Current issues in the management of adult faecal incontinence

Faecal incontinence is a common yet under-reported cause of reduced quality of life. Adequate assessment is crucial in establishing the underlying aetiology and appropriate treatment. This article explores such an assessment, its challenges and the management options for faecal incontinence.

Faecal incontinence is defined as 'the involuntary loss of liquid or solid stool that is a social or hygienic problem' (Norton et al, 2005), a definition often expanded to include loss of flatus. It affects 10% of adults at some stage of their lives (Perry et al, 2002), often disastrously with ensuing embarrassment, social isolation, stigmatization and unemployment (Rockwood et al, 2000). The overall prevalence of faecal incontinence in the adult population is estimated at 2–3%, affecting the daily activities of 1–2% of adults at any one time (Perry et al, 2002).

Faecal incontinence has also been associated with depression, reduced activity and perineal skin breakdown, with the latter sometimes resulting in significant sepsis (Bellicini et al, 2008). It is more common with age and is suffered by half of nursing home residents (Bellicini et al, 2008). Indeed, faecal incontinence is the second most common reason for admission to nursing homes; more so even than dementia. As the population continues to age this will present yet greater challenges (Perry et al, 2002). However, faecal incontinence is also prevalent in young adults and is experienced by 4% of women after vaginal delivery (MacArthur et al, 1997), with a further 30% displaying occult sphincter damage (Sultan et al, 1993). It is certain, however, that its true prevalence is underestimated, with patients too embarrassed to seek help and doctors not proactive enough in seeking cases.

Faecal continence

Continence depends on the functioning of the smooth internal sphincter, the striated external sphincter and the pelvic floor musculature. The latter comprises the puborectalis (which is continuous with the external sphincter), pubococcygeus, ileococcygeus and (ischio)coccygeus. These muscular units in turn rely upon local and spinal neural reflexes, and are modified by somatic inputs from the brainstem and frontal lobes. Defecation is a complicated process, instigated when rectal volume approaches 300 ml and triggers pelvic floor pressure receptors. Reflexively, the internal anal sphincter, puborectalis and anorectal angle relax. Unless inhibited by higher control the rectum contracts, the pelvic floor descends and the external anal sphincter relaxes to allow defecation. The latter plays an important role in preventing inadvertent defecation caused by increased intra-abdominal pressure (e.g. caused by coughing) by means of a spinal reflex arc involving the pudendal nerve.

Faecal incontinence and classification

Abnormalities in any component of this system may jeopardize continence, and can be broadly classified as in *Table 1* (Kamm, 1998). Other methods of classification include that of predominant symptom (e.g. urgency or liquid leakage).

In general, abnormalities of the internal sphincter and impacted stool cause passive soiling, whereas abnormalities of the external sphincter and diarrhoea cause urgency. Incontinence of flatus suggests mild incontinence, becoming progressively more severe with loss of liquid and then solid stool (Kamm, 1998). Conditions causing faecal incontinence are myriad including congenital abnormalities, obstetric trauma, structural disease (mass lesions, constipation, prolapse and surgery), aberrant compliance (inflammatory bowel disease and radiation), muscular degeneration and myopathies, peripheral and central neurological injury (pudendal neuropathy, stroke, spinal injury and multiple sclerosis), and abnormalities of stool (diarrhoea and constipation) (Bellicini et al, 2008). This may be compounded by physical and mental impediments preventing timely access to toilet facilities. While the most common causes of faecal incontinence are obstetric, surgical injury and idiopathic degeneration (Kamm, 1998), there is a great deal of crossover with

Table 1. A classification of faecal incontinence

Type	Examples of causes
Intact but weak anal sphincter	Rectal prolapse, inflammatory bowel disease, neuropathy, constipation and diarrhoea
Simple defects of anal sphincter	Obstetric disruptions, postoperative
Complex defects of anal sphincter	Trauma, complex congenital malformations

Adapted from Kamm (1998)

Mr John M Findlay *is Core Surgical Trainee 1, John Radcliffe Hospital, Oxford OX3 9DU and* **Mr Charles Maxwell-Armstrong** *is Consultant Colorectal Surgeon, Queen's Medical Centre, Nottingham*

Correspondence to: Mr JM Findlay

urinary incontinence, with up to 26% experiencing double incontinence (Khullar et al, 1998). In truth, however, the aetiology is often poorly understood.

