CHAPTER 24

Committee 18

Conservative and Pharmacological Management of Faecal Incontinence in Adults

Co-Chair

C. NORTON (U.K),

W.E. WHITEHEAD (USA)

Members

D.Z. BLISS (USA),

P. METSOLA (FINLAND),

J. TRIES (USA)

CONTENTS

Conservative and Pharmacological Management of Faecal Incontinence in Adults

C. Norton, W.E. Whitehead

D.Z. Bliss, P. Metsola, J. Tries

A. INTRODUCTION

A working definition of Anal Incontinence (AI) was adopted at the last consultation [1] as:

"Anal incontinence is the involuntary loss of flatus, liquid or solid stool that is a social or hygienic problem".

It is proposed by this committee that the consultation adopt this definition, with the additional definition of "faecal incontinence" (FI) as identical except excluding flatus. FI is covered in this chapter, except where anal incontinence is specified.

This chapter covers conservative management of FI in adults. Covered elsewhere in the volume are surgical management (Committee 19), and management in children (Committee 11), people with neurological disease or injury (Committee 12) and frail older people (Committee 13). Risk factors and prevention are covered for all groups. Some techniques developed and evaluated in these specific groups may have applications to an adult population, but most have not yet been evaluated.

Conservative management is defined as any non-operative, non-drug intervention designed to improve FI incontinence or prevent deterioration. No studies were found exploring how to select patients for operative vesus conservative or drug management, nor comparing the approaches in comparable patient groups, so patient selection remains empirical. However, the committee recommends a trial of conservative and drug management in the vast majority of patients before considering surgical options because these conservative options are comparatively inexpensive and involve no significant morbidity (see algorithm). Exceptions would be patients with acute traumatic anal sphincter rupture or an endosonographically confirmed major defect in the external anal sphincter in the presence of gross faecal incontinence: these patients would be referred for surgical evaluation.

B. RISK FACTORS FOR FAECAL INCONTINENCE AND STRATEGIES FOR PREVENTION

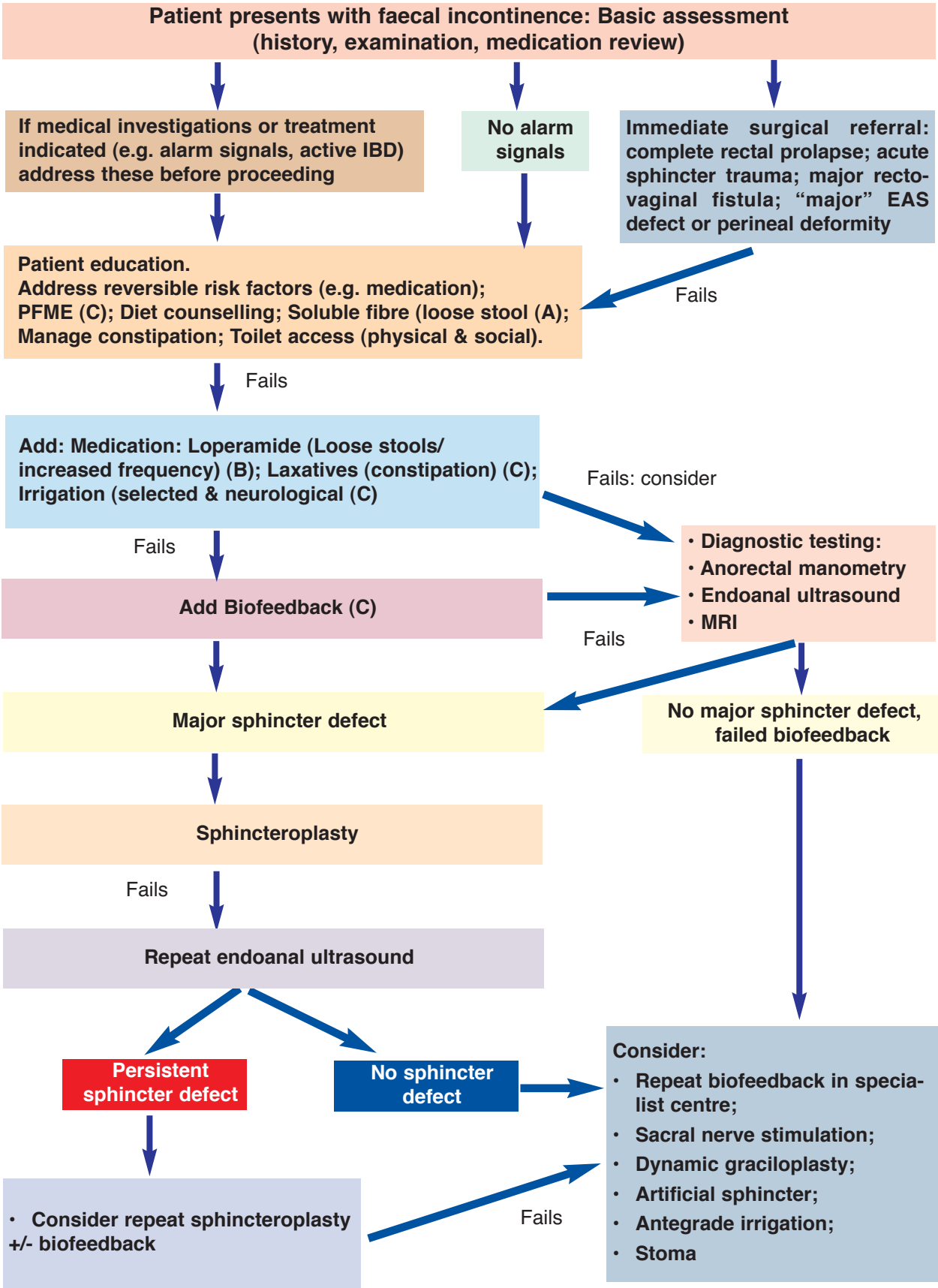
I. AIMS

The goals of this section are (a) to identify risk factors for faecal incontinence (FI) and (b) to identify and evaluate prevention studies in adults.

• Definition of Prevention

Primary prevention is defined as the elimination of risk factors. Examples are fluorination of drinking water and vaccination for diseases. Secondary prevention refers to detecting and treating diseases at an early stage when there may be few or no symptoms, in order to prevent disease progression and possible sequelae. Examples are screening programmes to

Management of Faecal Incontinence in Adults



detect mild hypertension, followed by treatment of identified cases. Tertiary prevention refers to the treatment of established disease to prevent or reverse symptoms. The distinction between secondary and tertiary prevention is sometimes blurred. The scope of this section includes a discussion of primary and secondary prevention but does not include tertiary prevention (covered in other sections).

II. METHODOLOGY

The Medline database was searched for all articles in all languages published in the last 10 years that were captured by the following search terms: epidemiology, risk factors and prevention cross-referenced with faecal incontinence or anal incontinence. These searches captured 459 articles which were reviewed for their relevance to the topic first by titles, then by abstracts, and finally by text of the articles. This search strategy was supplemented by a search of the Cochrane database and by focused searches on disease entities or other risk factors (e.g. haemorrhoids, ulcerative colitis, Crohn's disease) that were not identified as risk factors in the search but that were known to the authors to be associated clinically with FI.

The epidemiological data were evaluated on two primary criteria: representativeness and sample size. We gave preference to population-based studies of at least 1000 subjects for the identification of demographic and other major risk factors (**Table 1**). However, FI is a relatively infrequent condition affecting approximately 2.2% of the population [2, 3] and many of the diseases or conditions that are risk factors for FI are themselves rare (e.g., spina bifida, imperforate anus). Consequently, population based studies are unlikely to identify these risk factors. We therefore also included "enriched" samples which, for example, estimated the prevalence of FI in patients with these disorders. However, we avoided studies in which the method of ascertaining the sample was likely to over-estimate the association of the disease or condition with FI, for example, patients with diabetes who were referred to a gastroenterologist for evaluation.

In some instances we have reported possible risk factors on the basis of small or methodologically flawed studies when better studies were not available. We have identified these instances. **Table 2** gives surveys of prevalence in Nursing Homes.

III. PREVALENCE OF FI AND RISK FACTORS

The median prevalence of FI among high quality population-based studies is 3.0%, including both men and women, but the range is up to 17% for men and 25% for women [4]. When the data are broken down by type of incontinence, liquid incontinence is 2-3 times more common than solid stool incontinence, and incontinence for flatus is 2-3 times more common than the combination of liquid and solid (5-8). Soiling or staining of underclothes, which is often not included in the definition of FI, was reported to occur in 21% of men and 14.5% of women in one study [9].

1. Patient characteristics associated with increased risk of FI

a) Age

Nine population-based studies (Table 1 includes only good quality studies), assessed age as a risk factor: 6 of these studies found age to be a significant risk factor in both men and women [2, 3, 6, 8, 10, 11], one reported it to be significant for women only [9], one found it to be significant in men only [4], and one found no significant association with age [7]. Some of the age-related increase in prevalence of FI may be attributable to age-related declines in general health, muscle strength, mobility, and cognitive functioning, and the increased prevalence of other diseases that may contribute to FI (see below).

b) Gender

Of 8 population-based studies that surveyed both men and women (**Table 1**), exactly half [3, 8-10] found a greater prevalence in women. No study reported a significantly greater prevalence in men. A greater preponderance of FI in women, when it has been found, has usually been attributed to obstetrical injury.

c) Race

A postal survey carried out in 7,879 women who delivered babies during the same year at three hospitals – one in England, one in Scotland, and another in New Zealand – showed a higher incidence of FI in Asian women than in Caucasians (OR=3.2) [12]. A higher incidence of obstetrical injury in Asian as compared to Caucasian women was reported in two other surveys [13, 14]. The prevalence of obstetrical tears during spontaneous vaginal delivery was also

Table 1. Population Based Surveys

Source	Design	Risk Factors	Results
Nelson et al [1995]	Random digit dialing telephone survey in 6,959 community dwelling adults, all ages. FI definition includes flatus.	Age Female sex Physical limitations Poor general health	Adjusted OR=1.01 (CI, 1.01-1.02) OR=1.51 (CI, 1.10-2.11) OR=1.82 (CI, 1.20-2.74) OR=1.64 (CI, 1.48-1.91) Overall prevalence 2.2%
Perry et al [2002]	Postal survey in 15,904 randomly selected community dwelling adults aged ≥40 years. FI definition not include flatus but required frequency of "several times a month."	Age Gender	Significant association No difference Overall prevalence 3.0% age ≥40
Kalantar et al [2002]	Postal survey of 477 randomly selected community dwelling Australian adults, all ages. FI definition excluded flatus and acute diarrhea.	Age Female sex Perianal injury/surgery Loose stool Stool urgency Poor general health Straining, hard stools	Significant association No difference Significant association Significant association Significant association Significant association No association Prevalence 2% solid, 9% liquid
Walter et al [2002]	Postal survey of 2000 randomly selected Swedish community dwelling adults aged 31-76. Distinguished flatus and soiling underwear from loss of solid or liquid. Threshold was at least monthly.	Age Female sex Loose stools	Significant association in women Significant association for solid or liquid stool. Men reported more soiling of underwear. Significant association Overall prevalence not given
Edwards, Jones [2001]	Interviews in home of 2,818 men and women ≥65 yrs. FI defined by the question,"Do you have any difficulty in controlling your bowels?"	Age Female sex Anxiety & depression Physical disability Urinary incontinence	Significant association Significant association Significant association Significant association Significant association Overall prevalence 3% age ≥65
Nakanishi et al [1997]	Interviews in home of 1,405 men & women ≥65 yrs.	Age Female sex Physical disability Stroke Dementia	Significant association No association Significant association Significant association Significant association Prevalence 8.7% men, 6.6% women
Roberts et al [1999]	Postal questionnaire in age-stratified population sample of 778 men and 762 women aged ≥50 yrs. FI definition did not include uncontrolled flatus.	Urinary incontinence Age	> half of FI men and women had urinary incontinence Significant for men but not women Prevalence 17.0% for men and 24.6% for women
Bytzer et al [2001]	Postal survey of 8657 randomly selected Australian adults (60% response rate), including 423 with self-reported DM.	Diabetes mellitus	FI "Sometimes": 12.8% for DM, 3.8% for controls (p<.001). FI "Often": 2.6% vs. 0.8%, OR=2.74 (CI, 1.40-5.37).
Chen et al [2003]	Door-to-door survey of 1,253 Taiwanese women representative of the population. FI definition included flatus.	POP Parity ≥1 Prior GYN surgery Hypertension Overactive bladder	OR=3.2 (CI, 1.1-8.9) OR=3.4 (CI, 1.2-9.5) OR=1.8 (CI, 1.1-2.9) OR=2.4 (CI, 1.2-4.9) OR=3.2 (CI, 1.6-6.7) Prevalence 2.8% FI, 8.6% flatus
MacLennan et al [2000]	Interviews in homes of 3,010 men & women ≥15 yrs. Distinguished incontinence for flatus from incontinence for stool.	Age Female sex Parity ≥1 Sphincter repair Vaginal vs. C-section Vaginal vs. Instrumental	Significant association OR=1.7 (CI, 1.3-2.2) for flatus; OR=1.6 (CI, 1.0-2.5) for stool Significant association No association No association No association Prevalence 2.3% for FI & 6.8% for flatus in men; 3.5% for FI & 10.9% for flatus in women
Fornell et al [2003]	Postal survey of 1000 randomly selected 40 year-old and 1000 randomly selected 60 year-old Swedist women. FI defined as leakage >1/month; UI defined as leakage weekly or more often.	3rd or 4th degree tear Parity Vacuum extraction Urinary Incontinence Pelvic Heaviness	OR=9.1 (CI, 3.0-27.3) for solid stool No significant association No significant association OR=5.9 (CI, 2.4-14.6) for solid stool OR=3.3 (CI, 1.6-7.0) for solid stool Prevalence 8.9% FI, 11.4% flatus

Table 2. Nursing Home Surveys

Source	Design	Risk Factors	Results
Nelson et al [1998]	HCFA minimum data set for Wisconsin skilled nursing facilities for 1992 & 1993.	Urinary incontinence	OR=12.6 (CI, 11.5-13.7)
		Tube feeding	OR=7.6 (CI, 5.6-10.4)
		Loss of ADLs	OR=6.0 (CI, 4.7-7.7)
		Diarrhoea	OR=3.3 (CI, 2.7-4.2)
		Truncal restraints	OR=3.2 (CI,4.7-7.7)
		Pressure ulcers	OR=2.6 (CI, 2.2-3.0)
		Dementia	OR=1.5 (CI, 1.4-1.7)
		Impaired vision	OR=1.5 (CI, 1.4-1.7)
		Faecal impaction	OR=1.5 (CI, 1.1-2.1)
		Constipation	OR=1.4 (CI, 1.3-1.6)
		Stroke	OR=1.3 (CI, 1.2-1.5)
		Male sex	OR=1.2 (CI, 1.1-1.3)
Johanson et al [1997]	All 388 residents of 5 nursing homes, both skilled & unskilled. Questionnaire was completed by patient if possible, otherwise by investigator or nursing staff. FI defined as any involuntary leakage or soiling.	Diarrhoea	OR=8.0 (CI, 3.0-21.0)
		Wheelchair dependent	OR=2.7 (CI, 1.7-4.0)
		Dementia	OR=4.3 (CI, 2.8-6.8)
		Male sex	OR=2.5 (CI, 1.5-4.1)
		Age <65 yrs	OR=2.6 (CI, 1.0-6.5)
		Daily exercise (-)	OR=0.5 (CI, 0.3-0.7)
		Hard stools (-)	OR=0.2 (CI, 0.1-0.7)
Chassagne et al [1999]	Incidence of new-onset FI in 1,186 residents of nursing homes or long-term care facilities. 234 (20%) developed FI within 296 days. Risk of long-lasting FI reported.	Hx urinary incontinence	OR=2.9 (CI, 1.8-2.6)
		Decreased mobility	OR=1.8 (CI, 1.1-3.0)
		Hx dementia	OR=2.1 (CI, 1.2-3.5)
		MMSE score <15	OR=2.5 (CI, 1.4-4.4)
		Associated mortality	16% vs. 6.7% (p<.001)

greater in Hispanic subjects and Filipinos [13]. No published data were found on the relative rates of FI in African Americans compared to Caucasians.

d) Diarrhoea

The only two population-based studies to assess loose stools as a risk factor [6, 9], reported an association between loose stools and FI. This is consistent with reports that FI is more prevalent in patients with irritable bowel syndrome [15], patients with illnesses that produce diarrhoea [16-18], and in people who run long distances for exercise [19]. The association between FI and diarrhoea is even more robust in nursing home studies [20, 21] Whenever the frequency of FI is reported separately for solid, liquid, and gas, liquid incontinence is found to be more frequent than solid stool incontinence, and gas is found to be the most common type of incontinence [5, 8, 22].

