Pediatr Surg Int
DOI 10.1007/s00383-011-2872-x

ORIGINAL ARTICLE

Slow-transit constipation with concurrent upper gastrointestinal dysmotility and its response to transcutaneous electrical stimulation

Yee Ian Yik · Melanie C. C. Clarke · Anthony G. Catto-Smith · Val J. Robertson · Jonathan R. Sutcliffe · Janet W. Chase · Susan Gibb · Timothy M. Cain · David J. Cook · Coral F. Tudball · John M. Hutson · Bridget R. Southwell

Accepted: 15 December 2010

Abstract
Purpose Transcutaneous electrical stimulation (TES) speeds up colonic transit in children with slow-transit constipation (STC). This study examined if concurrent upper gastrointestinal dysmotility (UGD) affected response to TES.
Methods Radio-nuclear transit studies (NTS) were performed before and after TES treatment of STC as part of a larger randomised controlled trial. UGD was defined as delayed gastric emptying and/or slow small bowel transit. Improvement was defined as increase of $\geq$1 Geometric Centre (median radiotracer position at each time [small bowel = 1, toilet = 6]).
Results Forty-six subjects completed the trial, 34 had NTS after stimulation (21 M, 8–17 years, mean 11.3 years; symptoms >9 years). Active stimulation increased transit in >50% versus only 25% with sham ($p = 0.04$). Seventeen children also had UGD. In children with STC and either normal upper GI motility (NUGM) and UGD, NTS improved slightly after 1 month (57 vs. 60%; $p = 0.9$) and more after 2 months (88 vs. 40%; $p = 0.07$). However, mean transit rate significantly increased with NUGM, but not UGD (5.0 ± 0.2: 3.6 ± 0.6, $p < 0.01$).

Y. I. Yik · M. C. C. Clarke · J. R. Sutcliffe · J. W. Chase · J. M. Hutson · B. R. Southwell (✉)
F Douglas Stephens Surgical Research and Gut Motility Laboratories, Murdoch Children's Research Institute, Royal Children's Hospital, Parkville, Melbourne, VIC 3052, Australia
e-mail: bridget.southwell@mcri.edu.au

M. C. C. Clarke
e-mail: mccclarke@gmail.com

J. W. Chase
e-mail: janchase@vicnet.net.au

Y. I. Yik · J. M. Hutson
Department of Paediatrics, University of Melbourne, Melbourne, Australia

Y. I. Yik
Division of Pediatric Surgery, Department of General Surgery, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia
e-mail: yiyik@um.edu.my

A. G. Catto-Smith
Department of Gastroenterology and Nutrition, Royal Children's Hospital, Melbourne, Australia
e-mail: tony.cattosmith@rch.org.au

J. M. Hutson
Royal Children's Hospital, Melbourne, Australia
e-mail: john.hutson@rch.org.au

V. J. Robertson
University of Newcastle, Newcastle, NSW, Australia
e-mail: val.robertson@newcastle.edu.au

S. Gibb
Department of Paediatrics, Royal Children's Hospital, Melbourne, Australia
e-mail: susie.gibb@rch.org.au

T. M. Cain · D. J. Cook · C. F. Tudball
Department of Medical Imaging, Royal Children's Hospital, Melbourne, Australia
e-mail: tim.cain@rch.org.au

D. J. Cook
e-mail: david.cook@rch.org.au

C. F. Tudball
e-mail: coral.tudball@rch.org.au

Published online: 04 March 2011

Conclusion Transcutaneous electrical stimulation was beneficial for STC, with response weakly associated with UGD. As measured by NTS, STC children with NUGM responded slightly more, but with significantly greater increased transit compared to those with UGD. Higher numbers are needed to determine if the difference is important.

