/transit disorders of the esophagus, stomach, small bowel, gall bladder, and anorectum,^{13,16,18,28} thus lending more support to the involvement of a dysfunctional enteric nervous system in slow transit constipation. The role of the neuroendocrine system in those with slow transit constipation has been investigated and the results have been conflicting. The levels of the pancreatic polypeptide hormone family (pancreatic polypeptide, peptide YY, and neuropeptide Y), serotonin, serotonin receptors, serotonin cell density, vasoactive intestinal peptide, substance P, and cholecystokinin have all been measured and found to be either elevated or decreased.^{29–31} This demonstrates that the neuroendocrine system is abnormal, resulting in slow transit constipation; however, the type of abnormality varies on an individual basis. Autonomic nervous system dysfunction has also been proposed as a cause of slow transit constipation. This is demonstrated by the development of slow transit constipation in those who have undergone pelvic surgery or have given birth.^{18–20}

WORK-UP AND DIAGNOSIS

A thorough history and physical should be obtained when evaluating a patient for constipation. Particular attention should be paid to dietary fiber intake, endocrine or metabolic abnormalities, neurological disorders, pharmacotherapy, and psychiatric disorders. The patient's specific symptoms in conjunction with physiologic testing will aid in differentiating between the various types of constipation. Some symptoms to inquire about are associated pain or blood, the presence of abdominal bloating or distention, the frequency and time between bowel movements, frequent straining, the sensation of incomplete evacuation, and the need for digital manipulation to assist a bowel movement. A valuable tool that can be used in the work-up for constipation is the Wexner constipation score³² (Table 2). The score will help ascertain the severity of constipation and will also aid in quantifying the success of any intervention performed. Findings on digital rectal exam may include anal stricture or mass, paradoxical contraction of the puborectalis, nondescent of perineum, rectocele/enterocele, or uterine/vaginal prolapse.

Table 2 Wexner Constipation Score³²

Constipation scoring system (minimum score 0; maximum score 30)			
Frequency of bowel movements	Score	Pain: Abdominal	Score
1–2 Times per 1–2 days	0	Never	0
2 Times per week	1	Rarely	1
Once per week	2	Sometimes	2
Less than once per week	3	Usually	3
Less than once per month	4	Always	4
Difficulty: Painful evacuation effort		Time: minutes in lavatory per attempt	
Never	0	Less than 5	0
Rarely	1	5–10	1
Sometimes	2	10–20	2
Usually	3	20–30	3
Always	4	More than 30	4
Completeness: Feeling incomplete evacuation		History: Duration of constipation (year)	
Never	0	0	0
Rarely	1	1–5	1
Sometimes	2	5–10	2
Usually	3	10–20	3
Always	4	More than 20	4
Failure: Unsuccessful attempts at evacuation per 24 hours		Assistance: Type of assistance	
Never	0	Without assistance	0
1–3	1	Stimulative laxatives	1
3–6	2	Digital assistance/enema	2
6–9	3		
More than 9	4		

Once a complete history and physical has been completed, laboratory studies should be obtained to aid in the diagnosis of endocrine or metabolic abnormalities. Then a colonoscopy should be performed to seek intraluminal pathology as the cause of constipation. In addition to colonoscopy a barium enema can help identify a variety of colonic abnormalities such as a redundant colon or extrinsic compression.

Then next step should be physiologic testing. The various studies used in the evaluation of constipation will be briefly mentioned here. Further details on these physiologic studies can be read about in an earlier article in this issue. The radiopaque marker method, first described by Hinton,²⁴ involves ingesting 24 markers and taking an abdominal radiograph on day 3 and 5. The distribution of the markers in the colon provides a measure of colonic transit. A normal study should have at least 80% of the markers eliminated by day 5. The scintigraphic technique involves ingesting pellets labeled with either technetium-99m or indium-111 and performing a scan to identify the distribution of signal. Scintigraphic techniques cannot only identify delayed segmental colonic transit, but can also identify delayed small bowel transit. Anal manometry is used to identify those with Hirschsprung's disease by the absence of the rectoanal inhibitory reflex. Anal sphincter electromyography is used to diagnose paradoxical contraction of the

puborectalis. Defecography can also identify paradoxical contraction of the puborectalis in addition to rectocele, enterocoele, internal intussusception, and perineal descent.