Potentially preventable causes of diarrhoea, which is a major risk factor for FI, include drugs, dietary supplements, and some foods. Drugs known to cause diarrhoea as a side-effect include antibiotics, especially the erythromycin analogs, tegaserod, the 5HT4 agonist used to treat constipation-predominant irritable bowel syndrome, the serotonin reuptake inhibitor class of antidepressants, digoxin, and laxatives. Chronic laxative dependence or abuse may cause frequent diarrhoea. Food supplements that have been reported to cause diarrhoea include lactose (in lactase deficient individuals), fructose, sorbitol, aspartame, and other artificial sweeteners that are poorly absorbed, and fat substitutes such as olestra. Some natural foods such as prunes and figs may also cause diarrhoea. The research literature has not established that these foods, food additives, and drugs cause FI, but it has established a link to diarrhoea.

e) Constipation

Constipation was found to be a significant positive risk factor for FI in one nursing home survey [21], but in another study, hard stools appeared to be protective [20]. Constipation is considered to be the most common aetiology for FI in children (often referred to in the paediatric literature as encopresis when there is no recognized structural anomaly to explain the incontinence) [23, 24] The mechanism that is presumed to explain constipation-related faecal incontinence is overflow: a mass of hard stool in the rectum or sigmoid blunts sensitivity for perceiving the movement of new stool into the area and also reflexly dilates the internal anal sphincter allowing liquid stool to seep out [23].

f) Poor general health

In population-based surveys, poor general health is a risk factor for FI that is independent of diarrhoea [6]. FI is associated with increased mortality both in community dwelling older subjects [11] and in nursing home patients [25].

g) Physical limitations

Three population-based surveys assessed physical limitations and found them to be risk factors for FI [3, 10, 11]. In nursing home patients, mobility impairment is consistently found to be a predictor of FI [20, 21, 25].

h) Cognitive impairment

Dementia is also a significant predictor of FI both in the community [11] and in nursing homes [20, 21, 25]. Stroke, which may be associated with either cognitive impairment or focal lesions in the neurological pathways responsible for reflex and voluntary control mechanisms, is also a risk factor for faecal incontinence [11, 21]. The prevalence of FI in those with mild learning disability is little different from that of the general population, rates for those with moderate and severe learning disability are higher than population norms and are similar to each other, prevalence is substantially higher in those with a profound learning disability [26]. Nevertheless, around half of those with a profound disability will acquire bowel control by adulthood.

i) Physical exercise and work

Exercise has been found to increase propagated colonic contractions [27, 28], which is the presumed mechanism for runner's diarrhoea. Exercise is often recommended as a means of promoting a regular bowel habit with complete evacuation, but exercise may not be feasible for people with mobility difficulties. The epidemiological literature is consistent in showing less constipation in people who report that they exercise [29]. It is known that endurance running is associated with diarrhoea and FI in over 10% of individuals [30]. There are no studies on prevention of runner's diarrhoea and faecal incontinence, although presumably avoiding the activity is an option.

Excessive exercise may also be a factor in rectal prolapse. This is seen in young women with anorexia nervosa who combine excessive exercise with extreme nutritional impairment, presumably thus compromising tissue quality. Women whose jobs involving a lot of heavy lifting are more likely to need vaginal prolapse surgery than the general population (OR 1.6) [31], but it is not known if they are also at increased risk of FI. It is not known if avoidance of heavy lifting would prevent FI development in an at-risk population.

j) Weight

Overweight or obese body mass index (BMI) is associated with an increased risk of anal and faecal incontinence in women, with a BMI over 30 having an OR of 1.8 for gas incontinence, 2.5 for loose stool incontinence and 1.3 for solid stool incontinence when compared to women with a normal BMI [i.e. <25) [5]. It is not known whether weight loss would improve FI in obese women, nor if weight gain prevention strategies would contribute to preventing FI in adults.

2. Obstetrical and other injuries to the pelvic floor

Table 1 shows population-based studies some of which assessed obstetrical history, and **Table 3** shows surveys of samples enriched by recruitment from obstetrical hospitals or urogynaecology clinics.

a) Parity

Three out of 4 surveys found that parity was a risk factor for FI. The first vaginal delivery carries the greatest risk of new onset FI [32], and each subsequent delivery adds to that risk [5, 7, 8, 33].

b) Forceps delivery

Three of 4 studies show that forceps delivery is a risk factor for FI, and one of two studies shows that forceps delivery is a risk factor for sphincter tear.

c) Vacuum extraction

The evidence here is more equivocal, one of three studies show that vacuum extraction is a significant risk factor for FI, and one (of one) study shows that vacuum extraction increases the risk of sphincter tear.

d) Episiotomy

Two of two studies showed a significant association between midline episiotomy and sphincter tears, but the single study looking at the risk of FI found no significant association with episiotomy.

e) Large baby

Two of two studies [32, 34] assessing the effects of having a large baby on sphincter tears were positive, as was a study of the effects of gestational age [32], however, a single study examining the impact of baby size on the risk of FI showed no association [33].

Table 3. Obstetric Samples

Source	Design	Risk Factors	Results
MacArthur et al [2001]	Postal questionnaires sent to all women delivered during 1 year at 3 hosptials: one in Scotland, one in England, and one in New Zealand. Questionnaires completed 3 months post-partum. N=7879 (71% response)	Forceps delivery C-section Age >35 yrs Asian origin Vacuum extraction Episiotomy Body mass index	OR=1.94 (CI, 1.30-2.89) OR=0.58 (CI, 0.35-0.97) OR=1.75 (CI, 1.04-2.94) OR=3.21 (CI, 2.04-5.05) No association No association No association
Faltin et al [2001]	Questionnaire study in 666 women from general outpatient clinic, 298 from antenatal clinic, 264 from urogynecology, and 984 from a population sample. FI definition was solid, liquid, or flatus at least monthly.	Parity Anal sphincter tear Baby over 4 Kg Operative delivery	OR=3.1 (CI, 1.6-6.0) OR=4.4 (CI, 2.0-9.1) No association Univariate but not multivariate association
MacArthur et al [1997]	Home interview by midwife about 10 months after delivery in 906 women. Assessed new onset FI for solids or liquids.	Vacuum extraction Forceps delivery C-section (elective)	Significant association Significant association Trend favoring protective effect
Fenner et al [2003]	Medical chart review of 2,858 primiparous mothers delivered vaginally at U. Michigan. Assessed risk of 3rd & 4th degree lacerations. Questionnaire also mailed at 6 months but response rate was only 29%.	Age Baby wt >4 Kg Forceps delivery Vacuum delivery Midline episiotomy	OR=1.04 (CI, 1.02-1.06) OR=2.19 (CI, 1.61-2.99) OR=4.75 (CI, 3.43-6.57) OR=3.51 (CI, 2.64-4.66) OR=2.24 (CI, 1.81-2.77)
Zetterstrom et al [1999]	For dependent measure of sphincter tears, hospital records of 845 women evaluated. FI was assessed by postal questionnaire at 0, 5, & 9 months postpartum, but data on relative risk were not presented.	First delivery Gesta age >294 days Fundal pressure Midline episiotomy Increasing foetal weight	OR=9.8 (CI, 3.6-26.2) OR=2.5 (CI, 1.0-6.2) OR=4.6 (CI, 2.3-7.9) OR=5.5 (CI, 1.4-18.7) OR=1.3 (CI, 1.1-1.6)

3. SEQUAELAE OF GI SURGICAL PROCEDURES

a) Colectomy and ileoanal anastamosis

Because ulcerative colitis and familial polyposis both convey a high risk of colon cancer, the colon is often removed prophylactically. While a number of variations in surgical technique have been described, the commonest procedure is to create a neorectum from loops of ileum sewn together to create a pouch and to connect this to the anal canal. A temporary ileostomy is usually performed to give the pouch time to heal. Post-operatively, 25-35% of these patients have daytime FI [35-38] and 32-52% have nocturnal FI [35, 35, 36]. Fazio and colleagues [38] reported that the preoperative frequency of FI was as great in their series of patients as was post-operative FI. The mechanisms that lead to FI in this population include frequent bowel movements (8 or more per day), high pouch pressures that exceed anal canal pressures, and high amplitude contractions of the pouch [39]. Such pouch contractions are recorded in continent as well as incontinent patients with an ileal pouch because the pouch is constructed from innervated bowel, however, the contractions produce higher pouch pressures in the incontinent patients.

When it is possible to preserve the rectum, the ileum can be sutured directly to the rectum, substantially reducing the risk of FI [40]. When bowel resection is performed for the treatment of colon or rectal cancer, some or the entire colon may be preserved, and the remaining colon may be sutured directly to the anal canal or it may be used to create a pouch that is connected to the anal canal. This is associated with a lower incidence of FI (estimated at 18%) according to some authors [41, 42], but others [43] reported a rate of 49% FI following colo-anal anastamosis.

One randomised controlled study has investigated the use of daily irrigation of a colonic J-pouch prior to ileostomy closure. Irrigation was not found to improve post-closure nocturnal continence or defaecation frequency [44].

b) Internal anal sphincterotomy

Patients with chronic anal fissure or haemorrhoids may be offered internal anal sphincterotomy (slit in the internal anal sphincter for 50-60% of its length to reduce anal canal pressures). In a large series of 585

patients with chronic anal fissures treated in this fashion at the Mayo Clinic, 45% developed FI at some point in their recovery. However, this tended to improved with time from surgery, and at follow-up an average of 72 months after surgery, 11% reported FI [45].

c) Radical prostatectomy for prostate cancer

Published prevalence rates of FI following radical prostatectomy alone range from 9% [46] to 15% [47]. In the largest survey, Bishoff and colleagues [48] reported that prostatectomy by the retropubic approach was associated with FI in 17% of cases whereas prostatectomy by the perineal approach was associated with FI in 32% of cases, the loss of moderate to large amounts of stool was reported by 4% and 10% respectively. Rates of FI are higher when prostate cancer is treated by radiation therapy [47]. However, these differences may be confounded by differences in severity of disease before treatment, extent of resection, and dose of radiotherapy. However, in some large unpublished series FI has not been noted as an adverse event (Wein A.J., personal communication) and other studies do not mention FI at all.

d) Haemorrhoidectomy

A large series of 507 patients who received the Milligan-Morgan surgical treatment for haemorrhoids were followed up by postal questionnaire 2-11 years after surgery (average of 6 years). A total of 33% [139/507) reported anal incontinence including 72 who were incontinent of gas only, 56 who were incontinent to liquid faeces, and 11 who were incontinent to solid faeces [49]. Other reports of surgical treatment for haemorrhoids list a lower incidence of FI [50], but only cases with loss of liquid or solid stool are usually reported.

4. Sequelae of radiotherapy for cancer

The prevalence of FI following external beam radiation therapy for prostate cancer ranges from 14% [46] to 21% [47]. One group estimated the prevalence at up to 46% for a mixed group most of whom had been treated with both surgery and radiotherapy [51]. Radiotherapy for cervical cancer is associated with FI in 25% of cases compared to 8% for cervical cancer patients treated exclusively by surgery [52]. The mechanism through which radiotherapy contributes to FI is believed to be a decrease in rectal compliance [53], leading to increases in symptoms of urgency and loose stools [52][47].

5. Diseases that predispose to FI

a) Urinary incontinence and pelvic organ prolapse

In community-based surveys, urinary incontinence is strongly associated with FI [4, 5] and overactive bladder [7] in both men and women. Among nursing home patients, urinary incontinence is an even stronger risk factor for faecal incontinence [21, 25]. Pelvic organ prolapse is also a significant risk factor for FI [5, 7]. Further research is needed to understand these associations. The comorbidity of FI with urinary incontinence and pelvic organ prolapse may reflect involvement of the whole pelvic floor, or alternatively, the comorbidity of urinary incontinence with FI may help to identify patients whose FI is related to dementia or mobility impairment.

b) Diabetes mellitus

Bytzer and colleagues [54) carried out a large population-based postal survey in 8,657 adults including 423 with self-reported diabetes mellitus (DM). The response rate was 60.0%. When patients with DM were contrasted to the remainder of the sample the frequency of faecal incontinence occurring at least "sometimes" was 12.8% vs. 3.8% ($p<.001$) and the prevalence of faecal incontinence occurring "often" was 2.6% vs. 0.8%. The odds ratio (after adjusting for confounders) was 2.74 (CI, 1.40-5.37). The prevalence of faecal incontinence was shown to be related to self-reported degree of glycaemic control. These results were confirmed by two other studies that contrasted patients from a diabetes register [55] or a diabetes clinic [22]. The risk of faecal incontinence among patients with DM is known to be related to weakness of anal canal resting and squeeze pressures and impaired sensation in the rectum [56, 57], and these physiological defects are related to duration of DM and the presence of microcirculatory abnormalities and autonomic and peripheral neuropathies [56].

c) Spinal cord injury

Traumatic spinal cord lesions result in substantial or complete denervation of pelvic floor muscles and loss of voluntary control over the external anal sphincter. However, many of these patients avoid faecal incontinence because they are constipated due to delayed whole gut transit and/or hyper-reflexia of the external anal sphincter. Occasional faecal incontinence is reported by 33-66% [58-60] but frequent FI (more than monthly) is limited to 11% [61] to 14% [60, 62]. Approximately 70% require mechani-

cal or manual assistance to initiate defaecation [62]. In patients with congenital spinal cord lesions (spina bifida), anorectal dysfunction may be more common: 53% report that they soil regularly [63]. As with traumatic spinal cord lesions, the majority of patients with spina bifida are constipated, which reduces the frequency of faecal incontinence that would otherwise occur in these patients who have partial or complete disruption of the efferent innervation to the pelvic floor muscles [64].

d) Stroke

Two large studies have assessed the incidence of faecal incontinence following stroke. In the Copenhagen Stroke Study of 935 consecutive admissions for stroke [65], 34% were fully incontinent and 6% were partially incontinent on admission to the hospital, 6 months later, 5% were fully incontinent and 4% were partially incontinent. In a study of 1069 patients taken from the South London Stroke Register [66], 29.7% were faecally incontinent 7-10 days after stroke, 10.8% were still incontinent at 3 months, 10.9% at one year, and 15.0% at 3 years. These data suggest that faecal incontinence is transient for the majority of patients affected, but the prevalence of faecal incontinence remains elevated compared to population norms at one year and shows little further improvement. A study of 186 stroke patients in Spain showed a similar pattern: 56% had FI at admission, and 22% remained incontinent 6 months later. Risk factors for FI include age, severity of stroke, diabetes, and comorbidity of other diseases [65].

e) Traumatic brain injury

An excellent study [67] of the prevalence of FI following traumatic brain injury was carried out in 1,013 patients consecutively enrolled in any of 17 acute rehabilitation facilities. Prevalence rates were 68% at admission, 12.4% at discharge, and 5.2% at one year follow-up. The risk of incontinence at each time point was significantly related to all measures of the severity of brain injury including Glasgow Coma Scores and length of stay. In addition, at discharge from the rehabilitation facility, FI was significantly associated with pelvic fracture, urinary tract infection, and patient age (older patients were more likely to be incontinent). At one-year follow-up, FI was significantly associated with urinary tract infection and patient age. Patients with FI were more likely to be discharged to an institution rather than to return to their homes.

f) Multiple sclerosis

FI is reported by 29% [68] to 38% [69] to 51% [70] of multiple sclerosis patients living in the community, and it is frequent in 5% [68] to 25% [70]. Among 14,000 nursing home residents with multiple sclerosis, FI was present in 58% and occurred more than twice a week in 7.5% [71]. Incontinence in this group is associated with weak strength of contraction of pelvic floor muscles, a low threshold for elicitation of the internal anal sphincter inhibitory reflex, and impaired sensation for rectal filling [72, 73]. Approximately half of patients with multiple sclerosis are also constipated, but constipation seems to occur about equally often in multiple sclerosis patients with and without FI [70].

g) Imperforate anus

High anal atresia is associated with faecal incontinence 85% of the time and low anal atresia about 57% of the time [74]. The surgical correction of high anal atresia involves identifying the striated external anal sphincter and pulling the healthy portion of the bowel down through this sphincter to create an anus. Contributing causes of incontinence are absence of an internal anal sphincter (passive barrier to soiling), weak contraction of the external anal sphincter, and decreased compliance of the neorectum [75-78]. The outcome of surgical repair is improving with improved surgical techniques and the use of the Malone antegrade colonic enema technique, but 10-30% of these patients remain totally incontinent for faeces [79].

h) Haemorrhoids

A significant number of patients with prolapsing haemorrhoids (Grade 3 and 4) experience faecal soiling, although this has not been the specific focus of any study. Johansson and colleagues [49] reported that 21% of 507 patients treated for haemorrhoids listed hygiene or soiling as an indication for seeking treatment. Following treatment with the Milligan-Morgan procedure, 24% of patients who had not listed soiling or hygiene as an indication for surgery developed new onset FI.

i) Ulcerative colitis and Crohn's disease

Faecal incontinence is more common in patients with inflammatory bowel disease, although the precise prevalence has not been the focus of study and is not known. Two mechanisms are recognized for this association: Both ulcerative colitis and Crohn's disease are associated with diarrhoea, which is a risk factor for faecal incontinence. Crohn's disease is also associated with the development of anal fistulae that may drain liquid stool to the skin surface and that may create anatomical defects in the external anal sphincter.