Keywords Upper gastrointestinal dysmotility (UGD) · Delayed gastric emptying (DGE) · Slow small bowel transit (SSBT) · Transcutaneous electrical stimulation (TES) · Nuclear transit study (NTS)

Introduction

Slow-transit constipation (STC) describes a clinical syndrome characterised by intractable constipation that is not readily responsive to laxatives, diet or a change in lifestyle [1]. It is characterised by delayed colonic transit without an underlying systemic disorder or pelvic floor dysfunction. Although it was initially described in young women of reproductive age [2, 3], it has been recognised recently as a condition affecting children of all ages [4].

Slow colonic transit is readily demonstrated by transit studies using either radio-opaque pellets or nuclear scintigraphy [5]. The association of concurrent upper gastrointestinal dysmotility (UGD) and its significance has been increasingly studied and reported [6–9]. Various tests have been described for the assessment and measurement of upper gastrointestinal motility and no single test is specific [10–12], however, scintigraphy is also useful to study upper gastrointestinal motility, as well as colonic transit [6–9]. Delayed gastric emptying (DGE) and slow small bowel transit (SSBT) in adults with STC are well reported [9, 13], but not in STC children. Nuclear transit study (NTS) is only available in a few centres worldwide; however, its use has characterised and categorised children with chronic constipation according to colonic transit [5, 14, 15].

Recently, it has been suggested that STC may be part of a pan-enteric disorder as alterations in oesophageal motility [16], gastric emptying [7, 16–18] and small bowel motility [7, 17, 19, 20] have been observed in some patients with STC. In adults, different treatment strategies have been employed to treat this subgroup of patients as they respond poorly to standard medical and surgical therapies. Hence, we are unsure whether the same rules apply in treating STC children with UGD.

Recent trials of transcutaneous electrical stimulation (TES) conducted in our institute, have encouraging results in treating children with STC [21–24]. The overall results have already been reported, but the response of the subgroup of these STC children with associated UGD has not been reported and is unknown. Hence, this retrospective study aimed to reanalyse the results in our recent randomised, controlled trial to see if there is a difference in response to TES if STC children with colonic dysmotility also have UGD.

With the analysis of the nuclear transit studies (NTS) already performed during the trial, we were able to sort children with STC into subgroups with or without associated UGD [14, 15], classified as delayed gastric emptying and/or slow small bowel transit.

Methods

Patients were recruited into a randomised, controlled trial (RCT) using TES to treat children with STC at The Royal Children's Hospital (Ethics #23040C). All children were diagnosed with STC using NTS (as described previously [5, 14, 15]) prior to trial entry. Those who had chronic constipation, but not STC were excluded:

Inclusion criteria

- Children (aged 8–18 years) ≥2-year history of chronic constipation (consistent with Rome II criteria) ± soiling ± appendix stoma utilised for antegrade continence enemas.
- Blood tests to exclude hormonal, allergic and metabolic causes for their constipation [thyroid function tests (TFT), flood blood count (FBC) and coeliac screen].
- Proven slow-transit constipation on a recent (within the last 2 years) NTS or have had abnormal colonic motility demonstrated by colonic manometry.

Exclusion criteria

- Children with a normal colonic transit time or functional faecal retention (FFR) demonstrated on NTS.
- Children with any metabolic or hormonal cause underlying their constipation.
- Children with Hirschsprung's disease or previous anorectal malformation.
- Children who have undergone any surgical procedure (other than the formation of an appendix stoma) that has resulted in discontinuity of their gastrointestinal tract.
- Children who have any contraindication to receiving transcutaneous electrical therapy (e.g. skin sensitivity, pacemaker in-situ).
- Children who are unable to respond to the questionnaires due to intellectual disability or short attention spans.
- Previous transcutaneous electrical therapy for treatment of constipation.

Clinical assessment

Bowel function (history, medical management, neonatal issues, existing bowel symptoms), weight, height, blood pressure, pulse rate and assessment of faecal loading by abdominal palpation were performed followed by 4 weeks recording of daily diary. The diary recorded defecation frequency, response to urge, consistency (Bristol Stool Scale), soiling, medications usage (laxatives, stool softeners) and abdominal pain.