Diagnosis

History and examination

Faecal incontinence may be caused by a simple and reversible underlying factor, or be complex and multifactorial. Consequently, initial assessment must be comprehensive and structured so as not to miss reversible and sinister causes (*Table 2*); it is important to remember that faecal incontinence is not a diagnosis in itself. Clinicians must be proactive, and tactfully question high-risk patients so as not to miss cases. It is also crucial to consider the capacity and cognitive status of patients, as one may often be dealing with vulnerable adults. Examination should be both general and specific to the abdomen and anorectum, although it is important to remember that assessment of anal tone digitally may not correlate with formal manometry. Other findings on examination may include obvious deformities of the anus.

Investigations

These should be tailored to the individual (*Table 3*). As a baseline, bowel diaries should be undertaken, usually over a 3-month period. These allow the clinician to understand the patient's level of symptoms over time, provide an accurate assessment of the efficacy of any therapeutic procedure instituted, and allow an incontinence score to be determined. A number of such scoring systems have been described, of which the Wexner score (Jorge and Wexner, 1993) is probably the most common and scores a patient's continence on the basis of frequency of incontinence, use of pads and lifestyle alteration.

Colonic pathology should be excluded by rigid or flexible sigmoidoscopy, or colonoscopy; barium enema is a valid alternative. Subsequent investigations should be undertaken by specialist continence centres to guide management. Anal manometry, via pressure-transducing catheters and balloons, can assess the magnitude and duration of resting and squeeze anal pressures, the rectoanal inhibitory reflex (preventing defecation during raised intra-abdominal pressure), rectal compliance and threshold of first rectal sensation (Wald, 1994). The latter is important, with poor rectal sensation often precluding successful use of biofeedback (Wald, 1983).

Endoanal ultrasound is a valuable and well-tolerated advance, used to delineate structural abnormalities within the sphincters and highlight those amenable to surgical correction (Saclarides, 1998). If endoanal ultrasound is unavailable, magnetic resonance imaging is a useful second line. Electromyography is a more invasive test that uses surface or needle electrodes to assess neuromuscular units (Tjandra et al, 1993). Its uses include the assessment of traumatic and neurological damage to the sphincters and their suitability for surgical reconstruction (Sangwan et al, 1996). Another investigation is defaecography of barium paste which can aid the diagnosis of rectocoele, and visualize the sphincters and anorectal angle; it may, of course, be impossible because of the severity of incontinence itself (Barnett et al, 1999). Such specialist investigations are invaluable, and alter the diagnosis and management of up to one fifth of patients (Keating et al, 1997).

Table 2. Important aspects of assessment

Warning signs of lower gastrointestinal malignancy
Treatable causes of diarrhoea (e.g. inflammatory bowel disease, infection)
Faecal loading and overflow incontinence
Cauda equina compression, acute disc prolapse
Rectal prolapse, third degree haemorrhoids
Obstetric history, including traumatic vaginal and forceps deliveries (pudendal neuropathy)
Comorbidities
Neurological or physical impairment, conditions associated with neuropathy
Anti-diarrhoeals and laxatives, drugs predisposing to faecal incontinence (*Table 4*)
Differentiation from pseudoincontinence (loss of non-faecal material, e.g. blood)
Neoplasms, sexually transmitted infections, vaginal discharge

Table 3. Investigations for faecal incontinence

Baseline
Bowel diaries, stool microbiology
Imaging
Proctoscopy and rigid sigmoidoscopy, flexible sigmoidoscopy and colonoscopy, barium enema
Specialist
Anal physiology (manometry and sensation), endoanal ultrasound, electromyography, magnetic resonance imaging, defecating proctography

Management

The management spectrum for faecal incontinence comprises conservative, medical and non-invasive interventions (pelvic floor physiotherapy, biofeedback and electrical stimulation), surgery, and recently neuromodulation (sacral nerve and posterior tibial nerve stimulation) (*Figure 1*). As with diagnosis, management should be tailored to the individual, particularly focussing on reversible and sinister causes (*Table 2*). It is important to treat the individual patient; what is acceptable to one may be completely unacceptable to another. However, patients should be forewarned that treatment may be protracted, potentially involving trials of multiple interventions and limited success (National Institute for Health and Clinical Excellence (NICE), 2007).