IV. PRIMARY PREVENTION

Table 4 summarizes the modifiable risk factors identified by our literature review, all of which are potential targets for primary prevention. The first of these targets, preventing the spread of infectious causes of diarrhoea, is largely achieved in developed countries by providing safe water and sewage disposal in urban areas and by inspecting wells and sewage treatment facilities in rural areas. This remains a major problem in developing countries, however.

Table 4. Targets for Primary Prevention

Infectious causes of diarrhoea
Obstetrical practices: episiotomy
Internal anal sphincterotomy for anal fissures or haemorrhoids

Episiotomy, especially midline episiotomy, has been shown to increase the risk of sphincter laceration rather than to reduce it, and some urogynecologists now recommend avoiding episiotomy [80].

Vaginal childbirth is itself a significant risk factor for sphincter lacerations leading to FI, and this risk is augmented when forceps or vacuum extraction are required. There is evidence from retrospective studies that Caesarean delivery reduces the risk of postpartum FI [12], although other, smaller retrospective reports failed to show any significant benefit from Caesarean section [81]. There is also evidence for a small but significant increase in the risk of maternal morbidity and mortality associated with Caesarean delivery [82]. A published decision analysis concludes that elective Caesarean delivery should be offered to women as an option, and surveys suggest that a sizable minority of obstetricians would prefer elective Caesarean delivery to vaginal delivery for themselves [83, 84]. However, this issue is very contentious [85]. There is one randomised controlled trial on this issue: in the Term Breech Trial [86], 1940 women from 110 centres worldwide who had a single foetus in breech position at term were randomised to elective Caesarean or vaginal delivery, resulting in 725 Caesarean deliveries and 456 vaginal deliveries. There were no differences in the rate of FI (0.9% for C-section vs. 1.1% for vaginal delivery) or flatus incontinence (10.8% for C-section vs. 9.1% for vaginal delivery) at 3 months postpartum follow-up. There is currently no consensus among gynaecologists and obstetricians as to whether elective Caesarean section should be offered as a means of primary prevention of obstetrical trauma to the pelvic floor and FI.

Primary prevention of FI secondary to obstetrical injury could also take the form of providing pelvic floor muscle training prior to or immediately following childbirth. Pelvic floor muscle training is often recommended by obstetricians as preparation for childbirth, but no published studies have evaluated their benefit for primary prevention. However, see below for studies of pelvic floor muscle training used as secondary or tertiary prevention.

Pelvic floor muscle training was investigated as a primary prevention measure for patients undergoing colectomy with ileoanal pouch procedure [87]. Twenty-six patients were randomised to receive either 5 weeks of exercises or no additional treatment prior to their surgery. There was no significant difference in the incidence of FI following surgery, although there was a trend favouring the group that performed exercises. This study may have been underpowered to detect a clinically significant benefit.

Internal anal sphincterotomy—slitting the internal anal sphincter longitudinally to reduce anal canal pressure – may be used to treat anal fissures, haemorrhoids, or Hirschsprung's disease. Nyam and colleagues [45] have shown that this procedure is associated with a high incidence of FI. It is possible that conservative guidelines for minimizing the use of sphincterotomy would reduce the incidence of FI, but none have been proposed or tested.

Diarrhoea is a major risk factor for FI, and a number of drugs, food supplements, and foods can cause diarrhoea. In patients who are at increased risk of FI for other reasons such as age or history of obstetric injury, it would be appropriate to screen for these agents and to eliminate them. However, there are no published data on the benefits of this approach.

V. SECONDARY PREVENTION: EARLY DETECTION AND TREATMENT

The greatest obstacle to secondary prevention is the reluctance of patients to report their FI to physicians and to request care. The proportion of patients with FI identified through surveys who report they have

discussed this problem with their doctor ranged from 5% to 27% [6, 33, 88].

Table 5 identifies a variety of patient characteristics, diseases, surgical procedures, and radiological treatments that are associated with a substantial risk of FI. Since patients are reluctant to report FI and to seek help, physicians should be alert to the possibility of FI in these patient groups, they should routinely inquire about FI and should offer treatment or referral.

Table 5. Targets for Secondary Prevention Through Early Recognition

Patient characteristics:
Dementia/cognitive impairment
Physical limitations/ impaired mobility
Diseases and disorders:
Urinary incontinence
Pelvic organ prolapse
Haemorrhoids, grade 3 and 4
Irritable bowel syndrome
Diarrhoea
Constipation
Diabetes mellitus
CNS injury: stroke, head injury, Alzheimer's,
Spinal cord injury: traumatic cord injury, spina bifida
Multiple sclerosis
Congenital anorectal anomalies: imperforate anus
Surgical interventions:
Vaginal delivery with sphincter laceration
Instrumented vaginal delivery
Colectomy, with or without ileal reservoir
Internal anal sphincterotomy for anal fissure, haemorrhoids, Hirschprung's disease
Radical prostatectomy, especially by perineal approach
Drugs and Diet
Drugs that cause diarrhoea as a side-effect
Foods that cause diarrhoea: dairy products in lactase deficient individuals, some fruits
Food additives that cause diarrhoea or gas: artificial sweeteners
Radiological treatment of pelvic cancer

Pelvic floor muscle training and education have been evaluated as secondary/tertiary prevention strategies in a small number of studies. Glazener and colleagues [89] compared nurse assessment plus reinforcement of pelvic floor muscle training and bladder training to standard management in 747 women with urinary incontinence 3 months following delivery. FI was present as a comorbid condition at baseline in 57/371 in the active treatment group and in 54/376 in the standard care condition. At follow-up 12 months after delivery, the prevalence of FI was significantly lower in the active intervention group compared to the control group (4.4% vs. 10.5%, p<.012). However, other studies have suggested no benefit for the treatment of early onset FI with pelvic floor muscle training: Meyer and colleagues [90] performed a prospective, randomised controlled trial in which half of 107 primiparous women received 12 weeks of pelvic floor exercises with biofeedback and electrostimulation beginning 9 weeks after delivery. Assessment was at 10 months after delivery. The incidence of FI was low in this study (4-5%) and was not significantly different in the group receiving biofeedback. Two other studies whose primary aim was to treat urinary incontinence with pelvic floor exercises and education, reported that these exercises did not reduce the incidence of FI relative to the control group [91, 92].

Further research is needed to evaluate low-cost and widely available treatments (e.g., patient education, pelvic floor exercises) that could be employed by community-based health care providers for both primary and secondary prevention. These studies need to be adequately powered (large), they need to characterize the at-risk population that is being targeted (e.g., different prevention strategies may be appropriate for childbirth, frailty, and dementia), they should employ large samples of at-risk individuals, and they should include direct and indirect health care costs as secondary outcome measures. Further research is also needed that could identify early precursors of FI such as straining to defaecate over a period of many years, such studies may help to explain the late onset of FI in some patients and may identify preventable risk factors.

Recommendations for Primary Prevention

1. Encourage and support public hygiene measures to reduce diarrhoeal diseases (Grade A).
2. Discourage episiotomy except in restricted circumstances (Grade B).
3. Discourage the use of internal anal sphincter myectomy for treatment of anal fissure and haemorrhoids (Grade A).
4. Continue the debate on elective Caesarean delivery to prevent sphincter laceration, but no recommendation is given by this working team.

Recommendations for Secondary Prevention

1. Because less than 1/3 of patients with FI report this to their physician, we recommend that health care providers aggressively investigate all patients for FI who present with the most common risk factors: urinary incontinence, pelvic organ prolapse, diarrhoea, vaginal delivery with sphincter laceration, multiparity, mobility impairment, dementia, and other neurological conditions. See Table 2 for other, less prevalent conditions that are associated with a high risk of FI (Grade C).
2. In patients with frequent, loose stools, screen for drug side-effects, lactose intolerance, and high intake of artificial sweeteners or other foods likely to cause diarrhoea (Grade C).
3. Research is needed to assess the benefits of pelvic floor muscle training for the prevention of FI in women undergoing vaginal delivery.

C. EDUCATION & "LIFESTYLE"

Most patients do not know how the bowel works and what might improve bowel function. Many also have attitudes to defaecation that are influenced by stigma and taboos prevalent in their particular family and wider cultural group within a society [93].

Expert opinion supports the use of general health education, patient teaching about bowel function and advice on lifestyle modification [94, 95], but the evidence base does not exist. Unlike urinary incontinence, there have been few "lifestyle" associations identified with FI and little is known about whether interventions designed to reduce potential risk factors might improve FI. Diet and fluid intake are covered below.

The following databases were searched using the keywords "faecal incontinence" and the relevant intervention: Cochrane library, AHMED 1985-2004, BNID 1994-2004, EMBASE 1974-2004, MEDLINE 1951-2004, PsychInfo 1987-2004. All seemingly relevant articles were retrieved in hard copy, and the reference lists searched for further studies.

I. PHYSICAL EXERCISE AND WORK

One study in a nursing home population had found that a structured daily exercise programme, combined with increased fluid intake and regular toileting opportunities, significantly improved FI and increased the percentage appropriate toilet use compared with controls [96, 97]. No studies on the impact of physical exercise on FI or bowel habit in non-institutionalised adults were found. Abdominal massage in the direction of colonic peristalsis is often recommended [98].

II. SMOKING

Nicotine is thought to slow upper gut motility and increase total transit time [99], but it seems that it can speed recto-sigmoid transit [100], and this stimulation of distal colonic motility may exacerbate a tendency to faecal urgency. This fits with many anecdotal reports that smoking a cigarette facilitates initiation of defaecation. Smoking is a known risk factor for urinary incontinence and genital prolapse (OR 2.9) [101], presumably via chronic coughing. No association has been found between antenatal smoking and postnatal FI [102]. Smoking cessation is anecdotally reported to be useful for reducing urgency of defaecation, but no formal studies were identified.

III. MEDICATION SIDE-EFFECTS

Medication used specifically to treat FI is covered in section F below. A vast number of drugs have direct or indirect effects on the gastrointestinal system, tending to cause constipation, diarrhoea, or either in different people. A careful drug history (including all over the counter or "herbal" preparations) should be taken in each person with FI. It is beyond the scope of this chapter to review drug effects in detail, and prescribers should be aware of unintended side-effects on faecal incontinence. No studies were identified that evaluated the benefits of changing the patient's drug regimen on FI.

One single case report was found reporting that a combination of olestra in the diet and orlistat given to treat obesity led to symptoms of FI, which resolved when the olestra was stopped [103]. Patients reporting soiling while on treatment with orlistat for

obesity have been found to have pre-existing impaired anorectal function, thus predisposing them to symptom development [104].

V. TOILET FACILITIES

In individuals who have physical or mental impairments, adverse physical or social environments may impair the ability to maintain continence. This is particularly relevant to those in institutional settings (see Committee 13). Adverse environmental factors include: (a) toilet facilities that are inaccessible or that lack privacy so that the person avoids using the toilet, (b) care providers who are insensitive to the individual's needs and bowel habit, (c) clothes which are difficult to manipulate in a hurry, and a variety of other factors which vary with abilities of the individual. The toilet itself may be too high, leaving the feet dangling and thus making abdominal straining difficult. The toilet may be too low, making sitting and rising difficult for those with immobile hips. A social environment in which care-givers are overworked and harassed may lead the patient to repeatedly ignore the call to stool, in the hope of finding a quieter time later.

There are many adaptations that can be made to a toilet to facilitate access and stability in use [105]. Effective bowel evacuation is helped by sitting well-supported, with feet slightly raised to enable appropriate use of abdominal effort if needed, and leaning forward slightly [106]. Horizontal grab rails assist pushing up from a seated position, while vertical ones can enable pulling up. A raised seat or foot blocks can adjust the height as needed. For lateral transfer from a wheelchair, both seats need to be at the same height. Where it proves impossible for a person to use the toilet, alternative commodes or chemical toilets are available with appropriate features for the individual's needs. No studies were found examining the effect of modifying the physical or social environment in treating FI.

VI. PATIENT AND CARE-GIVER EDUCATION AND ATTITUDES

The strongest data on education and lifestyle comes from a single RCT randomising patients to nurse-led education and advice alone, or with the addition of exercises, biofeedback or home biofeedback. The education and advice group showed reduced frequency of FI and was as effective as biofeedback or exercises [107]. Other support for the benefits of patient education comes from a study reported in abstract form [108] which showed that education and standard medical care, when provided systematically to a group of FI patients who had failed prior attempts at medical management, led to a successful outcome in 38%. Success in this trial was defined as a patient's report that they had experienced adequate relief of bowel symptoms. Another randomised study examined the efficacy of a nurse-administered single session education and advice intervention in patients with bowel problems following stroke and found improved bowel function up to one year later [109, 110].

For people with dementia or other severe intellectual impairments, expert opinion holds that the attitude and management methods adopted by carers is as important as bowel function in maintaining continence [150]. No controlled studies on this subject were found. However, one quasi-experimental study examined care-givers' knowledge and compliance before and after an educational intervention [111]. Forty home care-givers of people with dementia, over half of whom had some degree of FI, completed a study-specific questionnaire before and after receiving a videotape and information booklet entitled "a practical approach to maintaining bowel control in people with dementia". Ninety percent of the care-givers accessed the information and there was an improvement in post-intervention knowledge scores measured on a 55-point scale, with the mean score increasing from 23 pre-test to 32 post-test ($p=<0.001$). However, it is not known if this improved knowledge translates into improved care or reduced FI.

VII. COMPLIMENTARY THERAPIES

Hypnotherapy has been found to improve urinary incontinence associated with detrusor overactivity [112]. However, no hypnosis treatment study was found which included FI as an outcome variable. Psychotherapy does not appear to enhance the effectiveness of behavioural intervention for FI in children [113], but no studies were found in adults. Likewise, there have been no studies of the use of acupuncture, reflexology, homeopathy or any other complimentary approach reported in the literature.

D. DIET AND FLUID INTAKE

I. RATIONALE FOR DIETARY INTERVENTIONS

The basis for investigating diet modification as a strategy for managing faecal incontinence comes from anecdotal reports by persons with faecal incontinence and clinicians, as well as from physiological principles of gastrointestinal (GI) function. People with faecal incontinence report anecdotally that they manipulate their diet and eating patterns as a strategy for managing their faecal incontinence [114]. Dietary manipulation is frequently employed by the approximately 20% of patients with irritable bowel syndrome (IBS) who also have faecal incontinence [95, 115] and by the approximately 19% to 40% of patients with inflammatory bowel disease who have faecal incontinence [15, 95, 116-119].

The way many patients with FI cope is to avoid eating anything on days they are going to be away from home: "nothing in, nothing out". They also avoid any food that they suspect may cause loose stools or gas. One survey of faecal incontinence and self-care practices for faecal incontinence administered to community-living elderly people showed that the respondents changed their diet and skipped meals. Changing diet was a significantly more common practice among women (35.4%) compared to men (12.5%) [120]. Bliss et al. [114] compared the nutritional profile of the usual diets of persons with faecal incontinence to the usual diets of age and gender matched controls with normal bowel function and found few significant differences. However, the group with faecal incontinence had a greater intake of carbohydrates, manganese and vitamin B1.

1. Fluid intake

Constipation is a recognized risk factor for FI (see section 2). Approximately 30% of elderly residents in long-term care institutions have faecal impaction [121, 122] and general clinical recommendations for faecal incontinence management in these cases are for an adequate intake of fluid to prevent hard stool consistency and constipation. However, there is no empirical data to support the recommendation of increased fluids either for constipation or for FI, and there is no evidence that the diets of patients with FI or constipation are deficient in fluids.