Sample size

During the pilot study [21], 5/8 (63%) children increased defecation frequency from <3 into the normal range. An initial power analysis concluded that with a sample size of 60, the RCT would show a statistically significant change if the number increasing to >3 defecations per week with treatment was 25% greater than the number increasing with sham. Sixty-two children were enrolled. Interim analysis of transit, soiling and quality of life was done on 35 randomised children (18 sham, 17 active) with active treatment producing a statistically significant benefit in transit time, soiling and abdominal pain as compared to sham. From this time, a further 11 children (who had already commenced) completed the protocol, but no further subjects were recruited as trial staff felt it was unethical to continue giving sham stimulation. This gave a total of 46 randomised subjects completing the protocol. Eight children with existing appendicostomies had active stimulation and 24-h colonic manometry.

Randomisation

Instructions for active or sham stimulation were randomised (in blocks of 6) and sealed into numbered envelopes before recruitment. For each child recruited, the next envelope was mailed to the treating physiotherapist.

In the RCT, 46 children were randomly assigned to active ($n = 23$) or sham ($n = 23$) stimulation in the first treatment session, 12 treatments over 1 month. Subsequently, all children were rested for a period of 8 weeks without any stimulation. During a second month of treatment, all children were given active stimulation (12 treatments over 1 month). At each treatment session, TES was applied by a physiotherapist for 20 min, 3×/week for 4 weeks using 4 electrodes (quadripolar stimulation), 2 on the anterior abdominal wall and 2 at the back paraspinally (T9–L2). The electrical settings were 4 kHz carrier frequency with a beat frequency of 80–150 Hz. Forty-two children completed the trial, 34 had NTS before TES, 20 had NTS after the first treatment session (12 active stimulation = A1, 8 sham = B1) and 19 had NTS after the second treatment session (13 active/active = A2, 6 sham/active = B2).

NTS were performed using liquid meals with radiotracers premixed (99 m-Technetium colloid or 67-gallium citrate, calculated doses at 160 and 12 MBq, respectively, equivalent to two standard abdominal radiographs) [14]. Gamma camera images were taken between 0 and 2 h (at 0, 0.5, 1 and 2 h) for gastric emptying study and further images were taken at 6, 24, 30 and 48 h for assessment of small bowel and colonic transit. Six regions of interest were identified: 1 small bowel; 2 ascending colon; 3 transverse colon; 4 descending colon; 5 rectosigmoid colon and 6 evacuation into toilet. For each patient, the geometric centre (GC = the median point of the radioactivity at each time-point) was calculated at 6, 24, 30 and 48 h after ingestion, using the formula described by Notghi et al. [Geometric centre = sum of (fraction of activity × region number)] [25].

Upper gastrointestinal dysmotility is classified into subgroups based on the gastric emptying rate and small bowel transit. Delayed gastric emptying was defined arbitrarily as $t_{1/2} > 50$ min and >15% retained tracer at 2 h (Fig. 1b), while slow small bowel transit was defined as >25% retained tracer in the small intestines at 6 h (Fig. 1d). Two groups were identified– STC with or without UGD. Their responses to electrical stimulation were examined to determine whether the presence of UGD would alter the outcome of treatment.

The GC calculated at 6, 24, 30 and 48 h [14, 15] was tabulated using Microsoft Excel and data analysis was performed using GraphPad Prism 3. Statistical analysis was performed using analysis of variance (ANOVA) to compare the GC of multiple treatments at each time-point and χ^2 test to compare the percentage of responders. A $p < 0.05$ was considered significant.

In addition, changes in transit were assessed visually to determine the number of children improving (responder). Improvement was defined as an increase in GC of ≥1 region at 48 h.

Results

Clinical outcome

At the start of the trial, most children took laxatives and had frequent small stools with ≥3 defecations per week. There was no change in the mean number of defecations per week. There was a decrease in soiling and in abdominal pain and laxatives use in the active, but not sham group. The detailed results have been submitted in a separate manuscript (Chase et al. submitted).