Conservative management

Initial management, in the absence of reversible causes, is usually conservative.

Modification of bowel habit

This aims to establish a predictable bowel habit of optimal frequency and stool consistency that is easier to control. This is achieved by patient education, and optimizing food, nutrient and fluid intake to ensure adequate hydration for those with constipation. Patients should be encouraged to experiment and exclude those foods contributing to their incontinence; they should also be encouraged to harness the gastrocolic reflex by defecating after meals, and ensuring privacy and adequate time for toileting (NICE, 2007).

Toilet access

Patients should be aware of local public toilets, and provided with a RADAR key if appropriate (which provides access to locked public toilets nationwide). Any home impediments to toileting should be addressed, if necessary by occupational therapists, and patients advised to wear easily removable clothing (NICE, 2007).

Pads and plugs

Absorbable body and bed-pads should be made available to all, although these are often limited by problems of hygiene, odour and skin breakdown (Brazzelli et al, 2002; NICE, 2007). Consequently, absorbable plugs are sometimes trialled with the aim of achieving pseudocontinence. While originally developed for those with neurological incontinence, some work suggests that they may achieve pseudocontinence in around 38% of patients with other forms of incontinence (Deutekom and Dobben, 2005).

Medical management

This also aims to establish a regimen of optimal frequency and consistency. Any contributory conditions, such as inflammatory bowel disease, should be treated appropriately.

Medications

Medications that may be contributing to incontinence (*Table 4*) should be reviewed. The goals of medications for faecal incontinence are to treat underlying diarrhoea or constipation and enhance anal sphincter function. If diarrhoea predominates, having investigated it adequately, antidiarrhoeals should be trialled. These rely on either opiate or anticholinergic activity; first choice is loperamide hydrochloride (which in addition to opioid action, also improves anal sphincter tone and rectal compliance; Read et al, 1982), followed by codeine phosphate or co-phenotrope (diphenoxylate plus atropine) (NICE, 2007). Low dose amitriptyline may also be used. With constipation, a regular bowel regimen should be established with laxatives, supplemented by manual disimpaction and tap water enemas as necessary (Bellicini et al, 2008). While medications enhancing anal sphincter function remain largely experimental, improvements have been demonstrated with sodium valproate and phenylephrine gel (Kusunoki et al, 1990; Carapeti et al, 2000a); however, findings in the latter have been variable (Carapeti et al, 2000b). Overall, unfortunately, while drugs may be effective options remain limited (Cheetham et al, 2002).

Non-invasive interventions

Pelvic floor physiotherapy

This is an established treatment for urinary incontinence (Laycock and Haslam, 2002), but its use in faecal incontinence is less well defined. Varying exercise programmes aim to condition the magnitude, duration and speed of contractions of the external sphincter (Norton et al, 2006a).

Biofeedback

Biofeedback incorporates adjuncts into this process. Repetitive inflation of a rectal balloon aims to condition patients to recognize distension at progressively lower thresholds; this can be used to allow those with high sensory thresholds (i.e. reduced sensation) to identify the need to defecate earlier, and to improve tolerance in

Figure 1. An algorithm for the management of faecal incontinence. Adapted from National Institute for Health and Clinical Excellence (2007).