2. Dietary fibre, prebiotics, probiotics, and synbiotics

A prebiotic is a general term describing a food ingredient that is not digested in the human intestine and thus stimulates the growth and/or activity of one or more types of bacteria in the colon that have the potential to improve the health of the host. Because of its ability to stimulate growth of bacteria in the colon, dietary fibre can be considered a prebiotic. Fructo-oligosaccharides and galacto-oligosaccharides are popular prebiotics. A probiotic is a general term for a supplement (available as a tablet, powder, or food) containing live non-pathogenic and non-toxic microbes that have the potential to affect the balance of colonic microbes or improve the host's health. Bifidobacteria and lactobaccilli are the most commonly used probiotics, and yogurt which has active microbial cultures can be considered a probiotic. A synbiotic refers to a product that combines a prebiotic and probiotic. Probiotics have been investigated for their ability to prevent or reduce diarrhoea associated with antibiotics, ulcerative colitis, in acute infant dehydration due to diarrhoea and in treating *Heliobacter pylori* infections [123]. However, there are no published data on the use of probiotics or synbiotics to treat FI.

Dietary fibre is the non-starch, polysaccharide component of plant cell walls and lignin that resists digestion by human intestinal enzymes. Dietary fibre supplementation has been shown in an RCT to reduce FI associated with loose stool (see Section 4.5). Moreover, persons with normal bowel function who had diarrhoea inducing by administration of phenolphthalein reported that they had fewer days with urgency to defaecate or fear of FI when they ingested the soluble fibre psyllium compared to wheat bran, calcium polycarbophil, or placebo in an unblinded manner [124]. On the other hand, there are reports that dietary fibre may exacerbate FI in some patients. It has been observed that some patients with FI benefit from moderating their intake of foods containing largely insoluble fibre, such as whole grain breads and cereals, nuts, beans, fruits and vegetables with skin, and sweet corn [125]. One clinical letter reported that treating constipation in elderly immobile people with a supplement of insoluble fibre and bran, resulted in faecal incontinence in half of them [126].

3. Lactose, yogurt, sorbitol, fructose, caffeine, and alcohol

Certain dietary components such as lactose, sorbitol,

fructose, caffeine, and alcohol may cause loose stools that can potentially aggravate FI. A deficiency of the small intestinal enzyme, lactase, prevents hydrolysis of the disaccharide lactose and its absorption. The presence of lactose creates an osmotic shift of intestinal water into the small intestine and increased transit. In the large intestine, fermentation of lactose by colonic bacteria typically results in flatulence, distension, diarrhoea, and cramps. However, the majority of adults who have lactase deficiency can tolerate a small amount of lactose in foods [127]. Yogurt is usually well tolerated by lactose maldigesting individuals because the lactose is partially digested by the beta-galactosidase of the bacteria used to ferment the yogurt. However, yogurt has not been found to aid the digestion or tolerance for additional lactose simultaneously consumed with it [128].

Due to its prevalence in approximately 25% of the population, lactose maldigestion is currently regarded as a normal physiological pattern rather than a disease [129]. It occurs in 6% to 19% of whites, 53% of Mexican Americans, 62% to 100% of Native Americans, 80% of African Americans, and 90% of Asian Americans [129, 130].

Malabsorption of fructose and sorbitol results in osmotic diarrhoea and adverse symptoms, similar to lactose. A diet reduced in fructose and sorbitol content is suggested for some patients with irritable bowel syndrome to reduce adverse GI symptoms [131].

Caffeine, of which coffee is a popular source, has numerous effects on the GI system, it reduces lower esophageal sphincter pressure, stimulates acid and gastrin secretion as well as secretion from the small intestine, and induces a desire to defaecate [132-136]. Caffeine has also been observed to stimulate defaecation urgency in some patients with FI [125].

Chronic consumption of alcohol has been associated with accelerated gastric emptying and small bowel transit in animal studies whereas a single large dose has an inhibitory effect on these parameters [137-139]. Excessive alcohol consumption leads to injury of the duodenal and upper jejunal mucosa and inhibition of sodium and water absorption. There is an increased prevalence of bacterial overgrowth in the small intestine of alcoholics, which may contribute to loose stools, diarrhoea, incontinence, and other GI symptoms.

II. LITERATURE SEARCH STRATEGY

The following databases were searched for studies to include in this review of dietary interventions for faecal incontinence management: Medline (1966 to 2004), CINAHL (1982 to 2004), BIOSIS (1996 to 2004), PUBMED (1950 to 2004), Cochrane Library of Controlled trials (4th quarter 2004) and the Cochrane Library of Systematic Reviews (3rd quarter 2003). The following key words were linked with faecal incontinence and used in the database searches: food, diet, dietary therapy, dietary fibre, fibre, prebiotic, probiotic, synbiotic, caffeine, lactose, yogurt, fructose, sorbitol, fluid, fluid intake, and fluid therapy. The Table of Contents of major journals on gastroenterology, digestive diseases, colon and rectal surgery, nutrition, and wound, ostomy and continence nursing were searched for relevant articles from November 2003 to January 2004. Reference lists were scanned from the systematic reviews of the Cochrane Collaboration, practice-related articles, or review papers about treatment of faecal incontinence from 2000 to 2003, and relevant papers were retrieved.

III. CRITERIA FOR CONSIDERING STUDIES FOR THIS REVIEW

1. Types of studies

Only studies in the English language were reviewed. Systematic reviews and meta-analyses of randomised controlled trials and full-length manuscripts reporting individual studies published in a peer-reviewed journal were considered. Individual studies or reports were required to have one of the following designs as defined in the ICUD review guidelines: randomised, controlled trial, prospective, non-randomised cohort, case-control, or recommendations from an expert consensus panel or Delphi process.

2. Types of study participants

Studies that involved people who were 18 years or older, had faecal incontinence, and received a dietary intervention were included. People who were tube-fed or had an intestinal ostomy of any sort were ineligible.

3. Types of dietary interventions

A dietary intervention was defined as any type of food, supplement, dietary product, or fluid that is purposefully consumed or restricted, limited or avoided to manage faecal incontinence. Studies were excluded if it was not possible to distinguish any direct effect of the dietary intervention from other interventions introduced simultaneously. For example, a study was excluded if it combined pelvic floor muscle training and a dietary intervention and compared it to another intervention such as drug therapy making it impossible to determine the effect of the dietary intervention alone.

4. Types of outcome measures

Faecal incontinence was required to be a primary outcome measure of the studies. Studies which focused primarily on the outcomes of stool consistency or form, stool amount, volume or bulk, defaecation frequency, diarrhoea, or constipation without including any measure of faecal incontinence were excluded.

IV. METHOD OF REVIEW

The reviewer examined the list of citations and abstracts yielded from the electronic search strategy. Potentially relevant papers were retrieved in full text. The reviewer was not blind to the journal titles, authors' names or their institutional affiliations. The quality of the studies was evaluated using the checklist accompanying the CONSORT statement available at http://www.consort-statement.org. The levels of evidence for therapeutic interventions developed by the 3rd ICI 2004 were adopted.

V. FINDINGS

1. General description and setting

One study was found that met the inclusion criteria for this review [140]. Subjects were community-living people with incontinence of loose or liquid stools in the United States. The intervention was supplementation with one of two soluble dietary fibres compared to placebo.

2. Methodological qualities of studies

a) Study Design.

The study design was a randomised, parallel-group, placebo-controlled, single blind trial. A block scheme resulted in equal numbers in each group. Details of the procedures for random assignment and allocation concealment were not provided. The participants, statistician, and participants' clinicians were blinded [141]. The interval for data collection during the baseline and intervention periods was equal.

b) Participants and Follow-Up.

Participants were adults who had faecal incontinence of loose or liquid stool at least weekly. The cause of their faecal incontinence, if known, was not reported. The inclusion and exclusion criteria were reported. Thirty-nine (8 men, 31 women) of 42 people completed the study. Drop-outs occurred during the baseline period before any fibre supplementation, and reasons for attrition were reported. Follow-up was completed by 95%. There were 13 participants in each group. How sample size was determined and the power of the study was not explained. The characteristics of the groups were comparable in the baseline period.

c) Dietary Intervention.

Participants consumed one of the following soluble fibre supplements mixed into fruit juices: 25 g of psyllium source/day, 25 g of a gum arabic source/day, or placebo (0.25 g of a pectin source/day). Based on the percent of fibre in each of the sources, the amount of dietary fibre that was administered in the supplements could be calculated as follows: 7.1 g of psyllium/day, 21.5 g of gum arabic/day, and 0.2 g of the pectin placebo. The timing of supplement consumption and periods of administering the supplements and follow-up were defined. The supplements were taken in addition to usual diet intake, which was determined by a prospective diet record for 8 days in each period. Adherence to taking the supplements was determined by self-report of the participants. There were attempts to control concomitant treatments, e.g., none of the participants was taking biofeedback training for pelvic muscle exercises, those who were taking anti-diarrhoeal medications were instructed not to alter the type or amount of medication taken, anti-diarrhoeal compliance was determined by self-report of the participants. Measures of stool characteristics were made using standard, objective laboratory procedures.

d) Outcome measurement.

Participants prospectively reported faecal incontinence on a daily stool diary for 8 days in each period. The proportion of incontinent stools during the baseline and fibre supplementation periods was the primary measure of faecal incontinence.

Secondary clinical measures included stool consis-

tency and frequency (both obtained from the stool diary), stool wet and dry weights, and percentage water content of stool (determined by freeze-drying). Measures of the effects of the fibres on the stools included the water-holding capacity of stool solids, total fibre content of stool, stool pH, and faecal short chain fatty acids.

e) Adverse events.

The adverse event of flatulence was monitored daily by self-report of the participants on the stool diary. Flatus did not differ between the baseline and fibre supplementation periods or among fibre groups.

f) Results

The rate of faecal incontinence for the groups ingesting psyllium or gum arabic were significantly lower that those taking the placebo. The rate of stools with loose and unformed or liquid consistency for the groups ingesting psyllium or gum arabic were significantly lower that those taking the placebo. The water-holding capacity of stool solids was highest for the group ingesting psyllium. There were no differences among the groups in stool frequency, wet or dry weight of stool, weight of stool solids, total fibre content of stool, stool pH, or short chain fatty acids. A limitation of the study was the relatively small sample size (n=13 per group).

VI. CONCLUSIONS

The one published study provided level 1 evidence to suggest that dietary fibre can reduce the rate of FI in patients with loose stool. Dietary fibre supplementation appears to be a safe and tolerable intervention (see below). Further studies on the topic are encouraged to build a greater body of evidence.

VII. RECOMMENDATIONS FOR FUTURE STUDIES ON DIET

Additional studies are recommended that have larger sample sizes and follow participants for a longer period of supplementation. Both dietary fibres administered in the reviewed study appeared to be fermented to a similar extent. Since the fermentability of dietary fibre may be affected by the amount of fibre administered, the role of fermentation on the effectiveness of dietary fibre in managing faecal incontinence warrants investigation.

Future studies could then determine whether there is an optimal type of fibre to use for faecal incontinence and whether there is a dose of fibre beyond which no additional benefit will be seen. Whether dietary fibre can augment other behavioural interventions such as pelvic floor muscle exercises (especially during the period of muscle conditioning) or bowel training needs to be studied further. Studies are needed to elucidate possible mechanisms underlying the benefit of dietary fibre.

The methods of future studies would benefit from a more objective measure of FI. Measuring compliance with a dietary intervention, although very difficult in an incontinent population, would strengthen the design of future studies. Investigators should be attuned to a broad range of symptoms of tolerance to dietary fibre over time.

Recommendation: soluble dietary fibre is recommended for the management of FI associated with loose stool. (Evidence level 1. Recommendation Grade B).

VIII. OTHER DIETARY INTERVENTIONS

No studies of any other dietary intervention for managing faecal incontinence were found, so no recommendations about other dietary interventions for managing FI can be made.

E. BOWEL MANAGEMENT AND RETRAINING PROGRAMMES

Note: Committee 13 has reviewed the evidence for bowel retraining programmes in nursing homes and frail elders. This section is limited to studies in younger adults.

The following databases were searched using the keywords "faecal incontinence" and the relevant intervention: Cochrane library, AHMED 1985-2004, BNID 1994-2004, EMBASE 1974-2004, MEDLINE 1951-2004, PsychInfo 1987-2004. All seemingly relevant articles were retrieved in hard copy, and the reference lists searched for further studies.

I. BOWEL HABIT

Expert opinion supports the importance of attempting to establish a regular predictable pattern of bowel evacuation by patient teaching and adherence to a routine [94, 142]. Because peristaltic contractions of the colon that are associated with defaecation increase in frequency following awakening from sleep and following meals [143, 144] the period after breakfast is the best time for scheduled defaecation, but no studies have evaluated the effectiveness of this in adults. In children with constipation-related FI (encopresis) bowel habit training is effective at reducing the frequency of FI and increasing the frequency of self-initiated bowel movements (see Committee 11). Toileting programmes for assisting dependent older people to access the toilet are covered in Committee 13 and protocols for neurological bowel management are in Committee 12.

II. RESISTING URGENCY

In contrast to urinary incontinence, where a body of knowledge has developed on the efficacy of bladder training techniques, particularly in relation to the overactive bladder syndrome (Committee 14), the possibility of bowel retraining for resisting urgency to defaecate is almost unexplored. Some biofeedback protocols focus on altering rectal sensory thresholds (see below) and some protocols for treating soiling in children focus on establishing a regular bowel routine.

One RCT compared patients who received education, including urge resistance techniques [125], and dietary advice, to a group of patients who received the same training plus anal sphincter exercises with or without home or clinic biofeedback. There were no significant differences in outcomes [107]. However, this study did not assess the effectiveness of the behavioural training compared to an appropriate control group.

III. EVACUATION TRAINING

A common factor in the genesis of pelvic floor problems may be chronic straining with perineal descent from constipation, this may lead to pelvic floor damage (direct or neurological) [145, 146] and may be associated with pelvic organ prolapse or urinary or faecal incontinence. In one small study women who reported straining were more likely to develop urogynaecologic symptoms such as prolapse and stress urinary incontinence [147]. No studies were identified examining the effect of treating constipation or decreasing straining on preventing or treating FI in non-institutionalised adults.

Clinically, many patients with FI are taught evacuation techniques or are encouraged to use laxatives, enemas or suppositories in an attempt to ensure that the rectum remains empty most of the time, thus giving less chance of FI. There is a well-recognised association between rectal loading and FI in both frail elders [148] (Chapter 18) and in children with soiling (Chapter 16).

Children with FI have been found to respond to training to relax the external anal sphincter in order to evacuate effectively, although improvement in sphincter co-ordination does not necessarily equate to symptom change [149]. Laxative regimens reduce FI in institutionalised adults if complete rectal evacuation can be achieved [121]. However, complete evacuation is not easy to achieve (see section F below). Clinically many older women with FI report former constipation as a young adult, but data to support this are lacking. The evidence for treatment options is lacking.

One RCT of a combination treatment package for FI included training on evacuation techniques and noted that patients reported improved ease of evacuation after treatment [107]. No separate data on FI were presented. No studies were found utilising specific evacuation training to treat FI in younger adults.

IV. BEHAVIOUR MODIFICATION

Toilet training with rewards, either alone or in combination with laxatives has been found helpful in children with encopresis [149] (see Committee 11). It is not known if a similar approach might be applicable to adults with learning difficulties or frail older people in institutional settings, although a behavioural approach to such problems is recommended by expert opinion [150]. Adults with learning difficulties may respond to formal behaviour modification techniques, but only small case series are currently available as evidence [151] Similarly there are no controlled studies of training in non-retentive encopresis [152].

V. COMBINATION THERAPIES

It is recognised that in many people the symptom of faecal incontinence is the result of a complex combination of disordered anatomy and physiology, stool consistency and gut motility, emotional and psychological status and restricted access to toilet facilities, amongst other factors (see Committee 5). Hence in clinical practice most patients receive a combined approach addressing diet, medications, lifestyle, muscle function and bowel habit simultaneously, depending on the result of initial assessment [153, 154]. However, with the exception of one study [107] the few well-conducted studies on the conservative management of FI in adults have usually focused on evaluating a single intervention such as biofeedback, often not specifying what other advice (that might confound the results) was given to patients.

Norton et al compared a combination of conservative measures, including patient teaching, advice on diet, medication titration, and bowel retraining, with the same measures combined with anal sphincter exercises and/or biofeedback [107] No statistically significant differences were detected between the four groups on any of the outcome measures (including diary, symptom questionnaire, manometry, anxiety, depression and quality of life). Over 50% of those randomised (171 patients) reported improved continence. Of those completing the protocol, 74% felt that they remained improved at one year following the end of treatment. The authors of this study suggest that the most effective element may have been education and therapist-patient interaction rather than specific interventions.