MANAGEMENT

In those patients in which slow transit constipation has been identified, medical management consisting of dietary counseling, ~25 to 30 g of fiber daily, various cathartics, and/or enemas is appropriate. In addition, pharmacologic therapy may be added to the treatment. Colchicine and misoprostol have both been shown to increase stool frequency and colonic transit.^{25,26} Erythromycin, a motilin receptor agonist, can also stimulate colonic motility.²⁷ Prucalopride and tegaserod, both 5HT₄ receptor agonists, increase colonic transit and improve symptoms in constipated patients.^{29,33,34} If both medical and pharmacological treatments fail, other treatment modalities can be used. Biofeedback, sacral nerve stimulation, and surgery are options with varying levels of success. Slow transit constipation in combination with pelvic floor dyssynergia complicates the treatment algorithm.

Biofeedback has traditionally been used and has been successful in the treatment of constipation due to pelvic floor dysfunction. It has been used in slow transit

constipation only when it is associated with pelvic floor dysfunction. There is no supporting evidence in a randomized, controlled fashion with its use in isolated slow transit constipation. Conflicting reports on the normalization of transit time after biofeedback exist.^{35,36} Emmanuel et al³⁷ reported not only improvement in symptoms, but also an increase in transit, but the follow-up period was only 5 months. Long-term success of biofeedback also has conflicting data; some report poor success of only 20 to 35% after 12 months,^{36,38} whereas others report good success of 57 to 100% over 9 to 23 months.^{39,40}

Sacral nerve stimulation has been used for many years to treat urinary incontinence.⁴¹ Fecal incontinence improved in this same patient population and thus a new indication was found.⁴² Likewise, some patients with sacral nerve stimulators had improvement in their constipation. As a result, several studies have shown that sacral nerve stimulation can not only increase pan-colonic propagating pressure waves, but can also improve the symptoms of constipation.⁴³⁻⁴⁶ As with biofeedback, there is little randomized, controlled data to support its use.⁴⁴

Most of the successful data on the treatment of slow transit constipation has been surgical. Various methods have been used including antegrade colonic enemas, subtotal colectomy with ileorectal or cecorectal anastomosis, segmental colectomy, ileal pouch anal anastomosis, and creation of a stoma. In recent years, a laparoscopic approach to many of these procedures has been attempted.

The antegrade colonic enema was first described by Malone⁴⁷ and has been used successfully in children. Its use in adult constipation has come with mixed results. A prospective study done by Rongen⁴⁸ demonstrated an improvement from one bowel movement per week to one bowel movement per day in a cohort of 12 women. Four ultimately needed a subtotal colectomy with two ending up with permanent ileostomies. In a review of 32 patients who underwent antegrade colonic enemas, 88% needed a revision and 59% needed reversal.⁴⁹ After 36 months, 47% had satisfactory function.

In 1999, Knowles⁵⁰ published a review of 32 articles written on the outcomes of surgery done for slow transit constipation. The median satisfaction/success rate was 86%, the median small bowel obstruction rate was 18%, and the median reoperative rate was 14%. The median number of bowel movements per day was 2.9. The percentage rate for incontinence was 14%, for diarrhea 14%, for recurrent constipation 9%, for pain 41%, and for stoma 5%. Outcomes based on the type of resection were better with the subtotal colectomy with ileorectal anastomosis than with either subtotal colectomy with cecorectal or ileosigmoid anastomosis.⁵⁰ Pikarsky et al⁵¹ reported a high degree of satisfaction among 30 patients who underwent subtotal colectomy

with ileorectal anastomosis after a mean follow-up of 106 months. The reoperative rate for small bowel obstruction was 10%, the mean number of bowel movements was 2.5 per day, and the rate for both continued cathartic use and the need for antidiarrheals was 6%.⁵¹ Multiple studies have reported significantly poorer outcomes after subtotal colectomy in patients with concomitant small bowel dysmotility. The most common issues were continued abdominal pain, bloating, constipation, recurrent small bowel obstruction, or postoperative diarrhea.^{50,52,53}