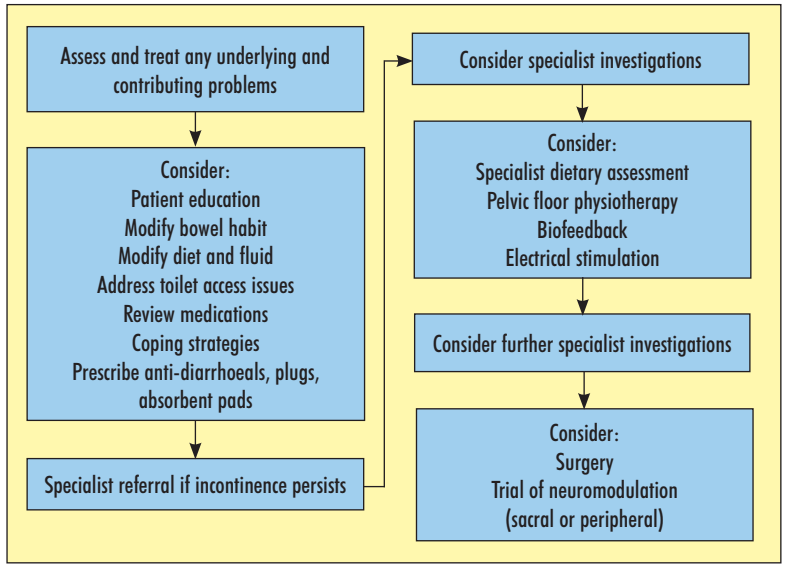

Table 4. Medications potentially exacerbating faecal incontinence

Medications affecting sphincter tone
Beta blockers, nitrates, calcium-channel antagonists, selective serotonin-reuptake inhibitors
Topical medications
Nitrates and diltiazem ointment
Medications causing loose stools
Laxatives, digoxin, orlistat, metformin
Medications causing constipation
Opioids, loperamide, antacids (aluminium-containing), tricyclic antidepressants

From National Institute for Health and Clinical Excellence (2007)

those with low thresholds and resultant urgency. This can be augmented with electromyography or pressure transducers, which convert sphincter pressures into audio or visual output to reward effort. This can be further adapted with the use of multiple balloons to teach patients to counter internal anal sphincter relaxations with volitional external sphincter contraction (Norton et al, 2006a).

However, despite widespread use of these techniques and some studies suggesting benefits (particularly in urgency), this has been disputed and a Cochrane review concluded that there was inadequate evidence on which to judge their efficacy (Norton et al, 2006b).

Electrical stimulation

Direct electrical stimulation of the anal sphincter aims to improve anorectal sensation and pressures, sometimes in conjunction with other modalities. It has been hypothesized that this may work via sensory upregulation (Norton et al, 2006b), although reduced muscle fatigue and increased vascularity have been demonstrated (Hudlicka et al, 1982). While improvements have been demonstrated in conjunction with biofeedback, this has also been disputed (Mahony et al, 2004); current consensus is that the evidence is very limited.

Surgery

Surgical interventions, advocated since Sir Alan Parks first described the postanal repair in 1975 (Parks, 1975), aim to repair a sphincter or pelvic floor defect, bolster a structurally intact but weak sphincter mechanism, fashion a neosphincter or ultimately site a stoma. Owing to its inherent risks, variable outcome and often limited supportive evidence surgery should be offered judiciously.

Repair of structural sphincter defects

The two most common procedures are the anterior sphincter repair and sphincter plication. The former is used for defects in the external anal sphincter, which are apposed or (preferably) overlapped via sutures. It is most successful in those with obstetric defects, resolving symptoms in up to 80% (Kamm, 1998) but at a significant risk of complications, sometimes mandating stoma formation. It is least successful for those with coexistent internal or multiple sphincter defects and pudendal neuropathy (Fraser et al, 2004). In those with isolated internal sphincter defects, plication can be used to narrow the anal aperture with some success (Deen et al, 1995). There is evidence with this approach that even though initial results are encouraging, they do deteriorate with time (Maxwell-Armstrong et al, 2001).

Repair of the pelvic floor and augmentation of intact sphincter mechanisms

This can be achieved by anterior levatorplasty, postanal repair, or the two in combination (total pelvic floor repair). Anterior levatorplasty involves plication of the levator ani while postanal pelvic floor repair involves strengthening the musculature posterior to the anus with a lattice of sutures, to augment the anorectal angle and puborectalis sling. Initial success rates were quoted as 81% (Parks, 1977), however, follow-up studies have reported rates of just 28% (Jameson et al, 1994), and these procedures are rarely – if ever – used in the current management of faecal incontinence.

Neosphincters

Procedures creating neosphincters are inherently more aggressive, with higher failure and morbidity rates. These comprise muscle transfer procedures, artificial sphincters and implants. In muscle transfer procedures, skeletal muscle is transposed from nearby sites and wrapped around the anal canal, and is subsequently retrained to generate a tonic contraction via an implanted nerve stimulator (Baeten et al, 1988). Donor muscles include gracilis, gluteus maximus and obturator internus, and success rates are in the region of 60% (Madoff et al, 1999), comparable to those of total pelvic floor repair (Yoshioka et al, 1999). Artificial hydraulic silastic balloon sphincters, while uncommon, improve incontinence in 50%, although at a 50% risk of significant complications (Wong et al, 2002).