An RCT of a combination nurse-led intervention for bowel problems in 146 stroke patients found that a single therapeutic visit with a detailed information booklet improved bowel dysfunction up to 6 months later, and changed diet and fluid behaviour up to one year later compared to controls who received routine care. The intervention group were more likely to have sought professional help from their family practitioner for bowel problems demonstrating a heightened awareness of the possibility of treatment [109, 110]. However, there was no difference in the rates of FI between the intervention and control groups.

In children a combination of behavioural training techniques and laxative therapy is as effective alone as it is when combined with biofeedback [149].

Anecdotally, laxatives may enhance the effect of behaviour modification alone.

VI. RECTAL IRRIGATION

1. Antegrade irrigation

Surgical construction of an irrigation port to allow for the use of antegrade enemas or colonic irrigation is covered in Committee 19. An alternative is the insertion of an artificial irrigation port into the left colon (e.g. percutaneous endoscopic colostomy or PEC [155, 156] or the right colon [157]. Infection at the insertion site may be a problem in the medium to long term [158].

2. Retrograde irrigation

Anal irrigation to control FI has been reported in a few isolated case series [159, 160] and is reported anecdotally to be widely used in Europe to manage FI symptoms, although there is no published evidence to support this. Irrigation may be by gravity-fill using a stoma irrigation cone [160, 161] or via a rectal tube, or using a stoma irrigation pump with either of these [162]. Some patients find the procedure painful or ineffective, and other discontinue because irrigation is time-consuming (taking between 10 and 90 minutes), but up to two-thirds of patients unresponsive to other non-surgical management methods have been reported to benefit, and those with soiling are more likely to continue than those with major incontinence [160].

Shandling [163] has developed a catheter incorporating an inflatable balloon and reported 100% success with 40% of 112 children with spina bifida, however, he did not make selection criteria clear or state

the length of follow-up [163]. Others are more cautious in their appraisal of retrograde irrigation. Thirty one children with spina bifida aged 3-19 years (mean 9 years) who were dissatisfied with current bowel management were treated by infusing 20ml/kg of saline every 24 or 48 hours through this catheter [164]. There were 6 immediate dropouts and a further 9 had stopped using the catheter by 30 months. Of those who continued to use the catheter the percentage of continent stools rose from 28 to 94% and the percentage of constipated stools dropped from 55 to 15%. The authors suggest that potential adverse effects include bowel perforation, and allergic reaction to latex in the catheter. One group [165] reported that 83 of their 190 patients with spina bifida achieved continence using irrigation daily or on alternate days via a cone in the anus. They used water at 20ml/kg body weight, with evacuation within 30 minutes. The investigators stress the importance of a specialist nurse overseeing the programme. The irrigation did not help with bladder compliance or instability.

Details of the technique are given by Scholler-Gyure and colleagues [165], who use 20ml/kg tap water one hour after the evening meal, run in half the volume and then allow 5 minutes without the cone, then run in the rest of the water, allowing up to 20 minutes for defaecation afterwards. They state that the volume needed varies and is often only found by trial and error. Of 41 patients with spina bifida who had failed other bowel management, 66% were continent at a mean follow up of 33 months, seven had monthly incontinence and 7 weekly incontinence, none had daily incontinence. Side effects included abdominal pain, headaches and poor appetite, but these were rare. Parental satisfaction was high in 63% and good in 37%. 66% of the children rated continence as the most important advantage, but half felt that it took up too much time and energy. Six found irrigation painful and three found it unpleasant, five were dependent on others to help.

There has been virtually no research on the best irrigation regimen, which fluid to use, and in what volumes to promote optimum evacuation either via an artificial port or by rectum. Christensen has evaluated retrograde colonic washout in a laboratory setting, using scintigraphy to assess segmental evacuation [166]. Six patients with FI were found to empty most of the rectosigmoid and descending colon, with fluid reaching on average just beyond the hepatic flexure. However, the clinical significance of this finding has yet to be explored. Gattuso et al [167] found that colostomy irrigation with water at volumes of 500mls and above (but not at 250mls), produced high-pressure propagated waves of colonic contraction and effective evacuation without subsequent breakthrough. Patients preferred 500mls to larger volumes. This work had not been done with irrigation anally.

Some patients find it impossible to retain fluid instilled rectally. Clinical experience suggests that the response to rectal washout is very individual, and it is worth experimenting with volumes, temperatures and fluids, with or without addition of enemas, to find the optimum for an individual. There is a danger of autonomic dyssreflexia in those with a thoracic level spinal lesion, and this will need careful monitoring and patient teaching.

A mechanical pump which introduces warm water in pulses into the rectum has been described [162]. It was reported to clear faecal impaction in 24 of 37 procedures on the first attempt and was well tolerated by most patients. The pump has been shown to be safe in a case series over 6 years [168]. It is not known if continence improved because the main focus of the study was relief of constipation.

3. Adverse events

A note of caution has been given about using tap water if a water softening system is in place because electrolyte disturbance may occur in vulnerable individuals such as children [169]. Phosphate enemas or tap water irrigation may also cause volume overload or electrolyte imbalance in patients who have compromised renal function or who retain the fluid for excessively long periods of time [170, 171]. Long-term use of phosphate enemas in irrigation fluid may also cause electrolyte imbalance in vulnerable patients. There is a case report of hyperphosphataemia and symptomatic hypocalcaemia with irrigation via an ACE [172]. Similar problems are theoretically possible with rectal irrigation. Isolated reports of perforation and serious complications are rare, but are a possibility, particularly if extension tubes are used [173].

VII. DIGITAL OR OTHER STIMULATION

Use of a finger or other stimulator to trigger reflex rectal contraction and anal relaxation is a technique commonly employed to control timing of evacuation after spinal cord injury (see Committee12). Expert opinion suggests that this may work in the non-neurogenic population [142] but no study was found.

VIII. MANUAL EVACUATION

A controversy has arisen in nursing about using manual rectal evacuation because of possible complications [174]. There is almost no research on this, and no evidence of harmful effects. The majority of patients with lower spinal cord injury and cauda equina syndrome need to use this regularly, although the evidence base is lacking [175, 176]. The Royal College of Nursing in England [174] has reviewed manual evacuation and suggested a procedure and safety points. No study was found evaluating the efficacy of manual evacuation for managing FI.

F. DRUG TREATMENT OF FI

I. GOALS

The goals of this section are to identify the drugs and other medical interventions that have been used to treat faecal incontinence and to evaluate the evidence regarding their efficacy. The medical management of faecal incontinence has focused exclusively on three mechanisms:

1 Reduction of diarrhoea. Diarrhoea is consistently found to be a strong risk factor for FI (see Section B above).

2 Increasing resting anal canal pressure. Low resting anal canal pressures are a risk factor for passive FI, and are commonly seen following some types of anorectal surgery (e.g. sphincterotomy, ileal pouch procedures, abdominoperineal pull-through for imperforate anus).

3 Control of constipation. Constipation is frequently found to be a risk factor for FI, especially in children and the elderly (see Section B above).

II. METHODS

The Medline database and the Cochrane reviews [149, 177) were searched for studies in any language and any year which matched the following search terms:

1 "Faecal incontinence" OR "anal incontinence" AND "drug" OR "medical management" OR "medical treatment."

2 "Faecal incontinence" OR "anal incontinence" AND "loperamide" OR "diphenoxylate."

3 "Faecal incontinence" OR "anal incontinence" AND "laxative" OR "polyethylene."

4 "Faecal incontinence" OR "anal incontinence" AND "phenylephrine gel."

These search terms captured a total of 347 unique articles. These articles were screened by title, then by abstract, and 148 articles identified as potentially relevant were retrieved and read. Additional articles were identified by examining systematic reviews.

III. TREATMENT OF DIARRHOEA-ASSOCIATED FAECAL INCONTINENCE WITH ANTIDIARRHOEAL DRUGS

1. Loperamide and diphenoxylate

Table 6 lists all studies found that were judged to be relevant to the topic of drug treatment of diarrhoea-related FI. All the studies that assessed loperamide treatment were rated as level 2 evidence: they were randomised cross-over studies but had methodological flaws which limited generalisability. Some of these studies reported that loperamide was superior to placebo [178-180] while others reported only a trend favouring loperamide [181, 182]. The study by Palmer and colleagues [182] is significant because it directly compared loperamide (average of 4.6 mg per day) to codeine (average of 103 mg per day) and diphenoxylate (average of 12.5 mg per day) in 30 patients with diarrhoea, of whom 19 had FI prior to treatment. However, FI was not the primary outcome measure. Loperamide was superior to diphenoxylate and similar to codeine with respect to decreased stool frequency, improved stool consistency, and reduced side-effects. Although not statistically significant, there was a trend for less FI while taking loperamide compared to diphenoxylate.

2. Sucralfate

This drug was compared to placebo in a double-blind, parallel group, RCT [183] carried out in 123 patients with diarrhoea that occurred secondary to radiation treatment for pelvic cancer. FI was not required for inclusion and was a secondary endpoint in data analysis. This study showed, contrary to hypothesis, that sucralfate made FI worse.

3. Valporate sodium

Gamma-amino butyric acid (valporate sodium) was

Table 6 . Trials of Drug Treatment for Diarrhoea-Associated Faecal Incontinence

Source	Drug tested	Patients	Design	Results	Comments
Harford et al 1980	Diphenoxylate & placebo vs. placebo	15 w/ FI & diarrhoea	Blinded crossover; 3 day treatments, 1 day washout	Decreased BM volume & frequency; tendency to decreased FI	Level 2 evidence. Treatment too short; Too few subjects
Palmer et al 1980	Loperamide 4.6 mg vs. codine 103 mg vs. diphenoxy-late 12.5 mg	30 w/ diarrhoea, of whom 19 had FI	Blinded crossover; 4 week treatments, no washout	Loperamide similar to codine & superior to diphenoxylate for BM frequency, urgency, & side-effects; trend for loperamide to reduce FI more than comparators	Level 2 evidence. Underpowered. Did not analyze by ITT.
Kekomaki et al 1980	Loperamide 2-6 mg vs. placebo	7 children w/ con-genital anomalies	Blinded crossover; 3 week treatments at 3 different doses altern-ated with placebo	Improved BM frequency, consistency, & FI in dose-dependent way	Level 2 evidence. Too few subjects; mixed aetiologies.
Read et al 1982	Loperamide 12 mg vs. placebo	26 w/ diarrhoea, FI, & urgency	Blinded crossover; 7 day treatments, no washout	Decreased FI, urgency, stool weight, % loose stools, BM frequency; increased resting anal pressures	Level 2 evidence. Treatment period too short
Arnbjornsson et al 1986	Loperamide, titrate up	8 children w/ FI 2nd high imperforate anus repair	Open, uncontrolled 6-8 week study	Decreased FI in 7/8; Intolerable constipation in 2.	Level 3 evidence. Uncontrolled study

compared to placebo in a double-blind, randomised crossover study [184] in 17 patients with diarrhoea related FI secondary to colectomy and ileoanal anastamosis. Valproate sodium decrease FI episodes and stool frequency relative to baseline and increased anal canal pressure, whereas placebo did not have these effects. Limitations of the study were (a) small sample size and (b) statistical comparison to baseline rather than to placebo. Valporate sodium is of possible benefit in this population.

4. AMITRIPTYLINE

Santoro and colleagues [185] carried out an uncontrolled study of the tricyclic antidepressant, amitriptyline, given 20 mg at bedtime, in 18 patients with FI, diarrhoea was not required. Thirteen of 18 became continent and 3 reported improvement. The authors attributed the benefits to increased anal resting pressure and decreased numbers of "rectal motor complexes." This study suggests that amitriptyline and other tricyclic antidepressants are of possible benefit for treating FI.

• Mechanism of action

Three possible mechanisms of action have been identified in the these studies of the drug treatment of diarrhoea-related FI: Loperamide, diphenoxylate, and amitriptyline appear to work in part by decreasing bowel movement frequency through an effect on motility and absorption. Fibre supplements (reviewed in Section 4 above), on the other hand, work by binding more water into the stools. Resting anal canal pressures were reported to be increased in response to loperamide [178, 179], valporate sodium [184], amitriptyline [185], and phenylephrine gel [186, 187].

VI. INCREASING ANAL CANAL PRESSURE IN PATIENTS WITH PASSIVE FI

A subgroup of patients with FI have passive incontinence, defined as FI that is not preceded by a sensation of urgency to defaecate and that occurs without awareness. This is believed to be related to decreased resting pressure in the anal canal due to an impaired internal anal sphincter and/or to decreased sensation for rectal distension. A specific aetiology for passive FI is the patient with a colectomy (usually for ulcerative colitis) with a surgically constructed ileal reservoir connected to the anal canal [36].

Phenylephrine gel, an alpha-1 adrenergic agonist, has been investigated for the treatment of passive FI in patients with ileoanal pouches [187]. This was a double-blind crossover study comparing topical (intranal) administration of 10% phenylephrine gel to placebo in 12 patients. However, the investigators noted carryover effects and therefore reported only the first treatment period, making this a parallel group study. They found a significant reduction in a faecal incontinence severity score during treatment with the active drug. The same research team reported a second study [188] in which they randomised 36 patients with idiopathic causes of FI (not including ileal pouch procedures) to receive either phenylephrine gel or placebo, and found no significant improvement in either severity of FI (incontinence score) or anal canal pressures. These patients all had structurally intact sphincters. Thus, the clinical utility of phenylephrine gel (if any) may be limited to patients with passive incontinence associated with ileal pouches. Oral preparations have been found to have significant side-effects but this has not been reported in these topical studies.

Loperamide also increases anal canal resting pressure by an unknown mechanism [178, 179], and it has been suggested that this may contribute to the treatment of FI [180]. As reported in the last section, loperamide does decrease the frequency of diarrhoea-related FI [178-180], but it is unknown how much of this effect is due to increased resting pressure in the anal canal as compared to changes in stool consistency. No studies of the effectiveness of loperamide in patients with passive FI were found.

V. DRUG TREATMENT OF CONSTIPATION-ASSOCIATED FI

Constipation-associated FI, sometimes referred to as overflow incontinence, occurs more frequently at the two ends of the lifespan. The prevalence of FI in children is estimated to be 0.8% [189] to 3% [190] and in 35% (191) to 96% [25] of cases, FI in children is associated with constipation. FI occurs in 46%[192] to 47% [21] of nursing home residents and is more common in those with constipation [193]. However, the proportion of faecally incontinent nursing home residents whose FI is attributable to constipation is not known. Constipation-associated FI is also common in patients with spinal cord injury, occurring in an estimated 33% [58].

Constipation-associated FI in nursing homes is often treated with the prescription of daily or frequent laxatives. However, we found only two RCTs which tested the effectiveness of laxatives for treating FI

associated with constipation in adults. Ryan [194] randomised 87 new admissions to a single nursing home to receive either 15 ml daily of sorbitol for up to 15 days or routine care without the use of a laxative. Patients were enrolled whether or not they had constipation or FI. The outcome measures recorded by nurses were amount of nursing time required for the care of FI and amount of soiled linen. Patients treated with sorbitol were found to have significantly less soiled linen, and they tended to require less nursing time. Limitations of the study included (a) analysing the aggregate amount of soiled linen used by each group rather than the proportion of the subjects in each group who had FI, (b) failure to control for expectancy by providing a placebo treatment to members of the control group, and (c) failure to include all randomised subjects in the data analysis, i.e. failure to use an intention to treat analysis.

A second study [121] compared daily enemas to no treatment in 206 nursing home residents who had FI and documented constipation. This was an open label RCT. Results showed no difference between groups either for frequency of FI or for amount of soiled linen. However, post hoc subgroup analysis showed that patients with complete rectal emptying on digital examination exhibited a significantly greater improvement than the group that continued to have a faecal impaction. Strengths of this study were the large sample size, randomisation, strict inclusion criteria, and assessment of whether the enema regimen in fact eliminated faecal impaction. A weakness was that the post hoc analysis of the physical examination data suggest that the trial is not interpretable since the daily enema regimen did not eliminate faecal impaction in most patients.

Two RCTs were reported for children with FI associated with constipation. In one of these, Nolan and colleagues [195] randomised 169 children who had FI and radiological evidence of stool retention to receive either combined treatment with laxatives plus behavioural toilet skills training or behavioural toilet skills training alone. This was an unblinded RCT. Children treated with laxatives plus behaviour modification were significantly more likely to improve than children treated with behaviour modification alone (63% vs. 43%, $p<0.02$), and improvement was more rapid. Limitations of the study include (a) failure to blind the study by using a placebo, and (b) analysing the data by symptom completers rather than by intent to treat.