In a recent study by Fitzharris et al⁵⁴ the quality of life of 75 patients who underwent subtotal colectomy was evaluated. Despite an improvement in their bowel habits, their quality of life was adversely affected by continued abdominal pain, postoperative incontinence, or diarrhea. Postoperative complication rates were 17% for lysis of adhesions, 4% needing permanent ileostomy, and 7% needing anastomotic revision. There is limited data on outcomes after a laparoscopic approach to subtotal colectomy with ileorectal anastomosis. It has been shown to improve bowel function, but it can be a technically challenging procedure.^{55,56}

Subtotal colectomy with antiperistaltic cecoproctostomy has also been evaluated as a surgical option in an attempt to alleviate postoperative symptoms. Sarli describes his technique⁵⁷ and the rationale⁵⁸ to preserve the physiologic value of the ileum, ileocecal valve, and cecum. In 2007, Marchesi et al⁵⁹ reported on his series of 43 subtotal colectomies with antiperistaltic cecorectal anastomosis for slow transit constipation. Clinical outcomes were available for 22 patients and quality of life data were available for 17 patients with slow transit constipation after a mean follow-up period of 72 months. The data show that this procedure is comparable to subtotal colectomy with ileorectal anastomosis with regards to postoperative success, complications, and quality of life. Iannelli reported the feasibility of this procedure done laparoscopically on 4 patients with a mean follow-up of 4.5 years.⁶⁰ Despite the small number of patients, the outcomes were good with no postoperative obstructions, a mean of 2.5 bowel movements per day, no incontinence, and no usage of antidiarrheals.

Another approach tried in an effort to reduce the diarrhea and obstruction rates found after subtotal colectomy with ileorectal anastomosis is segmental colectomy. The theories behind this are similar to cecoproctostomy in that the remaining colon can still provide storage and absorptive capacity; in addition, a smaller resection reduces the risk of adhesions and potential small bowel obstruction.²² The reports on the outcomes after segmental colectomy are conflicting. Several studies reported very poor results after segmental resection.⁵⁰ Two studies have reported much better results.^{61,62} After 2 years of follow-up, 25 of 28 patients undergoing segmental colectomy

had continued improvement in their constipation and symptoms. The 3 patients who continued to have symptoms underwent subtotal colectomy with good results.⁶² In Lundin et al,⁶¹ 23 of 28 patients undergoing segmental colectomy had successful outcomes with an increase in median bowel movements from 1 to 7 per week with a follow-up of 50 months. The 5 patients who failed segmental colectomy had impaired rectal sensory function on preoperative evaluation, which has been found to correlate with a poor outcome after ileorectal anastomosis.^{63,64} These reports stress the critical role that a complete and adequate preoperative evaluation has on the outcome of the type of surgery performed.^{50,61,62}

Others disagree about the critical role of preoperative evaluation stating that postcolectomy, the various symptoms still persist in addition to the continued use of cathartics and the necessity for additional surgery.⁶⁵ Mollen et al⁶⁵ showed that despite an adequate preoperative evaluation, at least 48% of patients still had symptoms and 33% required further surgery. In contrast, Ripetti, et al⁶⁶ underscores the importance of the preoperative evaluation, reporting that the 15 patients who underwent surgery for slow transit constipation had improvement in the physical pain, emotional, psychological, and general health aspect of the SF-36 test. The follow-up was 38 months and all but one patient had daily bowel movements.