Stoma placement

Often perceived as a last resort, stomas may be placed to provide a conduit (often via the appendix or ileum) for anterograde colonic irrigation (controlling defecation with timed irrigation) or a defunctioning colostomy providing symptomatic relief (Madoff et al, 1992).

Neuromodulation

Neuromodulation, the modification of nerve function, is another import from urological practice. Sacral neuromodulation has become established practice with a solid evidence base over the last decade, while peripheral neuromodulation, although enticing, remains in the early stages of development. The mechanisms by which they exert their effects are not fully understood, but likely derive from both sensory and motor neuromodulation. Such putative effects include alterations in rectal sensory perception, upregulation of striated muscle function (allowing generation of increased pressures), and reduced unwanted spontaneous anal relaxations and rectal contractions (Shafik et al, 2003; De La Portilla et al, 2009).

Sacral nerve stimulation

Originally developed for urinary incontinence (Tanagho and Schmidt, 1982), this involves application of a low voltage current to a sacral nerve root via its corresponding sacral foramen. A 2-week percutaneous trial is recommended by the National Institute for Health and Clinical Excellence for all those unable to undergo sphincter surgery (NICE, 2007). Between 56 and 92% of patients subsequently receive a permanent subcutaneous stimulator (Matzel et al, 2003; Fraser et al, 2004), and of these

41–75% achieve complete continence, with up to 100% achieving at least a 50% reduction in incontinence (Fraser et al, 2004). It is seemingly effective across the spectrum of aetiologies and in those with intact and defective sphincters, but carries a complication rate of 13% (largely infection, pain or lead migration), of whom half require relocation or removal of the device (Fraser et al, 2004). Sacral nerve stimulation is also limited by its high cost and the need to replace stimulator batteries after 8 years

Peripheral neuromodulation

Peripheral neuromodulation has been used to treat urinary incontinence, chronic pelvic pain and sexual dysfunction since 1983 (McGuire et al, 1983; Nakamura et al, 1983). However, it is still undergoing early trials in faecal incontinence. It accesses, indirectly, the same sacral nerve roots via the posterior tibial nerve (anterior to the medial malleolus), containing sensorimotor and autonomic fibres. It is technically simple to perform as an outpatient, with no requirement for anaesthesia or insertion in the operating theatre; treatment is estimated to cost less than a tenth that of sacral nerve stimulation (Klinger et al, 2000). Patients undergo anywhere from one to three sessions of 15–30 minutes per week, and while it has yet to be tested against placebo, tentative results suggest that it may represent a minimally invasive and safe alternative, or precursor, to surgery or sacral nerve stimulation (Shafik et al, 2003; De La Portilla et al, 2009).

Conclusions

Despite often being unrecognized, faecal incontinence is a common and significant cause of both psychological and physical morbidity. Its aetiology is usually multifactorial, with often complex interplay between neuromuscular and structural pathology. A thorough and structured clinical assessment is paramount, with subsequent referral for specialist investigation and management. BJHM

Conflict of interest: none.

Baeten C, Spaans F, Fluks A (1988) An implanted neuromuscular stimulator for fecal continence following previously implanted gracilis muscle. *Dis Colon Rectum* **31**: 134–7

Barnett JL, Hasler WL, Camilleri M (1999) American Gastroenterological Association medical position statement on anorectal testing techniques. American Gastroenterological Association. *Gastroenterology* **116**: 732

Bellicini N, Molloy PJ, Caushaj P, Kozlowski P (2008) Fecal incontinence: a review. *Dig Dis Sci* **53**(1): 41–6

Brazzelli M, Shirran E, Vale L (2002) Absorbent products for containing urinary and/or fecal incontinence in adults. *J Wound Ostomy Continence Nurs* **29**(1): 45–54

Carapeti EA, Kamm MA, Nicholls RJ, Phillips RKS (2000a) Randomized, controlled trial of topical phenylephrine for fecal incontinence in patients after ileoanal pouch construction. *Dis Colon Rectum* **43**(8): 1059–63

Carapeti EA, Kamm MA, Phillips RKS (2000b) Randomized controlled trial of topical phenylephrine in the treatment of faecal incontinence. *Br J Surg* **87**(1): 38–42