A second RCT in children [196] randomised 40 children with constipation (only 3/4 had FI at enrolment) to one of 3 groups: senokot, placebo tablets, or no treatment. All children also received toilet training. The authors reported that all three groups improved (i.e. reduced frequency of FI) and there were no differences between groups. However, the study did not have sufficient power to test the hypothesis.

A double-blind RCT testing the effectiveness of the prokinetic drug cisapride in paediatric FI found no evidence for efficacy [197]. Adverse events subsequently led the U.S. Food and Drug Administration to restrict access to this drug. An alternative prokinetic drug, tegaserod, has been approved for the treatment of chronic constipation in adults, but has not been tested for its effectiveness in patients with constipation-associated FI.

Several trials [198, 199], including one high quality RCT [200] have compared laxatives alone to the combination of laxatives plus biofeedback in children with constipation-associated FI. For this indication biofeedback is designed to teach the patient to relax the pelvic floor muscles during attempts to defaecate in order to overcome a tendency to paradoxically contract these muscles and to obstruct defaecation. The RCT by van der Plas and colleagues [200] showed that combined treatment was associated with a higher success rate at the end of training (39% vs. 19%), but by follow-up 12 months later, there were no differences between groups. Other studies support these findings by showing either no difference between the laxative only group and a biofeedback group [198] or faster acquisition of continence in the biofeedback group but no long-term difference in success rate [199]. These trials suggest that laxatives alone are as effective as biofeedback for constipation-associated FI in children in the long term, but they were not designed to show that laxatives are superior to placebo or to no treatment.

VI. DRUG TREATMENT OF FI: SUMMARY AND RECOMMENDATIONS

Level 2 evidence supports the recommendation of loperamide for diarrhoea-associated FI and suggests that the loperamide may be superior to diphenoxylate. Our recommendation is to treat diarrhoea-associated FI with antidiarrhoeal drugs (Grade B). There is insufficient trial data to recommend phenylephrine gel or loperamide for the treatment of passive FI. For constipation-associated FI, there is level 2 evidence suggesting that daily or more frequent oral laxative regimens may

be effective for the treatment of constipation-associated FI in nursing home residents [194] and children [195], but there are conflicting data [121, 196]. We recommend the use of laxatives to treat constipation-associated faecal incontinence, Grade B. It is important to confirm that the impaction is resolved by the laxative regimen. Additional, well-designed studies are needed to validate the common clinical practice of using laxatives to treat constipation-associated FI.

G. BIOFEEDBACK AND/OR ANAL SPHINCTER / PELVIC FLOOR EXERCISES

I. INTRODUCTION

This section reviews all studies published in English of adult research on biofeedback for faecal incontinence. A MEDLINE search was conducted to identify all articles matching "biofeedback and faecal incontinence" published between 1970 and February 2004. The search was supplemented by a crosscheck of citations in the identified papers and other systematic reviews.

II. BIOFEEDBACK MODALITIES USED IN THE TREATMENT OF FAECAL INCONTINENCE

Biofeedback can be defined as the use of an instrument that delivers a concurrent measurement of selected biological responses to enable the individual to alter his/her physiological response in directions associated with improved function [201].

The earliest reported application of biofeedback to treat faecal incontinence used a simple pressure device in the anal canal to reinforce external anal sphincter (EAS) contraction [202], a procedure somewhat analogous to the vaginal perineometer that was used by Kegel to treat stress urinary incontinence [203]. However, the seminal biofeedback procedure [204] for FI, which was followed in a series of studies [57, 205-211], used a 3-balloon manometry probe to reinforce changes in 3 distinct physiological variables rather than just EAS contraction. The responses that were reinforced with this protocol included, (a) the perception of sensory cues associated with rectal distension and potential loss of stool, (b) a short-latency EAS contraction, and (c) inhibition of activity that would increase rectal pressure (i.e. contraction of the abdominus rectus and diaphragm). The overall goal of this protocol was to strengthen the presumed EAS reflex that normally counters the internal anal sphincter inhibitory response to rectal distension. However, reinforcement for EAS contraction was contingent upon maintaining stable rectal pressure, because increases in rectal pressure during stool urgency can overcome relative sphincter closure pressure, and thus would be counterproductive to retention. Subsequently, the EAS response to rectal distension was determined to be a learned, rather than an involuntary response. As a result, the theoretical basis for the use of operant conditioning (biofeedback) in the treatment of bowel disorders was established [212].

III. BIOFEEDBACK PROTOCOLS

There is wide variability (i.e. no standardisation) in the biofeedback literature for FI. Studies use different instrumentation, training procedures, adjunctive strategies, samples, outcome measures, or follow-up periods. Therefore, straightforward comparison of study outcomes and statistical analysis of multiple outcomes is not possible. Most biofeedback protocols can be placed into one of three general categories on the basis of the procedures used for training and include:

1 strength training, defined as the reinforcement of sphincter or pelvic floor muscle (PFM) contraction to improve EAS strength without attention to sensation [213-219],

2 sensory training, defined as the reinforcement of heightened sensitivity to stepwise reductions in rectal distension volumes without emphasis on improvements in sphincter strength [220, 221], and

3 combined training, defined as the reinforcement of rectal sensitivity, a short-latency EAS response in the absence of rectal pressure changes and also sustained EAS contraction to improve sphincter strength [222, 223]. Variations of these procedures include the reinforcement of tolerance to progressively larger volumes of rectal distension and control of urgency [224, 225]. Instrumentation

used to measure and reinforce the changes in biological activity include pneumatic and perfusion manometry, surface electromyography (EMG) and recently transanal ultrasound [226, 227]. Some workers have suggested the use of a multivariable EMG protocol that mirrors the manometric protocol by substituting surface abdominal EMG electrodes for the rectal pressure balloon to measure extraneous abdominal muscle wall contraction that is associated with increases in rectal pressure. An EMG probe is placed within the anal canal or vagina to measure external anal sphincter or pelvic floor muscle activity [228]. There is limited support for the use of abdominal EMG measures as valid and reliable indices of muscle activity associated with increased intra-abdominal pressure and for the use of pelvic floor EMG measures as indices of pelvic floor and EAS contractile function [229, 230].

IV. STUDY SAMPLES

A systematic review found 46 studies in adults published in English from 1974-2000 [231] that included a total of 1364 patients. Another, review assessed 35 studies published from 1973-1999 which met inclusion criteria as being prospective, using 5 or more subjects and included an a description of biofeedback procedures used [232]. Subsequently, 16 additional papers were published from 2000-2004 [90, 107, 226, 233-245].

The majority of patients in all published studies are female. Ages studied range from 6-97 (studies primarily of children were excluded). Aetiology and symptom severity was highly heterogeneous. However, some studies did select subjects according to specific problems such as obstetric injury [90, 245-247], severe pudendal nerve deficit (215), transit time [237], older people [223] or severe FI [235]. Other studies restricted their samples to patients who had undergone ileo-anal reservoir procedures (87), rectosigmoidectomy and creation of a J- pouch [248], and anterior resection of the rectum combined with total colectomy [249]. Subjects in two studies had stomas [224, 241] with treatment provided in preparation for stoma closure, but data was not provided regarding changes in actual faecal incontinence from pre- to post-treatment in these reports.

Two studies restricted their samples to patients with neurological disorders associated with diabetes mellitus [57] and multiple sclerosis [250].

V. VARIABILITY IN TREATMENT METHODS

There is considerable variation in the methods used in biofeedback training for FI. Seventeen studies used EMG to improve EAS contraction [213-219, 233, 236, 245, 248, 251-258], two reports used anorectal ultrasound [226, 227] while the remaining reports used pressure sensors, anal rectal manometry, or the method used was not stated. Using manometry, some studies have trained short-latency EAS contractions in response to recognition of rectal distension, while others have also reinforced sustained EAS contraction to improve strength.

Two studies attempted a component analysis to determine whether rectal sensory training or EAS strength training was more effective in reducing faecal incontinence. They concluded that the primary mechanism responsible for symptomatic improvement was increased rectal sensitivity [221, 259]. However, limitations in the applied strength training procedures preclude valid comparisons of strength vs. sensory training methods in these studies [260]. In a between-group design, one study [233] compared out-patient intra-anal EMG strength training to EMG plus sensory training using an intra-rectal balloon, EMG plus home biofeedback training, and EMG, sensory training plus home biofeedback. No added benefit was found when the more comprehensive protocols were used, but small group sizes make this study inconclusive. On the other hand, a protocol that applied well-defined strength and sensory training procedures in the treatment of severe stool incontinence [235] found that responders to treatment improved both EAS squeeze pressure and rectal sensation. However, those that did not respond to treatment showed improvements in squeeze pressure equivalent to those who had responded, but did not show similar changes in rectal sensation, indicating that a certain level of rectal sensation is necessary for the reacquisition of continence.

Between 1 and 28 treatment sessions have been provided over 1 day to one year. Time spent in each treatment session ranged from 30 to 90 minutes or is not stated. Some studies controlled for the use of anti-diarrhoeals [235] or provided specific bowel management interventions prior to active treatment [223]. Other studies simply stated that patients were selected from those who had failed prior medical management. Alternatively, one study using a between-group design of 4 different treatments, compa-

red management and advice that included the use of anti-diarrhoeals to the same protocol plus: EAS exercise, EAS exercise and clinic biofeedback, or EAS exercise, biofeedback and the use of a home trainer [107]. In addition to diet and bowel management, other adjunctive treatments that have been used with biofeedback include electrical stimulation [245-247] and home biofeedback equipment [107, 233].

Most studies report that patients are instructed in home exercises but many do not specify the precise instructions given to the patients. Some studies state that subjects were simply instructed to contract the EAS with any feeling of rectal distension at home, while others provided structured sphincter exercise programmes. Three studies reported the degree of patient compliance to the assigned exercises [107, 235, 238]. However, a clear relationship between long-term improvement and continued exercise was not established. For example in one study, [235] only 26% of the subjects reported that they continued to perform PFM exercise at 12 month follow-up even though all subjects who improved initially (71%) maintained the gains at follow-up.

VI. RESPONSE TO TREATMENT: RANDOMISED CONTROLLED STUDIES

A Cochrane review [261] of randomised or quasi-randomised studies found 5 eligible studies that used biofeedback to treat faecal incontinence [221, 223, 246, 259, 262]. The Cochrane review included the outcome data from a total of 109 patients who were participants in four published papers and a single abstract [261]. Four of the 5 trials were found to have potential methodological bias and only 2 trials provided data in a form suitable for statistical analysis. The reviewers [261] felt that the evidence from the reviewed trials was insufficient to conclude that biofeedback treatments are useful in the reduction of FI. Additionally, the reviewers [261] found limited evidence to support the usefulness of any specific component of the biofeedback protocols tested with the exception of rectal sensitivity, which was consistently found to be associated with improved continence when biofeedback was administered.

Subsequent to the Cochrane review [261], four additional RCTs were published. One [233] study compared 4 different biofeedback protocols but did not have a non-biofeedback control group. Before patients were randomised to the different biofeedback protocols, they underwent medical and bowel management but the time period of initial intervention was unspecified. Although there was an overall 74% reduction in stool incontinence after biofeedback, no difference in effect was found between the different protocols. However, interpretation of effects was hampered by small group size.

Another study compared manometric biofeedback, anal rectal ultrasound biofeedback and sphincter exercise taught with digital examination alone [226]. All groups showed modest improvements in bowel control with 70% of the subjects reporting improvement. Improvements in bowel control were associated with modest changes in anorectal measures. However, neither manometric or ultrasound biofeedback provided added benefit to digitally taught sphincter exercise on any of the nine outcomes measures.

Another RCT [107] used four groups to compare the effects of a behavioural treatment from a specialized nurse that included advice on bowel management, diet, urge control and the use of anti-diarrhoeal medication, to the same behavioural management but with the addition of: sphincter exercises, exercises plus clinically administered biofeedback, or exercises, clinical biofeedback and home biofeedback. Each group received a median of five (range 1-9), 45-90 minute treatment sessions. No difference was found between groups on ratings of bowel control or physiological measures. These findings suggest that biofeedback did not provide any additional benefit to behavioural and medical management. When outcomes were collapsed across all subjects, modest reductions in symptoms of 54% and 53% were reported to occur in the advice and biofeedback groups, respectively.

Although manometric indices of sphincter function improved across all groups, an unexpected and unexplained numerical decrease in squeeze pressure was reported in one of the biofeedback groups, and minimal change was reported in the other biofeedback group. However, the outcome analysis was appropriately conducted on an intention to treat basis. In this study a 20% drop out rate was reported, which is consistent with other studies that have also reported drop out rate. But unlike Norton [107], most studies have reported outcomes only on subjects completing treatment rather than on an intention to treat basis. As result of the more stringent analysis, the Norton outcomes appear to be lower than many of the other reports.

VII. RESPONSE TO TREATMENT: UNCONTROLLED STUDIES

Most studies use some form of self-report collected from diaries, symptom questionnaires, or continence scores as the primary dependent variable. Criteria used to determine a successful outcome varies from 90% [205, 263] to 50% [264] reduction in incontinent episodes or subject's ratings of improvement. Most studies report an overall response rate that combines improvement and cure rates. Reported improvement ranges from 0% [215] to 100% [265]. Four studies [215, 234, 266, 267] reported improvements below 50%, 21 reported improvement from 50-75% and 23 studies reported improvement of 75% or greater, with 3 of these reporting improvement over 90%. Thirty studies reported the number of patients that achieved symptom-free or nearly symptom free status after treatment for a total of 353 of 773 (46%) patients fitting in the continent or nearly continent category.

Twenty-three studies specified a mean follow-up period greater than 12 months, 8 studies reported a range or mean follow-up that fell above 3 months and 29 studies reported follow-up less than 3 months or the time was not stated. Some studies reported outcomes at the time that treatment was completed [225, 233, 236, 246]. Of those studies having a longer follow-up, six studies reported follow-up results at 2 or more years [211, 217, 221, 238, 239, 251]. One study reported the results immediately after treatment and then at approximately 3 years follow-up and found that initial responders regressed as a function of time, leading to a conclusion that patients may need follow-up evaluation and treatment to maintain optimal function obtained with the initial treatment [239]. However, compliance to the home programme was not reported in this case. On the other hand, one study found that a majority of patients maintained, and in some cases exceeded, improvements reported immediately after treatment [238]. The long-term positive outcome in this study was attributed to the 83% home exercise compliance during the active treatment phase.

VIII. MECHANISM OF BIOFEEDBACK TRAINING EFFECTS

Many studies have reported changes in physiological variables such as anal resting and squeeze pressures and changes in rectal sensory threshold volumes as an outcome of biofeedback training. A few studies also have reported changes in the duration of EAS contraction [107, 222, 245] with researchers concluding that the ability to sustain an EAS contraction is more important than maximum squeeze pressure. For example, two studies found that it was the subjects who learned to extend the duration their sphincter contraction who developed continence [222, 257] after biofeedback training. This notion was supported by a study that compared the EAS fatigue rates of healthy controls to patients with constipation, seepage and stool incontinence and found that the EAS fatigue rate in incontinent patients was significantly increased compared to healthy controls and those with seepage [268].

Several studies report that improved rectal sensation is most consistently linked to improvements in continence as a result of biofeedback training [221, 236, 259]. Conversely, changes in sphincter strength are not consistently found to be associated with reductions in incontinence [210, 221, 224, 255, 267, 269] and in two studies, squeeze pressure was found to increase even in those patients that did not improve in bowel control [235, 263]. These inconsistencies have lead to questions regarding the mechanism(s) presumed to be responsible for symptom reduction as a result of biofeedback treatment [270].

IX. PATIENT CHARACTERISTICS PREDICTING WHO WILL RESPOND TO TREATMENT

With the exception of rectal sensation [235], there are no established criteria that might predict which patients would most likely benefit from biofeedback therapy. One paper found that in addition to a rectal sensory threshold of 50ml or less, a lower EAS and IAS response threshold and an urge threshold less than 100ml were associated with better outcomes after biofeedback [235]. Another study noted that positive outcomes were associated with those patients 55 years and older and having normal defecation patterns while poorer outcomes were associated with those younger than 55 and having abnormal evacuation patterns [240]. One study noted that the need for more than 3 biofeedback sessions and a poor early response to biofeedback predicted poor long-term improvement at follow-up [217]. Several reports noted that improvements were not associated with the presence or absence of defects found with ultrasound [107, 216, 225, 268] but one study found that less robust improvements were obtained in those having passive incontinence rather than urge inconti-

nence [225]. In the one study that found that no functional improvement was obtained with biofeedback, all subjects had severe neurogenic faecal incontinence and pudendal nerve neuropathy and absent sensation for bowel urge [215]. Another study found that patients with spinal cord lesions were least likely to respond to treatment [205]. On the other hand, one study did find that subjects with pudendal neuropathy could improve bowel control with biofeedback but were less likely to show improvement in EAS strength [267]. However, the effects of bowel management strategies were not controlled. Currently, there is little evidence that shows a relationship between pre-treatment anorectal function as measured by manometry and biofeedback outcomes, with the exception of rectal sensitivity which, if found to be greater than 100ml before treatment, is associated with a poor response to biofeedback [205]. As a result, some studies have excluded patients having rectal sensory thresholds greater than 100 ml.