Restorative proctocolectomy with ileal pouch-anal anastomosis (IPAA) has been used as both a primary surgical procedure and as a salvage procedure for slow transit constipation.^{67,68} As a primary procedure, IPAA had a statistically significant improvement in quality of life as measured by the Rand 36-item health survey 1.0.⁶⁷ Out of 15 patients undergoing IPAA, only 2 patients required pouch excision and the mean number of bowel movements per day was 5. Postoperative complications included 5 patients with small bowel obstruction, one requiring operative intervention and 3 pouchvaginal fistulae, all needing endoanal advancement flaps.⁶⁷ As a salvage procedure, four of the eight

pouches were excised due to continued distension and a feeling of incomplete evacuation.⁶⁸

The treatment for coexisting slow transit constipation and paradoxical contraction of the puborectalis is ambiguous. A recent review by Wong et al²² outlines this ambiguity. Some reports describe good success when performing a subtotal colectomy first and then use biofeedback in those patients who are still experiencing obstructed defecation. On the other hand, mixed results have come from those choosing to perform biofeedback before subtotal colectomy.

CONCLUSION

Slow transit constipation can be a debilitating disease. An extensive evaluation to identify other causes of constipation must be performed. If medical management fails, other options to surgery exist in the form of biofeedback or sacral nerve stimulation. These modalities are not supported by good evidence. Patients must be informed that a successful surgical outcome cannot always be predicted from preoperative evaluation. The surgical procedure recommended and offered at our institution to our patient population is the subtotal colectomy with ileorectal anastomosis, which we often perform laparoscopically. Our algorithm can be seen in Fig. 1. As we have demonstrated, this procedure has excellent long-term outcomes (up to 10 years) and patients are quite satisfied with the results. In preoperative consultation, we educate all of our patients that this procedure is not perfect; the potential risks are an approximate 20% chance of obstruction and a 5% chance that laxatives or antidiarrheals may need to be used. Other surgical procedures have limited and equivocal data as does subtotal colectomy, but such procedures can only be done after appropriate patient selection and if the surgeon uses a sound technique, such as laparoscopic segmental colectomy or subtotal colectomy with antiperistaltic cecoproctostomy. Segmental colectomy may be a viable alternative in the future after scintigraphy becomes more widely used and accepted and

Figure 1 Algorithm for slow transit constipation.

prospective studies demonstrate outcomes equal to or better than subtotal colectomy with ileorectal anastomosis. Again, we choose not to perform these techniques until more convincing data has been published. We recommend biofeedback initially for our patients with combined slow transit constipation and pelvic floor dysfunction. If a good response to biofeedback is demonstrated, we then proceed to subtotal colectomy.