Cheetham MJ, Brazzelli M, Norton CC, Glazener CMA (2002) Drug treatment for faecal incontinence in adults. *Cochrane Database Syst Rev* **3**: CD002116

Deen KI, Kumar D, Williams JG, Grant EA, Keighley MRB (1995) Randomized trial of internal anal sphincter plication with pelvic floor repair for neuropathic fecal incontinence. *Dis Colon Rectum* **38**(1): 14–18

De La Portilla F, Rada R, Vega J, Gonazlez CA, Cisneros N, Maldonado VH (2009) Evaluation of the use of posterior tibial nerve stimulation for the treatment of fecal incontinence: preliminary results of a prospective study. *Dis Colon Rectum* **52**(8): 1427–33

Deutekom M, Dobben AC (2005) Plugs for containing faecal incontinence. *Cochrane Database Syst Rev* **3**: CD005086

Fraser C, Glazener C, Grant A et al (2004) *Systematic review of the efficacy and safety of sacral nerve stimulation for faecal incontinence.* Commissioned by the National Institute for Clinical Excellence. Review Body for Interventional Procedures, Aberdeen

Hudlicka O, Dodd L, Renkin EM, Gray SD (1982) Early changes in fiber profile and capillary density in long-term stimulated muscles. *Am J Physiol* **243**: 528–35

Jameson JS, Speakman CTM, Darzi A et al (1994) Audit of postanal repair in the treatment of fecal incontinence. *Dis Colon Rectum* **37**: 369–72

Jorge JMN, Wexner SD (1993) Etiology and management of fecal incontinence. *Dis Colon Rectum* **36**: 77–97

Kamm MA (1998) Faecal incontinence. *BMJ* **316**(7130): 528–32

Keating JP, Stewart PJ, Eyers AA et al (1997) Are special investigations of value in the management of patients with fecal incontinence? *Dis Colon Rectum* **40**: 896

Khullar V, Damiano R, Toozs-Hobson P, Cardozo L (1998) Prevalence of faecal incontinence among women with urinary incontinence. *Br J Obstet Gynaecol* **105**(11): 1211–13

Klinger HC, Pycha A, Schmidbauer J, Marberger M (2000) Use of the peripheral neuromodulation of the S3 region for treatment of detrusor overactivity: a urodynamic-based study. *Urology* **56**: 766–71

Kusunoki M, Shoji Y, Ikeuchi H, Yamagata K, Yamamura T, Utsunomiya J (1990) Usefulness of valproate sodium for the treatment of incontinence after ileoanal anastomosis. *Surgery* **107**(3): 311–15.

Laycock J, Haslam J (2002) *Therapetic management of incontinence and pelvic pain.* Springer, London

MacArthur C, Bick DE, Keighley MR (1997) Faecal incontinence after childbirth. *Br J Obstet Gynaecol* **104**: 46–50

Madoff RD, Williams JG, Caushaj PF (1992) Fecal incontinence. *N Engl J Med* **326**(15): 1002–7

Madoff RD, Rosen HR, Baeten CG et al (1999) Safely and efficacy of dynamic muscle plasty for anal incontinence: Lessons from a prospective, multicenter trial. *Gastroenterology* **116**: 549–556

Mahony RT, Malone PA, Nalty J, Behan M, O'Connell PR, O'herlihy C (2004) Randomized clinical trial of intra-anal electromyographic biofeedback physiotherapy with intra-anal electromyographic biofeedback augmented with electrical stimulation of the anal sphincter in the early treatment of postpartum fecal incontinence. *Am J Obstet Gynecol* **191**(3): 885–90

KEY POINTS

- Faecal incontinence is common, affecting 2% of adults at any one time, and up to 10% at some point in their lives.
- It is often multifactorial in origin, with obstetric damage, surgical intervention and idiopathic degeneration the commonest single causes.
- Specialist investigations (manometry, sensory measurements and ultrasound) improve diagnosis and treatment plans.
- Initial therapy with medications may be effective and physiotherapy or biofeedback are usually used as first-line treatments with variable success rates.
- Judicious surgical intervention, or more recently sacral and peripheral neuromodulation, may offer patients a reasonable chance of improvement.