X. PELVIC FLOOR MUSCLE EXERCISES

Historically, the use of pelvic floor muscle (PFM) exercise without biofeedback has not been used as a primary treatment for FI, unlike its application for UI where PFM exercise has been recommended as an intervention prior to the use of biofeedback. For FI, most exercise protocols have been used secondarily as anal sphincter exercise to augment the biofeedback protocol. Given that PFM exercise has been accepted as a valid treatment for UI, similar protocols may potentially improve FI as well. This is supported by one study [271] that found that levator ani function as measured by an intrarectal dynamometer was more related to severity of FI than EAS squeeze pressures obtained from anorectal manometry. Moreover, improvements in FI after biofeedback were associated with concomitant changes in levator ani strength but not changes in EAS strength. However, the two studies that have directly examined the effects of pelvic floor muscle exercise on FI report inconsistent outcomes [7, 226]. One RCT [226] found that when PFM exercises were prescribed in addition to a comprehensive behavioural management programme, no added benefit was obtained in primarily middle-aged subjects. In contrast, another RCT (7) found that when younger subjects were instructed in PFM exercises 3 months postnatally, the intervention group reported less FI at 12 months follow-up. However, in this study, the effect of PFM exercise was not studied separately from education. Given the limited data available, there is an obvious need to investigate the effectiveness of PFM exercise alone on FI because there are no known risks associated with its application and its cost is lower relative to biofeedback.

XI. SUMMARY AND RECOMMENDATIONS: BIOFEEDBACK, EXERCISES AND FI

In general, the outcomes reported from uncontrolled biofeedback studies for faecal incontinence have been favourable. However, most studies have been small and have a multitude of methodological flaws that include inadequate descriptions of subject characteristics and procedures, the use of heterogenic samples, and limited follow-up data. Only a handful of the non-randomised studies have made efforts to control for non-specific effects. In contrast to the mostly favourable outcomes reported in uncontrolled studies, two randomised controlled trials found no additional benefit when biofeedback was added to either a comprehensive behavioural and medical management programme [107] or to digitally taught sphincter exercise [226]. Although larger and randomly controlled, these trials [107, 226] have limitations.

We still lack precise knowledge of the mechanisms responsible for improvement when biofeedback is used to treat FI, and we do not yet understand the extent to which any specific biofeedback protocol alters parameters of anorectal function. The exception is rectal sensitivity, which is the single physiological parameter that has been reported to most consistently improve with biofeedback. However, not all subjects that show improvements in rectal sensitivity also develop continence. Thus, rectal sensitivity can be considered a necessary but not sufficient variable for continence. In contrast to rectal sensation, EAS strength has not been shown to consistently improve with biofeedback even when protocols have been directed to improve EAS function. Herein lies an essential empirical question for the field that must be answered before we can determine whether biofeedback is a useful tool in the treatment of stool incontinence. For, if changes in sphincter function are not observed when the stated goal of a biofeedback procedure is to improve sphincter strength, the validity of the protocol can be questioned and accordingly, conclusions based on the outcomes must be limited. Protocols then should

be appropriately altered to achieve the stated goal of changing EAS function before it can be adequately tested again. As in the field of psychophysiology from which biofeedback has evolved, a test of biofeedback effectiveness for any disorder cannot be accepted as an adequate evaluation of the treatment without evidence that the targeted physiology has been changed to a valid criterion of function [272]. Accordingly, any biofeedback protocol for faecal incontinence must be shown first to have altered some aspect of anorectal or bowel physiology, before it can be tested as a treatment. Without validation of the biofeedback procedure itself, the analysis of group effects tends to be primarily, a test of non-specific effects.

In summary, the primary problems in the biofeedback and pelvic floor muscle literature are:

1. Biofeedback studies for FI have employed a variety of methodologies that range from rectal sensitivity training to sphincter strength training but without standardisation of methodology.
2. Although uncontrolled studies using biofeedback for FI have reported mostly favourable outcomes, results from larger RCTs have not demonstrated a benefit of biofeedback over comprehensively administered behavioural and medical management or sphincter exercises alone.
3. There is a need to conduct further RCTs to determine whether specific biofeedback and pelvic floor muscle exercise protocols can alter physiological parameters of ano-rectal function with concomitant changes in bowel control.

RECOMMENDATIONS

Because recent RCTs have raised questions whether biofeedback provides a specific benefit relative to education and good clinical management despite a large body of uncontrolled studies supporting its efficacy, the consensus of the committee is that it is possibly effective but currently unproven.

1. The use of biofeedback as a treatment for FI is recommended after other behavioural medical management is tried given the numerous positive outcomes from uncontrolled trials, limitations in the current RCTs and low morbidity associated with its application. (Grade C recommendation)
2. PFM exercises are recommended as an early intervention in the treatment of FI based upon low cost and weak evidence suggesting efficacy (Grade C recommendation).

H. EXTERNAL ELECTRICAL STIMULATION FOR FI

Note: Surgically implanted electrodes are covered in Committee 19.

I. THE PHYSIOLOGICAL BASIS OF THE EFFECTS OF ELECTRICAL STIMULATION

Anal electrical stimulation (ES) was first described for treatment of faecal incontinence over 40 years ago, firstly as an implanted stimulator [273] and later as needle EMG stimulation [274]. As technology has developed, more comfortable surface electrodes have become available either as skin or intra-anal plug devices with a battery box. ES may be provided by a mains-powered machine or by a portable battery-powered stimulator. The advantage of a small device is that it is easier for the patient to use on a daily basis. Development of vaginal and anal electrodes make it possible for the patient to sit, stand or move during a training programme. There is at present no experimental evidence upon which to select optimum electrical stimulation parameters for different symptoms and clinical conditions.

An electric current of sufficient amplitude will excite nerve and muscle tissue in its field. In addition, the current will alter cell membrane potentials and therefore exert an influence on all living cells. The full extent of this influence is not known but studies have shown an increase in axonal budding following denervation and an increase in vascularisation and muscle bulk when the stimulating electrodes are placed in an area of striated muscles [275]. Also normalisation of the reflex activity of the bladder by using ES has been reported [276].

Maintenance of continence requires volitional cortical control which is dependent upon the sensory feedback from the ano-rectum [277] and the ability to sense rectal distension and impending defaecation and to relax or contract the striated muscles of pelvic

Table 7. Studies of electrical stimulation for FI

Author/year	No. of patients	Gender	Inclusion Criteria	Modality	Frequency	Pulse width	Stim duration
Fynes 1999	40	f	obstetric	anal plug ES + BFB + PFMT vs. vaginal BFB	20 & 50 Hz		5 sec (20 Hz), 8 sec 50 Hz
Jost 1998	30		atrophic muscles	anal plug ES	50 Hz		5 sec
Kienle 2003	70	37f, 33m	prior to stoma closure	anal plug ES + PFMT vs. BFB = PFMT	20 Hz		2 sec
Larpent	13	9f, 4m		anal plug ES			
Leroi 1999	33		self-selected	anal plug ES	50 Hz	1 ms	2 sec
Menard 1997	14	6f, 8m	congenital	anal plug + BFB	20 & 50 Hz		
Mills 1990	39	f		surface	10-33 Hz	80-200 ms	4 sec
Osterberg 1999	24	20f, 4m	idiopathic or "neurogenic"	anal & vaginal plug	25 Hz		1.5 sec
Pescatori 1991	15		partial FI	anal plug ES	20 Hz		10 sec
Peticca 2000	32			ES + BFB + PFMT	10-20 Hz		2 sec
Scheuer 1994	10		total incontinence, poor squeeze	anal plug	27-30 Hz		9 sec
Sylvester 1987	7			interferential current	10-100 HZ		

Author/year	Time off	Treatment frequency	Session duration	Intensity of stimulus	Length of treatment	Length of follow up	Outcome
Fynes 1999	8/30	weekly	15 min		12 weeks	Nil	ES + BFB better than BFB alone
Jost 1998	5 sec	daily	15 min	individual tolerance	3 months		
Kienle 2003	4 sec	1-2x per day	15 min		3 months min		BFB better than ES
Larpent		daily	30 min		10 days	8-25 months	8/13 improved
Leroi 1999	4 sec	2x day	15 min	individual tolerance	4 months		2 responded with loperamide
Menard 1997		2x week-monthly	5-10 min		mean 10 sessions		12/14 improved
Mills 1990	4 sec	daily home	10-60 mins		12 weeks +		20/39 completed; 17/20 improved
Osterberg 1999	3 sec		20 min	max tolerated	12 sessions in 4-5 weeks	3 months	11/24 improved
Pescatori 1991		daily	30 min		10 days		10 responded, anxiety improved
Peticca 2000	4 sec		5-15 mins	progressively increased	10 sessions	6-35 months	5 dropped out; 11 continent; 8 improved; 8 no change
Scheuer 1994	6-21 sec	2x day	30 min	individual tolerance	12 weeks		2/10 improved
Sylvester 1987		3x week	20 min	individual tolerance	12 sessions	1 month	not effective

floor [278]. The motor control of the pelvic floor muscles is a learned voluntary response albeit often at a subconscious level [212, 279].

Functional electrical stimulation activates both sensory and motor axons. The sensory axons send signals to the brain and it is thought may cause plastic changes in the representational area of a body part. The result of this is enlargement of the representation and improvement of awareness of the stimulated body part. This leads to better control of movements [278]. In theory ES may therefore reinforce weak functional signals that come from the pelvic floor musculature during the treatment [280], although this remains to be demonstrated experimentally.

There are many good studies of the use of ES to improve the function of other striated muscles and this knowledge may be applied to treatment of FI [281]. Stimulation parameters such as stimulation frequency, pulse width, on:off ratios, and current intensity are very important as it is possible to cause fatigue and other problems by using incorrect parameters, too long a treatment time or too high an intensity.

II. QUALITY OF DATA

There have been few studies of ES in faecal incontinence **(Table 7)**. Most studies included small numbers of patients and provided limited details of what was done within the intervention programmes.

III. RESULTS

One controlled study has been conducted [246]. Forty women with obstetric-related faecal incontinence were randomised to anal biofeedback and exercises with adjunctive electrical stimulation, or vaginal biofeedback and exercises (no electrical stimulation) with a different therapist. Both groups improved symptomatically, with no difference in symptoms between the groups. The stimulation group also improved manometric pressures. However, electrical stimulation was not the only variable in the study, there was no follow up beyond the 12-week study period and the difference in outcome found could have been the effect of a different therapist or biofeedback method. Another attempted controlled study in patients who had FI following repair of obstetric third degree tear abandoned stimulation because it caused discomfort [247].

A prospective study compared biofeedback and electrical stimulation in patients who, in the majority of cases, had so called “potential” faecal incontinence before stoma closure [241]. Forty patients were treated by biofeedback training, and 30 patients were treated by electrostimulation. All patients were encouraged to do pelvic floor exercises in addition to their training program. No description was given of stimulation. The biofeedback group was trained with a manometry-based balloon system. Biofeedback appeared to be more effective in this context than ES. Resting and squeeze pressure and resting and squeeze vector volume all increased significantly after biofeedback training ($p<0.05$ and <0.001). Only resting pressure and squeeze vector volume were significantly improved by electrostimulation ($p<0.05$ and <0.01). The increase in squeeze vector volume was significantly greater in the biofeedback group ($p=0.03$). The estimated median time period from commencement of training until stoma closure was 9 months in the biofeedback versus 21 months in the ES group.

Larpent and his colleagues [282] treated 13 patients with anorectal incontinence by ES in an uncontrolled study. The basal pressure of the external anal sphincter and the squeeze pressure improved significantly ($p<0.05$, $p<0.04$ respectively). Some studies may have used parameters that cause fatigue or a rest period that is too short for weak external sphincters [283]. Improved rectal sensation may be associated with a positive outcome [280]. Patients with the most severe incontinence and muscle impairment might be expected to benefit least [284]. Interferential current has not been found effective [285] and has since fallen out of use in most areas of physiotherapy.

IV. SUMMARY ON ELECTRICAL STIMULATION FOR FI

A Cochrane review of trials of electrical stimulation for faecal incontinence has concluded that "At present, there are insufficient data to allow reliable conclusions to be drawn on the effects of electrical stimulation in the management of faecal incontinence. There is a suggestion that electrical stimulation may have a therapeutic effect, but this is not certain" [286]. We concur with this conclusion.

Because there is a lack of consistency in electrical stimulation protocols and also a failure to use phy-

siological principles when employing using electrical stimulation, direct comparison between studies is impossible. There are many parameter and clinical applications that have not yet been investigated. We know little about which patients are likely to benefit from ES. Sensory awareness of the body schema and the possibility of improving this cortically by using ES may be important in motor relearning for those patients with severe sensory loss, but this has not been investigated.

V. FUTURE RESEARCH & RECOMMENDATIONS ON ELECTRICAL STIMULATION FOR FI

Randomised controlled trials with adequate sample sizes are necessary to investigate all aspects of the effectiveness of ES in faecal incontinence. Rectal hyposensitivity is common in patients with constipation and/or faecal incontinence [287]. Functional electrical stimulation combined with daily activities and its effectiveness in changing consciousness of the cortical schemes of the pelvic floor is one of the interesting future areas for research. When planning future research basic knowledge of electrical stimulation parameters and other important factors could be utilised from the other areas where electrical stimulation has been widely used [281].

I. CONCLUSIONS AND RECOMMENDATIONS

There is a number of potentially beneficial conservative and drug interventions for faecal incontinence in adults. The evidence base does not at this time provide strong evidence for efficacy or guidance for patient selection.

I. RECOMMENDATIONS FOR PRIMARY PREVENTION OF FI

1. Encourage and support public hygiene measures to reduce diarrhoeal diseases (Grade A).
2. Discourage episiotomy except in restricted circumstances (Grade B).
3. Discourage the use of internal anal sphincter myectomy for treatment of anal fissure and haemorrhoids (Grade A).
4. Continue the debate on elective Caesarean delivery to prevent sphincter laceration, but no recommendation is given by this working team.

II. RECOMMENDATIONS FOR SECONDARY PREVENTION OF FI

1. Health care providers should aggressively investigate all patients for FI who present with the most common risk factors: urinary incontinence, pelvic organ prolapse, diarrhoea, vaginal delivery with sphincter laceration, multiparity, mobility impairment, dementia, and other neurological conditions (Grade C).
2. In patients with frequent, loose stools, screen for drug side-effects, lactose intolerance, and high intake of artificial sweeteners or other foods likely to cause diarrhoea (Grade C).
3. Research is needed to assess the benefits of pelvic floor muscle training for the prevention of FI in women undergoing vaginal delivery.

III. LIFESTYLE INTERVENTIONS AND EDUCATION

At present there is no convincing evidence for these interventions in FI (Level D: possibly effective but unproven).

IV. DIET AND FLUID INTAKE

1. Soluble dietary fibre is recommended for the management of FI associated with loose stool. (Evidence level 1. Recommendation Grade B).
2. No studies of any other dietary intervention for managing faecal incontinence were found.

V. BOWEL MANAGEMENT AND RETRAINING PROGRAMMES

Level D evidence at present: possibly effective but unproven

VI. DRUG TREATMENT OF FI

1. Loperamide is supported for the treatment of diarrhoea-associated FI (Level 2 evidence, Grade B recommendation).
2. Laxatives can be used to treat constipation-associated faecal incontinence, (Level 2 evidence, Grade B recommendation).

VII. BIOFEEDBACK AND/OR ANAL SPHINCTER / PELVIC FLOOR EXERCISES

1. The use of biofeedback as a treatment for FI is recommended after other behavioural and medical management is tried given the numerous positive outcomes from uncontrolled trials, limitations in the current RCTs and low morbidity associated with its application (Grade C recommendation).
2. PFM exercises are recommended as an early intervention in the treatment of FI based upon low cost and weak evidence suggesting efficacy (Grade C recommendation).

VIII. ANAL ELECTRICAL STIMULATION

Level D evidence – possibly effective but unproven.

IX. OUTCOME MEASURES

The committee noted wide variety and inconsistency in reporting of outcomes. The committee agree with the findings of Committee 6: there is a need for development of validated outcome measures for studies of FI.