REFERENCES

- Arbutnot LW. The results of operative treatment of chronic constipation. *BMJ* 1908;i:126-130
- Drossman DA, Sandler RS, McKee DC, Lovitz AJ. Bowel patterns among patients not seeking health care. *Gastroenterology* 1982;82:529-534
- Drossman DA, Corazziari E, Talley NJ, Thompson WG, Whitehead WE. Rome II: The Functional Gastrointestinal Disorders. McLean, VA: Degnon Associates; 2000
- Preston DM, Lennard-Jones JE. Severe chronic constipation of young women: 'idiopathic slow transit constipation'. *Gut* 1986;27:41-48
- Bassotti G, de Roberto G, Sediari L, Morelli A. Toward a definition of colonic inertia. *World J Gastroenterol* 2004; 10(17):2465-2467
- Johanson J. Constipation. Washington, DC: US Department of Health and Human Services, Public Health Service, National Institutes of Health, NIDDK; 1994
- Sonnenberg A, Koch TR. Physician visits in the United States for constipation. *Dig Dis Sci* 1989;34:606-611
- Surrenti E, Rath DM, Pemberton JH, Camilleri M. Audit of constipation in a tertiary referral gastroenterology practice. *Am J Gastroenterol* 1995;90:1471-1475
- Probert CS, Emmett PM, Cripps HA, Heaton KW. Evidence for the ambiguity of the term constipation: the role of irritable bowel syndrome. *Gut* 1994;35(10):1455-1458
- Preston DM, Lennard-Jones JE. Pelvic motility and response to intraluminal bisacodyl in slow-transit constipation. *Dig Dis Sci* 1985;30:289-294
- Bassotti G, Chiarioni G, Imbimbo B, et al. Impaired colonic motor response to cholinergic stimulation in patients with severe chronic idiopathic (slow transit type) constipation. *Dig Dis Sci* 1993;38(6):1040-1045
- He C-L, Burgart L, Wang L, et al. Decreased interstitial cells of Cajal volume in patients with slow-transit constipation. *Gastroenterology* 2000;118:14-21
- Bassotti G, Stanghellini V, Chiarioni G, et al. Upper gastrointestinal motor activity in patients with slow-transit constipation. Further evidence for an enteric neuropathy. *Dig Dis Sci* 1996;41(10):1999-2005
- Camilleri M, Fealey RD. Idiopathic autonomic denervation in 8 patients presenting with functional gastrointestinal disease. A causal association? *Dig Dis Sci* 1990;35:609-616
- Kerrigan DD, Lucas MG, Sun WM, Donnelly TC, Read NW. Idiopathic constipation associated with impaired urethrovesical and sacral reflex function. *Br J Surg* 1989;76: 748-751
- Watier A, Devroede G, Duranceau A, et al. Constipation with colonic inertia. A manifestation of systemic disease? *Dig Dis Sci* 1983;28:1025-1033
- Knowles CH, Scott SM, Lunniss PJ. Slow transit constipation: a disorder of pelvic autonomic nerves? *Dig Dis Sci* 2001;46:389-401
- Roe AM, Bartolo DC, Mortensen NJ. Slow transit constipation. Comparison between patients with or without previous hysterectomy. *Dig Dis Sci* 1988;33:1159-1163
- Scott SM, Knowles CH, Newell M, Garvie N, Williams NS, Lunniss PJ. Scintigraphic assessment of colonic transit in women with slow-transit constipation arising de novo and following pelvic surgery or childbirth. *Br J Surg* 2001;88:405-411
- Giaroni C, De Ponti F, Cosentino M, Lecchini S, Frigo G. Plasticity in the enteric nervous system. *Gastroenterology* 1999;117:1438-1458
- Lyford GL, He C-L, Soffer E, et al. Pan-colonic decrease in interstitial cells of Cajal in patients with slow transit constipation. *Gut* 2002;51:496-501
- Wong SW, Lubowski D. Slow transit constipation: evaluation and treatment. *ANZ J Surg* 2007;77:320-328
- Hulzinga JD, Thuneberg L, Kluppel M, Malysz J, Mikkelsen B, Bernstein A. W/Kit gene required for interstitial cells of Cajal and intestinal pacemaker activity. *Nature* 1995;373: 347-349
- Ward SM, Burns AJ, Torihashi S, Sanders KM. Mutation of the proto-oncogene c-kit blocks development of interstitial cells and electrical rhythmicity in murine intestine. *J Physiol* 1994;480:91-97
- Shafik A, Shafik AA, El-Sibai O, Ahmed I. Colonic pacing. A therapeutic option for the treatment of constipation due to total colonic inertia. *Arch Surg* 2003;138:1007-1011
- Bassotti G, Gaburri M, Imbimbo BP, et al. Colonic mass movements in idiopathic chronic constipation. *Gut* 1988; 29(9):1173-1179
- Bassotti G, Chistolini F, Nzepe FS, Morelli A. Colonic propulsive impairment in intractable slow transit constipation. *Arch Surg* 2003;138:1302-1304
- Altomare DF, Portincasa P, Rinaldi M, et al. Slow-transit constipation. Solitary symptom of a systemic gastrointestinal disease. *Dis Colon Rectum* 1999;42:231-240
- El-Salhy M. Chronic idiopathic slow transit constipation: pathophysiology and management. *Colorectal Dis* 2003;5: 288-296
- Zhao R, Baig MK, Wexner SD, et al. Enterochromaffin and serotonin cells are abnormal for patients with colonic inertia. *Dis Colon Rectum* 2000;43:858-863
- Zhao RH, Baig MK, Thaler KJ, et al. Reduced expression of serotonin receptor(s) in the left colon of patients with colonic inertia. *Dis Colon Rectum* 2003;46:81-86
- Agachan F, Chen T, Pfeifer J, Reissman P, Wexner SD. A constipation scoring system to simplify evaluation and management of constipated patients. *Dis Colon Rectum* 1996;39:681-685
- Spiller R. Serotonergic modulating drugs for functional gastrointestinal diseases. *Br J Clin Pharmacol* 2002;54:11-20
- Emmanuel AV, Roy AJ, Nicholls TJ, Kamm MA. Prucalopride, a systemic enterokinetic, for the treatment of constipation. *Aliment Pharmacol Ther* 2002;16:1347-1356
- Koustsomanis D, Lennard-Jones JE, Kamm MA. Prospective study of biofeedback treatment for patients with slow and normal transit constipation. *Eur J Gastroenterol Hepatol* 1994;6:131-137
- Battaglia E, Serra AM, Buonafede G, et al. Long-term study on the effects of visual biofeedback and muscle training as a