Matzel KE, Bittorf B, Stadelmaier U, Hohenberger W (2003) Sacral nerve stimulation in the treatment of faecal incontinence. *Chirurg* **74**(1): 26–32

Maxwell-Armstrong CA, Bush D, Indar A, Speake W, Scholefield JH Armitage NC (2001) Long term results of surgery for faecal incontinence. *Colorectal Dis* **3** (Suppl 1): 83

McGuire EJ, Zhang SC, Horwinski ER, Lytton B (1983). Treatment of motor and sensory detrusor instability by electrical stimulation. *J Urol* **129**: 78–9

Nakamura M, Sakurai T, Tsujimoto Y, Tada Y (1983) Transcutaneous electrical stimulation for the control of frequency and urge incontinence. *Hinyokika Kiyo* **29**: 1053–9

National Institute for Health and Clinical Excellence (2007) *Faecal incontinence: the management of faecal incontinence in adults.* National Institute for Health and Clinical Excellence, London

Norton C, Whitehead WE, Bliss DZ, Metsola P, Tries J (2005) Conservative and pharmacological management of faecal incontinence in adults. In: Abrams P, Cardozo L, Khoury S, Wein A, eds. *Incontinence*. Health Publications, Plymouth: 1521–63

Norton C, Cody JD, Hosker G (2006a) Biofeedback and/or sphincter exercises for the treatment of faecal incontinence in adults. *Cochrane Database Syst Rev* **3**: CD002111

Norton C, Gibbs A, Kamm MA (2006b) Randomized, controlled trial of anal electrical stimulation for fecal incontinence. *Dis Colon Rectum* **49**(2): 190–6

Parks AG (1975) Royal Society of Medicine, section of proctology; meeting 27 November 1974. President's address. Anorectal incontinence. *J R Soc Med* **68**: 681–90

Parks AG (1977) Post anal pelvic floor repair (and the treatment of faecal incontinence). In: Todd IP, ed. *Operative Surgery: Colon, Rectum, and Anus.* 3rd edn. Butterworths, London: 249–54

Perry S, Shaw C, McGrother C et al (2002) Prevalence of faecal incontinence in adults aged 40 years or more living in the community. *Gut* **50**(4): 480–4

Read M, Read NW, Barber DC, Duthie HL (1982) Effects of loperamide on anal sphincter function in patients complaining of chronic diarrhea with fecal incontinence and urgency. *Dig Dis Sci* **27**: 807

Rockwood TH, Church JM, Fleshman JW et al (2000) Fecal Incontinence Quality of Life Scale: quality of life instrument for patients with fecal incontinence. *Dis Colon Rectum* **43**(1): 9–16; discussion 16–17

Saclarides TJ (1998) Endorectal ultrasound. *Surg Clin North Am* **78**: 237

Sangwan YP, Coller JA, Barrett RC et al (1996) Unilateral pudendal neuropathy. Impact on outcome of anal sphincter repair. *Dis Colon Rectum* **39**: 686–9

Shafik A, Ahmed I, El-Sibai O, Mostafa RM (2003) Percutaneous peripheral neuromodulation in the treatment of fecal incontinence. *Eur Surg Res* **35**(2): 103–7

Sultan AH, Kamm MA, Hudson CN, Bartram CI (1993) Anal sphincter disruption during vaginal delivery. *N Engl J Med* **329**: 1905–11

Tanagho EA, Schmidt RA (1982) Bladder pacemaker: scientific basis and clinical future. *Urology* **20**(6): 614–19

Tjandra JJ, Milsom JW, Schroeder T et al (1993) Endoluminal ultrasound is preferable to electromyography in mapping anal sphincteric defects. *Dis Colon Rectum* **36**: 689–92

Wald A (1983) Biofeedback for neurogenic fecal incontinence: rectal sensation is a determinant of outcome. *J Pediatric Gastroenterol Nutr* **2**: 302–6

Wald A (1994) Colonic and anorectal motility testing in clinical practice. *Am J Gastroenterol* **89**: 2109

Wong WD, Colngliosi SM, Spencer MP et al (2002) The safety and efficacy of the artificial bowel sphincter for fecal incontinence: results from a multicenter cohort study. *Dis Colon Rectum* **45**: 1139

Yoshioka K, Ogunbiyi OA, Keighley MRB (1999) Pilot study of total pelvic floor repair or GluteusMaximus transpostion for postobstetric neuropathic fecal incontinence. *Dis Colon Rectum* **42**(2): 252–7