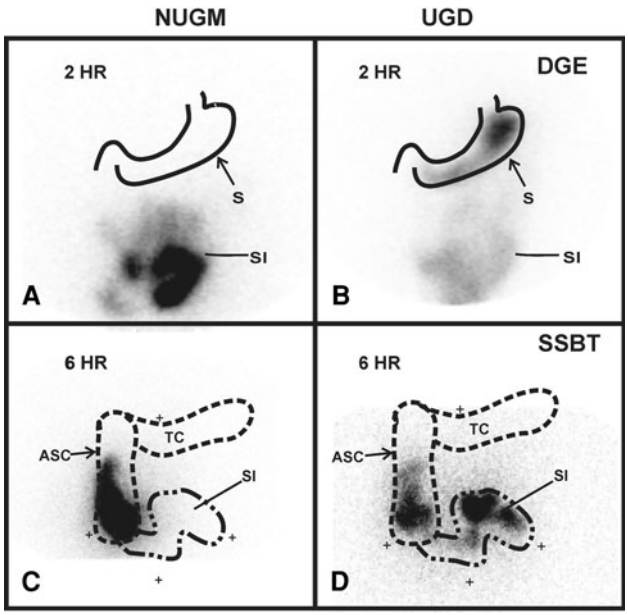

Fig. 1 Gastric emptying with retained tracer at 2 h. **a** Normal gastric emptying, <15% of tracer in stomach, **b** delayed gastric emptying, >15% of tracer in stomach. Gastrointestinal transit at 6 h. **c** Normal small bowel transit, **d** slow small bowel transit with >25% of tracer retained in small intestine (*UGD* upper gastrointestinal dysmotility, *NUGM* normal upper gastrointestinal motility, *DGE* delayed gastric emptying, *SSBT* slow small bowel transit, *S* stomach, *ASC* ascending colon, *TC* transverse colon, *SI* small intestine)

Transit studies

Active stimulation resulted in faster transit at 24 and 48 h after ingestion of radiolabel. There was no difference in colonic transit time in the sham group before and after stimulation [23]. The second period of stimulation and the presence of concurrent upper GI motility (i.e. gastric emptying rate and/or small intestinal motility) were not analysed in the previous report.

While 42/46 children completed the RCT, only 34 children (22M, 12F; age 8–17 years, mean 11.1 years; symptom duration mean 9.4 years) had NTS both before and after treatment (20 patients after first treatment and 19 patients after second treatment, Table 1). Only five patients had NTS after both the first and second stimulation periods, as it was difficult for families to spend 3 days at the hospital while the follow-up NTS was performed. After 1 month of treatment, active stimulation increased the transit speed in 58% (7/12) of children (Table 2), while only 25% (2/8) responded in the sham treatment group ($p = 0.04$). In the second month of stimulation, both groups received active treatment. The number of responders in the group given sham treatment in the first month increased from 25 to 83% (5/6) following active treatment ($p = 0.03$).

Looking at the individual patient's NTS data, 11/20 (at 1st stimulation) and 9/19 (at 2nd stimulation) STC children had concurrent UGD (Table 2). Six individual patient's data are illustrated (Fig. 2) to demonstrate their response after each treatment session. Patients with NUGM (Patients 1 and 2) showed a greater increase in colonic transit rate than those with UGD who responded to treatment (Patients 5 and 6). Patients 3 and 4 had STC with UGD and did not respond to TES.

For those with NUGM, after 1 month of active stimulation, 4/7 (57%) showed improvement and this increased to 7/8 (88%) after 2 months of active stimulation. In those with UGD 3/5 (60%) improved after 1 month, but only 2/5 (40%) after 2 months active stimulation. After 2 months of active stimulation, more patients with NUGM had

Table 1 Number of patients enrolled and with transit studies performed after each treatment period

	Number of patients (*n*)			
	1st period of stimulation		2nd period of stimulation	
	Active (A1)	Sham (B1)	Active (A2)	Active (B2)
Total enrolled	23	23		
Total completed	21	21	19	15
Transit study	12	8	13	6