- therapeutic modality in pelvic floor dyssynergia and slow-transit constipation. *Dis Colon Rectum* 2004;47:90–95
37. Emmanuel AV, Kamm MA. Response to a behavioural treatment, biofeedback, in constipated patients is associated with improved gut transit and autonomic innervation. *Gut* 2001;49:214–219
38. Wang J, Luo M-H, Qi Q-H, Dong Z-L. Prospective study of biofeedback retraining in patients with chronic idiopathic functional constipation. *World J Gastroenterol* 2003;9:2109–2113
39. Chiotakakou-Faliakou E, Kamm MA, Roy AJ, Storrie JB, Turner IC. Biofeedback provides long-term benefit for patients with intractable, slow and normal transit constipation. *Gut* 1998;42:517–521
40. Brown SR, Donati D, Seow-Choen F, Ho Y-H. Biofeedback avoids surgery in patients with slow-transit constipation: report of four cases. *Dis Colon Rectum* 2001;44:737–740
41. Brazzelli M, Murray A, Fraser C. Efficacy and safety of sacral nerve stimulation for urinary urge incontinence: a systematic review. *J Urol* 2006;175:835–841
42. Kenefick NJ, Christiansen J. A review of sacral stimulation for the treatment of faecal incontinence. *Colorectal Dis* 2004;6:75–80
43. Kenefick NJ, Nicholls RJ, Cohen RG, Kamm MA. Permanent sacral nerve stimulation for treatment of idiopathic constipation. *Br J Surg* 2002;89:882–888
44. Kenefick NJ, Vaizey CJ, Cohen RG, Nicholls RJ, Kamm MA. Double-blind placebo-controlled crossover study of sacral nerve stimulation for idiopathic constipation. *Br J Surg* 2002;89:1570–1571
45. Malouf AJ, Wiesel PH, Nicholls T, Nicholls RJ, Kamm MA. Short-term effects of sacral nerve stimulation for idiopathic slow transit constipation. *World J Surg* 2002;26:166–170
46. Dinning PG, Fuentealba SE, Kennedy ML, Lubowski DZ, Cook IJ. Sacral nerve stimulation induces pan-colonic propagating pressure waves and increases defecation frequency in patients with slow-transit constipation. *Colorectal Dis* 2007;9:123–132
47. Malone PS, Ransley PG, Kiely EM. Preliminary report: the antegrade continence enema. *Lancet* 1990;336:1217–1218
48. Rongen MJGM, van der Hoop AG, Baeten CGMI. Cecal access for antegrade colon enemas in medically refractory slow-transit constipation: a prospective study. *Dis Colon Rectum* 2001;44:1644–1649
49. Lees NP, Hodson P, Hill J, Pearson RC, MacLennan I. Long-term results of the antegrade continent enema procedure for constipation in adults. *Colorectal Dis* 2004;6:362–368
50. Knowles CH, Scott M, Lunniss PJ. Outcome of colectomy for slow transit constipation. *Ann Surg* 1999;230:627–638
51. Pikarsky AJ, Singh JJ, Weiss EG, Noguera JJ, Wexner SD. Long-term follow-up of patients undergoing colectomy for colonic inertia. *Dis Colon Rectum* 2001;44:179–183
52. Glia A, Akerlund JE, Lindberg G. Outcome of colectomy for slow-transit constipation in relation to presence of small-bowel dysmotility. *Dis Colon Rectum* 2004;47:96–102