Table 2 Number of patients with normal upper GI motility (NUGM) and upper GI dysmotility (UGD) showing improved transit after each treatment period

Motility as defined by NTS	Number of patients improved/total number (%)			
	1st month of stimulation		2nd month of stimulation	
	Active (A1) (%)	Sham (B1) (%)	Active (A2) (%)	Active (B2) (%)
NUGM	4/7 (57)	0/2 (0)	7/8 (88)	2/2 (100)
UGD (DGE, SSBT)	3/5 (60)	2/6 (33)	2/5 (40)	3/4 (75)
Total (*n*)	7/12 (58)	2/8 (25)	9/13 (69)	5/6 (83)

UGD upper gastrointestinal dysmotility, *NUGM* normal upper gastrointestinal motility, *DGE* delayed gastric emptying, *SSBT* slow small bowel transit

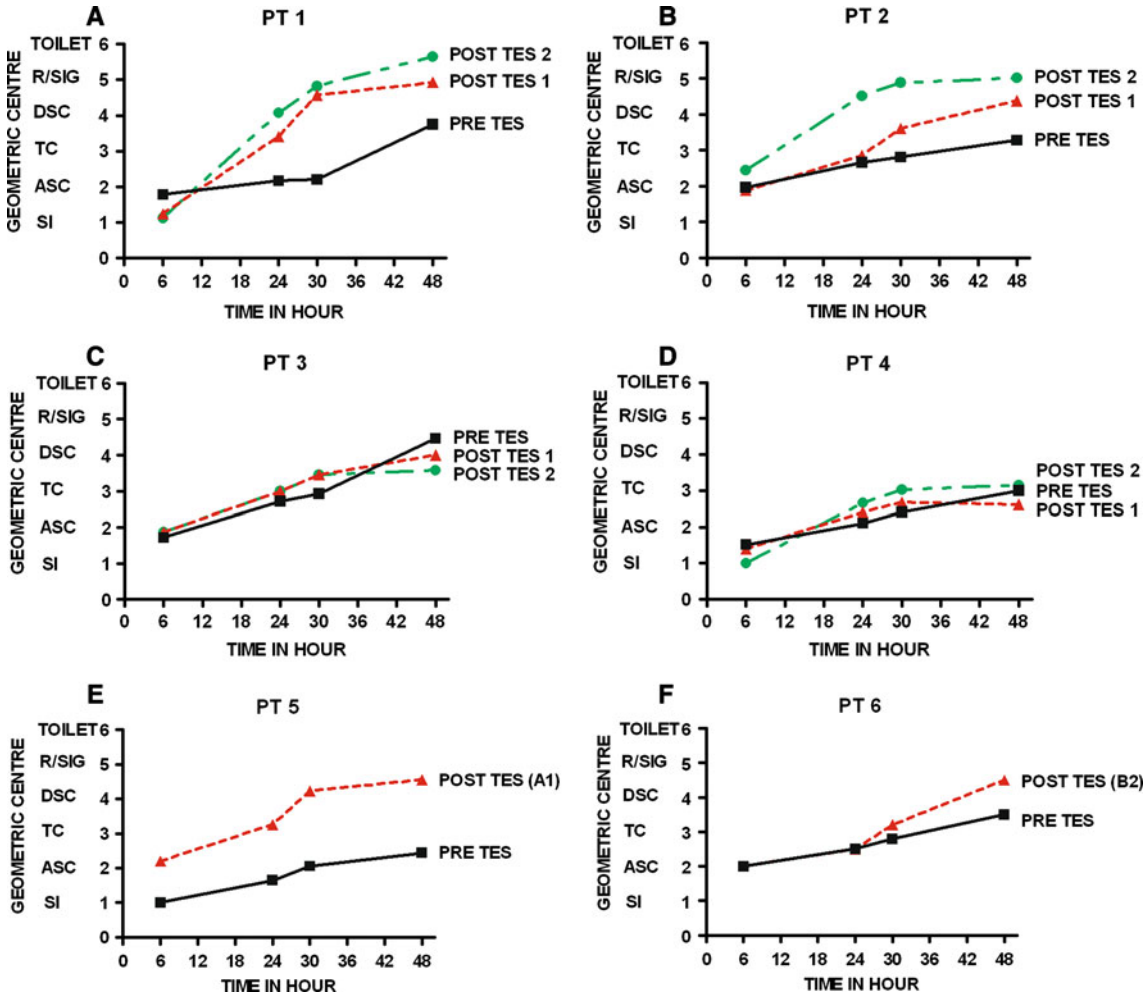

Fig. 2 Individual patient responses to TES. **a**, **b** NUGM. Colonic transit at baseline (pre) and after first month and second month of treatment. **a** Both months active treatment, **b** first month sham and second month active stimulation. **c**–**f** UGD. **c** Both months active, **d** first month sham and second month active stimulation. **e** After 1 month active. **f** First month sham and second month active (transit study post second month only) [responders = patients 1, 2, 5 and 6; non-responders = patients 3 and 4] (*UGD* upper gastrointestinal dysmotility, *NUGM* normal upper gastrointestinal motility, *TES* transcutaneous electrical stimulation, *SI* small intestines, *ASC* ascending colon, *TC* transverse colon, *DSC* descending colon, *R/SIG* recto-sigmoid colon)

responded than those with UGD (88 vs. 40%, $p = 0.07$). Sham stimulation did not change transit.

The patient group that had NUGM and active stimulation through 2 months of stimulation (A2) showed a significant increase in the mean speed of colonic transit at 48 h ($p < 0.01$), while there was no significant change in this patient group after 1 month of active stimulation (A1) (Fig. 3). For the patient group with both colonic and UGD, there was no change in mean colonic transit rate.

Discussion

Overall, our results showed significant improvement of colonic transit after active stimulation as compared to sham stimulation, and a slightly higher response rate in the group with NUGM after active stimulation for 2 months as compared to the group with UGD. Our results suggest that (1) STC children with NUGM respond only slightly more readily to TES therapy, and (2) 2 months of active stimulation were more effective than 1 month of active stimulation for those with NUGM, but not those with UGD. For children with UGD, more treatment may be required to achieve similar improvement of colonic transit, perhaps with longer duration of TES therapy. Alternatively, this may be a subgroup of STC that does not respond to TES.

Since its discovery in the early 1950s by Dr Nemec from Vienna, TES has mainly been used in treating pain and musculoskeletal conditions [26–29], wound healing [30] and urinary incontinence [31, 32]. Recently, we have found

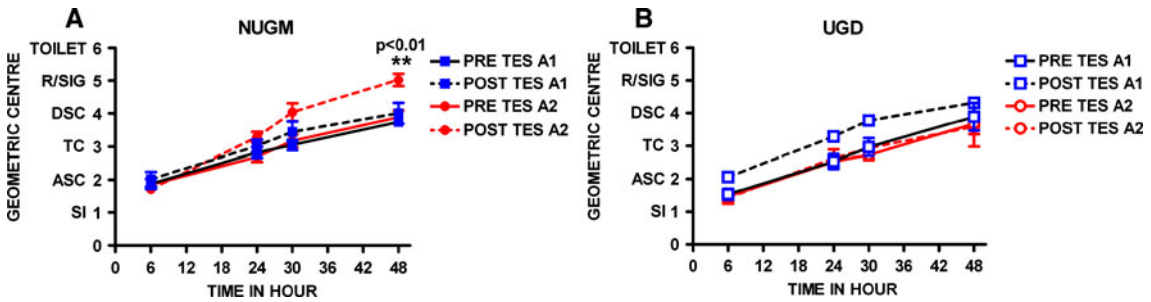

Fig. 3 Mean colonic transit in STC groups with **a** NUGM or **b** UGD. Colonic transit at baseline (pre) and after 1 month active (A1) and 2 months active (A2) stimulation (*TES* transcutaneous electrical stimulation, *UGD* upper gastrointestinal dysmotility, *NUGM* normal upper gastrointestinal motility, *SI* small intestine, *ASC* ascending colon, *TC* transverse colon, *DSC* descending colon, *R/SIG* recto-sigmoid colon) $^{**}p < 0.01$; mean (SEM)

treatment using TES showed promising results in treating children with chronic treatment-resistant constipation, i.e. slow-transit constipation (STC) [21–24]. However, the clinical response of these children based on the extent of gastrointestinal dysmotility has not been reported.