53. Verne GN, Hocking MP, Davis RH, et al. Long-term response to subtotal colectomy in colonic inertia. *J Gastrointest Surg* 2002;6:738–744
54. FitzHarris GP, Garcia-Aguilar J, Parker SC, et al. Quality of life after subtotal colectomy for slow-transit constipation: both quality and quantity count. *Dis Colon Rectum* 2003;46:433–440
55. Athanasakis H, Tsiaoussis J, Vassilakis JS, Xynos E. Laparoscopically assisted subtotal colectomy for slow-transit constipation. *Surg Endosc* 2001;15:1090–1092
56. Ho YH, Tan M, Eu KW, Leong A, Seow-Choen F. Laparoscopic-assisted compared with open total colectomy in treating slow transit constipation. *Aust N Z J Surg* 1997;67:562–565
57. Sarli L, Costi R, Sarli D, Roncoroni L. Pilot study of subtotal colectomy with antiperistaltic cecoproctostomy for the treatment of chronic slow-transit constipation. *Dis Colon Rectum* 2001;44:1514–1520
58. Sarli L, Iusco D, Donadei E, et al. The rationale for cecorectal anastomosis for slow transit constipation. *Acta Biol Med (Gdansk)* 2003;74(suppl 2):74–79
59. Marchesi F, Sarli L, Percalli L, et al. Subtotal colectomy with antiperistaltic cecorectal anastomosis in the treatment of slow-transit constipation: Long-term impact on quality of life. *World J Surg* 2007;31:1658–1664
60. Iannelli A, Fabiani P, Mouiel J, Gugenheim J. Laparoscopic subtotal colectomy with cecorectal anastomosis for slow-transit constipation. *Surg Endosc* 2006;20:171–173
61. Lundin E, Karlsson U, Pahlman L, Graf W. Outcome of segmental colonic resection for slow-transit constipation. *Br J Surg* 2002;89:1270–1274
62. You YT, Wang JY, Changchien CR, et al. Segmental colectomy in the management of colonic inertia. *Am Surg* 1998;64:775–777
63. Akervall S, Fasth S, Nordgren S, Oresland T, Hulten L. The functional results after colectomy and ileorectal anastomosis for severe constipation (Arbuthnot Lane's disease) as related to rectal sensory function. *Int J Colorectal Dis* 1988;3:96–101
64. Pluta H, Bowes KL, Jewell LD. Long-term results of total abdominal colectomy for chronic idiopathic constipation. Value of preoperative assessment. *Dis Colon Rectum* 1996;39:160–166
65. Mollen RM, Kuijpers HC, Claassen AT. Colectomy for slow-transit constipation: preoperative functional evaluation is important but not a guarantee for a successful outcome. *Dis Colon Rectum* 2001;44:577–580
66. Ripetti V, Caputo D, Greco S, Alloni R, Coppola R. Is total colectomy the right choice in intractable slow-transit constipation? *Surgery* 2006;140:435–440
67. Kalbassi MR, Winter DC, Deasy JM. Quality-of-life assessment of patients after ileal pouch-anal anastomosis for slow-transit constipation with rectal inertia. *Dis Colon Rectum* 2003;46:1508–1512
68. Keighley MRB, Grobler S, Bain I. An audit of restorative proctocolectomy. *Gut* 1993;34:680–684