Using NTS, we were able to show that some children with chronic treatment-resistant constipation have slow colonic transit [5, 14, 15]. In addition, gastric emptying rate and small bowel transit were useful information gathered in the same transit study. At our institute, these children were enrolled for TES as part of an RCT. The clinical response to treatment has been published [22, 23], with improved quality of life and faster colonic transit after TES. This study examined if concurrent UGD had an effect on the speed of colonic transit as measured by NTS after TES. The response rate was slightly better in STC children with NUGM; however, the magnitude of individual responses was much greater in children with NUGM. The treatment window may need to be longer for STC children with associated UGD, or it may be that this is a subgroup that does not respond to TES at all. Future studies on more patients may be required to address this issue.

Conclusion

Transcutaneous electrical stimulation was a beneficial treatment for paediatric STC, but the amplitude of response was associated with the extent of concurrent UGD. Children with associated UGD may need more aggressive therapy and TES may be required for longer than is required by STC children without UGD.

References

1. El-Salhy M (2003) Chronic idiopathic slow transit constipation: pathophysiology and management. Colorectal Dis 5:288–296
2. Watier A, Devroede G, Duranceau A et al (1983) Constipation with colonic inertia. A manifestation of systemic disease? Dig Dis Sci 28:1025–1033
3. Preston DM, Lennard-Jones JE (1986) Severe chronic constipation of young women: 'idiopathic slow transit constipation'. Gut 27:41–48
4. Benninga MA, Buller HA, Tytgat GN, Akkermans LM, Bossuyt PM, Taminiau JA (1996) Colonic transit time in constipated children: does pediatric slow-transit constipation exist? J Pediatr Gastroenterol Nutr 23:241–251
5. Southwell BR, Clarke MC, Sutcliffe J, Hutson JM (2009) Colonic transit studies: normal values for adults and children with comparison of radiological and scintigraphic methods. Pediatr Surg Int 25:559–572
6. Stivland T, Camilleri M, Vassallo M et al (1991) Scintigraphic measurement of regional gut transit in idiopathic constipation. Gastroenterology 101:107–115
7. van der Sijp JR, Kamm MA, Nightingale JM et al (1993) Disturbed gastric and small bowel transit in severe idiopathic constipation. Dig Dis Sci 38:837–844
8. Charles F, Camilleri M, Phillips SF, Thomforde GM, Forstrom LA (1995) Scintigraphy of the whole gut: clinical evaluation of transit disorders. Mayo Clin Proc 70:113–118
9. MacDonald A, Baxter JN, Bessent RG, Gray HW, Finlay IG (1997) Gastric emptying in patients with constipation following childbirth and due to idiopathic slow transit. Br J Surg 84:1141–1143
10. Camilleri M, Hasler WL, Parkman HP, Quigley EM, Soffer E (1998) Measurement of gastrointestinal motility in the GI laboratory. Gastroenterology 115:747–762
11. Bannister JJ, Timms JM, Barfield LJ, Donnelly TC, Read NW (1986) Physiological studies in young women with chronic constipation. Int J Colorectal Dis 1:175–182
12. Abid S, Lindberg G (2007) Electrogastrography: poor correlation with antro-duodenal manometry and doubtful clinical usefulness in adults. World J Gastroenterol 13:5101–5107
13. Mollen RM, Hopman WP, Kuijpers HH, Jansen JB (1999) Abnormalities of upper gut motility in patients with slow-transit constipation. Eur J Gastroenterol Hepatol 11:701–708
14. Cook BJ, Lim E, Cook D et al (2005) Radionuclear transit to assess sites of delay in large bowel transit in children with chronic idiopathic constipation. J Pediatr Surg 40:478–483
15. Sutcliffe JR, King SK, Hutson JM, Cook DJ, Southwell BR (2009) Gastrointestinal transit in children with chronic idiopathic constipation. Pediatr Surg Int 25:465–472
16. Reynolds JC, Ouyang A, Lee CA, Baker L, Sunshine AG, Cohen S (1987) Chronic severe constipation. Prospective motility studies in 25 consecutive patients. Gastroenterology 92:414–420

17. Bassotti G, Stanghellini V, Chiarioni G et al (1996) Upper gastrointestinal motor activity in patients with slow-transit constipation. Further evidence for an enteric neuropathy. Dig Dis Sci 41:1999–2005
18. Penning C, Vu MK, Delemarre JB, Masclee AA (2001) Proximal gastric motor and sensory function in slow transit constipation. Scand J Gastroenterol 36:1267–1273
19. Panagamuwa B, Kumar D, Ortiz J, Keighley MR (1994) Motor abnormalities in the terminal ileum of patients with chronic idiopathic constipation. Br J Surg 81:1685–1688
20. Penning C, Gielkens HA, Hemelaar M et al (2000) Prolonged ambulatory recording of antroduodenal motility in slow-transit constipation. Br J Surg 87:211–217
21. Chase J, Robertson VJ, Southwell B, Hutson J, Gibb S (2005) Pilot study using transcutaneous electrical stimulation (interferential current) to treat chronic treatment-resistant constipation and soiling in children. J Gastroenterol Hepatol 20:1054–1061
22. Clarke MC, Chase JW, Gibb S, Hutson JM, Southwell BR (2009) Improvement of quality of life in children with slow transit constipation after treatment with transcutaneous electrical stimulation. J Pediatr Surg 44:1268–1272 (discussion 1272)
23. Clarke MC, Chase JW, Gibb S et al (2009) Decreased colonic transit time after transcutaneous interferential electrical stimulation in children with slow transit constipation. J Pediatr Surg 44:408–412
24. Ismail KA, Chase J, Gibb S et al (2009) Daily transabdominal electrical stimulation at home increased defecation in children with slow-transit constipation: a pilot study. J Pediatr Surg 44:2388–2392
25. Notghi A, Hutchinson R, Kumar D, Tulley N, Harding LK (1994) Use of geometric center and parametric images in scintigraphic colonic transit studies. Gastroenterology 107:1270–1277
26. Johnson MI, Tabasam G (2003) An investigation into the analgesic effects of different frequencies of the amplitude-modulated wave of interferential current therapy on cold-induced pain in normal subjects. Arch Phys Med Rehabil 84:1387–1394
27. Tabasam G, Johnson MI (2006) The use of interferential therapy for pain management by physiotherapists…including commentary by Poitras S. Int J Therapy Rehabilitation 13:357–364
28. Ward AR (2009) Electrical stimulation using kilohertz-frequency alternating current. Phys Ther 89:181–190
29. Ward AR, Chuen WL (2009) Lowering of sensory, motor, and pain-tolerance thresholds with burst duration using kilohertz-frequency alternating current electric stimulation: part II. Arch Phys Med Rehabil 90:1619–1627
30. Ganne J-M (1988) Stimulation of bone healing with interferential therapy. Aust J Physiother 34:9–20
31. Dumoulin C, Seaborne DE, Quirion-DeGirardi C, Sullivan SJ (1995) Pelvic-floor rehabilitation, part 2: Pelvic-floor re-education with interferential currents and exercise in the treatment of genuine stress incontinence in postpartum women—a cohort study. Phys Ther 75:1075–1081
32. Kajbafzadeh AM, Sharifi-Rad L, Baradaran N, Nejat F (2009) Effect of pelvic floor interferential electrostimulation on urodynamic parameters and incontinency of children with myelomeningocele and detrusor overactivity. Urology 74:324–329