REFERENCES

1 Norton C, Christiansen J, Butler U, Harari D, Nelson R, Pemberton JH et al. Anal incontinence. In: Abrams P, Cardozo L, Khoury S, Wein A, editors. Incontinence. Plymouth: Health Books, 2002.

2 Perry S, Shaw C, McGrother C, Flynn RJ, Assassa RP, Dallosso H et al. The prevalence of faecal incontinence in adults aged 40 years or more living in the community. Gut 2002, 50:480-484.

3 Nelson R, Norton N, Cautley E, Furner S. Community-based prevalence of anal incontinence. JAMA 1995, 274 7 :559-561.

4 Roberts RO, Jacobsen SJ, Reilly WT, Pemberton JH, Lieber MM, Talley et al. Prevalence of combined fecal and urinary incontinence: a community-based study. Journal of the American Geriatrics Society 1999, 47 7 :837-841.

5 Fornell EU, Wingren G, Kjolhede P. Factors associated with pelvic floor dysfunction with emphasis on urinary and fecal incontinence and genital prolapse: an epidemiological study. Acta Obstetricia et Gynecologica Scandinavica 2004, 83:383-389.

6 Kalantar JS, Howell S, Talley NJ. Prevalence of faecal incontinence and associated risk factors, an underdiagnosed problem in the Australian community? Medical Journal of Australia 2002, 176 2 :54-57.

7 Chen GD, Hu SW, Chen YC, Lin TL, Lin LY. Prevalence and correlations of anal incontinence and constipation in Taiwanese women. Neurourol Urodyn 2003, 22 7 :664-669.

8 MacLennan AH, Taylor AW, Wilson DH, Wislon D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. British Journal of Obstetrics & Gynaecology 2000, 107 12 :1460-1470.

9 Walter S, Hallbook O, Gotthard R, Bengmark M, Sjodahl R. A population-based study on bowel habits in a Swedish community: prevalence of faecal incontinence and constipation. Scandinavian Journal of Gastroenterology 2002, 37 8 :911-916.

10 Edwards NI, Jones D. The prevalence of faecal incontinence in older people living at home. Age and Ageing 2001, 30:503-507.

11 Nakanishi N, Tatara K, Naramura H, Fujiwara H, Takashima Y, Fukunda H. Urinary and faecal incontinence in a community-residing older population in Japan. Journal of the American Geriatrics Society 1997, 45:215-219.

12 MacArthur C, Glazener CM, Wilson PD, Herbison P, Gee H, Lang GD. Obstetric practice and faecal incontinence three months after delivery. British Journal of Obstetrics & Gynaecology 2001, 108 7 :678-683.

13 Green JR, Soohoo SL. Factors associated with rectal injury in spontaneous deliveries. Obstet Gynecol 1989, 73 5 Pt 1 :732-738.

14 Combs CA, Robertson PA, Laros RK, Jr. Risk factors for third-degree and fourth-degree perineal lacerations in forceps and vacuum deliveries. Am J Obstet Gynecol 1990, 163 1 Pt 1 :100-104.

15 Drossman DA, Sandler RS, Broom CM, McKee DC. Urgency and fecal soiling in people with bowel dysfunction. Digestive Diseases & Sciences 1986, 31 11 :1221-1225.

16 Bliss DZ, Johnson S, Savik K, Clabots CR, Gerding DN. Fecal incontinence in hospitalized patients who are acutely ill. Nursing Research 2000, 49 2 :101-108.

17 Mintz ED, Weber JT, Guris D, Puhr N, Wells JG, Yashuk JC. An outbreak of Brainerd diarrhea among travelers to the Galapagos Islands. Journal of Infectious Disease 1998, 177 4 :1041-1045.

18 Kyne L, Merry C, O'Connell B, Kelly A, Keane C, O'Neill D. Factors associated with prolonged symptoms and severe disease due to Clostridium difficile. Age Ageing 1999, 28 2 :107-113.

19 Lustyk MK, Jarrett ME, Bennett JC, Heitkemper MM. Does a physically active lifestyle improve symptoms in women with irritable bowel syndrome? Gastroenterology Nursing 2001, 24 3 :129-137.

20 Johanson JF, Irizarry F, Doughty A. Risk factors for fecal incontinence in a nursing home population. J Clin Gastroenterol 1997, 24 3 :156-160.

21 Nelson R, Furner S, Jesudason V. Fecal incontinence in Wisconsin nursing homes: prevalence and associations. Dis Colon Rectum 1998, 41 10 :1226-1229.

22 Celik AF, Osar Z, Damci T, Pamuk ON, Ilkova H. How important are the disturbances of lower gastrointestinal bowel habits

in diabetic outpatients? Am J Gastroenterol 2001,96 4 :1314-1316.

23 Lowery SP, Srour JW, Whitehead WE, Schuster MM. Habit training as treatment of encopresis secondary to chronic constipation. Journal of Pediatric Gastroenterology & Nutrition 1985,4 3 :397-401.

24 Fishman L, Rappaport L, Schonwald A, Nurko S. Trends in referral to a single encopresis clinic over 20 years. Pediatrics 2003,111 5 Pt 1 :e604-e607.

25 Chassagne P, Landrin I, Neveu C, Czernichow P, Bouaniche M, Doucet J et al. Fecal incontinence in the institutionalized elderly: incidence, risk factors, and prognosis. American Journal of Medicine 1999,106 2 :185-190.

26 Von Wendt L, Simila S, Niskanen P, Jarvelin M-R. Development of bowel and bladder control in the mentally retarded. Developmental Medicine & Child Neurolgy 1990,32:515-518.

27 Cheskin LJ, Crowell MD, Kamal N, Rosen B, Schuster MM, Whitehead WE. The effects of acute exercise on colonic motility. Journal of Gastrointestinal Motility 1991,4:173-177.

28 Koffler KH, Menkes A, Redmond RA, Whitehead WE, Pratley RE, Hurley BF. Strength training accelerates gastrointestinal transit in middle-aged and older men.1992, 24:415-419.]. Med Sci Sports Exerc 1992,24:415-419.

29 Everhart JE, Go VL, Johannes RS, Fitzsimmons SC, Roth HP, White LI. A longitudinal survey of self-reported bowel habits in the United States. Digestive Diseases & Sciences 1989,34:1153-1162.

30 Sullivan S, Wong C. Runners' diarrhea. J Clin Gastroenterol 1992,14 2 :101-104.

31 Jorgensen S, Hein HO, Gyntelberg F. Heavy lifting at work and risk of genital prolapse and herniated lumbar disc in assistant nurses. Occupational Medicine 1994,44 :47-49.

32 Zetterstrom J, Lopez A, Anzen B, Norman M, Holmstrom B, Mellgren A. Anal sphincter tears at vaginal delivery: risk factors and clinical outcome of primary repair. Obstetrics & Gynecology 1999,94 1 :21-28.

33 Faltin D, Sangalli MR, Curtin F, Morabia A, Weil A. Prevalence of anal incontinence and other anorectal symptoms in women. International Urogynecology Journal and Pelvic Floor Dysfunction 2001,12 2 :117-120.

34 Fenner DE, Genberg B, Brahma P, Marek L, DeLancey JO. Fecal and urinary incontinence after vaginal delivery with anal sphincter disruption in an obstetrics unit in the United States. American Journal of Obstetrics & Gynecology 2003,189 6 :1543-1549.

35 Tiainen J, Matikainen M. Health-related quality of life after ileal J-pouch-anal anastomosis for ulcerative colitis: long-term results. Scandinavian Journal of Gastroenterology 1999,34 6 :601-605.

36 McIntyre P, Pemberton JH, Wolff BG, Beart RW, Dozois RR. Comparing functional results one year and ten years after ileal pouch-anal anastomosis for chronic ulcerative colitis. Dis Colon Rectum 1994,37 4 :303-307.

37 Hewett PJ, Stitz R, Hewett MK. Comparison of the functional results of restorative proctocolectomy for ulcerative colitis between the J and W configuration ileal pouches with sutured ileoanal anastomosis. Dis Colon Rectum 1995,38 6 :567-572.

38 Fazio VW, O'Riordain MG, Lavery IC, Church JM, Lau P, Strong SA et al. Long-term functional outcome and quality of life after stapled restorative proctocolectomy. Annals of Surgery 1999,230 4 :575-584.

39 Grotz RL, Pemberton JH, Ferrara A, Hanson RB. Ileal pouch pressures after defecation in continent and incontinent patients. Dis Colon Rectum 2004,37 11 :1073-1077.

40 Bjork J, Akerbrant H, Iselius L, Svenberg T, Oresland T, Pahlman L. Outcome of primary and secondary ileal pouch-anal anastomosis and ileorectal anastomosis in patients with familial adenomatous polyposis. Dis Colon Rectum 2001,44 7 :984-992.

41 Hildebrandt U, Lindemann W, Ecker KW, Walter P. [The coloanal pouch: indications, function and results]. Zentralbl Chir 1994,119 12 :886-891.

42 Barrier A, Martel P, Dugue L, Gallot D, Malafosse M. [Direct and reservoir colonic-anal anastomoses. Short and long term results]. Annales Chirurgiae 2001,126 1 :18-25.

43 Paty PB, Enker WE, Cohen AM, Minsky BD, Friedlander-Klar H. Long-term functional results of coloanal anastomosis for rectal cancer. American Journal of Surgery 1994,167 1 :90-94.

44 Thomas DS, Beck SL, Dayton MT, Kirchhoff KT. Evaluation of sceduled J-pouch irrigations on decreasing stool frequency after ileoanal pull-through and ileostomy closure. Journal of Wound, Ostomy, & Continence Nursing 1996,23:261-268.

45 Nyam DC, Pemberton JH. Long-term results of lateral internal sphincterotomy for chronic anal fissure with particular reference to incidence of fecal incontinence. Dis Colon Rectum 1999,42 10 :1306-1310.

46 Adolfsson J, Helgason AR, Dickman P, Steineck G. Urinary and bowel symptoms in men with and without prostate cancer: results from an observational study in the Stockholm area. European Urology 1998,33 1 :11-16.

47 Potosky AL, Legler J, Albertsen PC, Stanford JL, Gilliland FD, Hamilton AS et al. Health outcomes after prostatectomy or radiotherapy for prostate cancer: results from the Prostate Cancer Outcomes Study. J Natl Cancer Inst 2000,92 19 :1582-1592.

48 Bishoff JT, Motley G, Optenberg SA, Stein CR, Moon KA, Browing SM et al. Incidence of faecal and urinary incontinence following radical perineal and retropubic prostatectomy in a national population. Journal of Urology 1998,160:454-458.

49 Johannsson HO, Graf W, Pahlman L. Long-term results of haemorrhoidectomy. European Journal of Surgery 2002,168 8-9 :485-489.

50 Khan S, Pawlak SE, Eggenberger JC, Lee CS, Szilagy EJ, Wu JS. Surgical treatment of hemorrhoids: prospective, randomized trial comparing closed excisional hemorrhoidectomy and the Harmonic Scalpel technique of excisional hemorrhoidectomy. Dis Colon Rectum 2001,44 6 :845-849.

51 Vordermark D, Schwab M, Flentje M, Sailer M, Kolbl O. Chronic fatigue after radiotherapy for carcinoma of the prostate: correlation with anorectal and genitourinary function. Radiother Oncol 2002,62 3 :293-297.

52 Bergmark K, vall-Lundqvist E, Dickman PW, Henningsohn L, Steineck G. Patient-rating of distressful symptoms after treatment for early cervical cancer. Acta Obstetricia et Gynecologica Scandinavica 2002,81 5 :443-450.

53 Broens P, Van LE, Penninckx F, Kerremans R. Clinical and manometric effects of combined external beam irradiation and brachytherapy for anal cancer. International Journal of Colorectal Disease 1998,13 2 :68-72.

54 Bytzer P, Talley NJ, Leemon M, Young LJ, Jones MP, Horowitz M. Prevalence of gastrointestinal symptoms associated with diabetes mellitus: a population-based survey of 15,000 adults. Archive of Internal Medicine 2001,161 16 :1989-1996.

55 Icks A, Haastert B, Rathmann W, Wareham N. Prevalence of gastrointestinal symptoms in patients with type 2 diabetes: a population-based study. Archive of Internal Medicine 2002,162 9 :1067-1069.

56 Epanomeritakis E, Koutsoumbi P, Tsiaoussis I, Ganotakis E, Vlata M, Vassilakis JS et al. Impairment of anorectal function in diabetes mellitus parallels duration of disease. Dis Colon Rectum 1999,42 11 :1394-1400.

57 Wald A, Tunuguntla AK. Anorectal sensorimotor dysfunction in

faecal incontinence in diabetes mellitus. New England Journal of Medicine 1984,310:1282-1287.

58 Harari D, Sarkarati M, Gurwitz JH, McGlinchey-Berroth G, Minaker KL. Constipation-related symptoms and bowel program concerning individuals with spinal cord injury. Spinal Cord 1997,35:394-401.

59 De Looze DA, Van Laere M, De Muynck MC, Beke R, Elewaut AG. Constipation and other chronic gastrointestinal problems in spinal cord injury patients. Spinal Cord 1998,36 1 :63-66.

60 Glickman S, Kamm MA. Bowel dysfunction in spinal-cord-injury patients. Lancet 1996,347 9016 :1651-1653.

61 Schonherr MC, Groothoff JW, Mulder GA, Eisma WH. Functional outcome of patients with spinal cord injury: rehabilitation outcome study. Clinical Rehabilitation 1999,13 6 :457-463.

62 Han TR, Kim JH, Kwon BS. Chronic gastrointestinal problems and bowel dysfunction in patients with spinal cord injury. Spinal Cord 1998,36 7 :485-490.

63 Malone PS, Wheeler RA, Williams JE. Continence in patients with spina bifida: long term results. Archives of Disease in Childhood 1994,70:107-110.

64 Whitehead WE, Parker L, Bosmajian L, Morrill-Corbin ED, Middaugh S, Garwood M et al. Treatment of fecal incontinence in children with spina bifida: comparison of biofeedback and behavior modification. Archives of Physical Medicine & Rehabilitation 1986,67 4 :218-224.

65 Nakayama H, Jorgensen HS, Pedersen PM, Raaschou HO, Olsen TS. Prevalence and risk factors of incontinence after stroke: The Copenhagen Stroke Study. Stroke 1997,28 1 :58-62.

66 Harari D, Coshall C, Rudd AG, Wolfe CD. New-onset fecal incontinence after stroke: prevalence, natural history, risk factors, and impact. Stroke 2003,34 1 :144-150.

67 Foxx-Orenstein A, Kolakowsky-Hayner S, Marwitz JH, Cifu DX, Dunbar A, Englander J. Incidence, risk factors, and outcomes of fecal incontinence after acute brain injury: findings from the Traumatic Brain Injury Model Systems national database. Archives of Physical Medicine & Rehabilitation 2003,84 2 :231-237.

68 Hennessey A, Robertson NP, Swingler R, Compston DA. Urinary, faecal and sexual dysfunction in patients with multiple sclerosis. Journal of Neurology 1999,246 11 :1027-1032.

69 Trezza M, Krogh K, Egekvist H, Bjerring P, Laurberg S. Bowel problems in patients with systemic sclerosis. Scandinavian Journal of Gastroenterology 1999,34 4 :409-413.

70 Hinds JP, Eidelman BH, Wald A. Prevalence of bowel dysfunction in multiple sclerosis. Gastroenterology 1990,98:1538-1542.

71 Buchanan RJ, Wang S, Ju H. Analyses of the minimum data set: comparisons of nursing home residents with multiple sclerosis to other nursing home residents. Mult Scler 2002,8 6 :512-522.

72 Caruana BJ, Wald A, Hinds JP, Eidelman BH. Anorectal sensory and motor function in neurogenic faecal incontinence. Gastroenterology 1991,100:465-470.

73 Nordenbo AM. Bowel dysfunction in multiple sclerosis. Sexuality & Disability 1996,14 1 :33-39.

74 Holschneider AM. Treatment and functional results of anorectal continence in children with imperforate anus. Acta Chir Belg 1983,82 3 :191-204.

75 Molander ML, Frenckner B. Anal sphincter function after surgery for high imperforate anus--a long term follow-up investigation. Zeitschrift fur Kinderchirurgie 1985,40 2 :91-96.

76 Whitehead WE, Schuster MM. Anorectal physiology and pathophysiology. Am J Gastroenterol 1987,82 6 :487-497.

77 Emblem R, Diseth T, Morkrid L, Stien R, Bjordal R. Anal endosonography and physiology in adolescents with corrected low anorectal anomalies. J Pediatr Surg 1994,29 3 :447-451.

78 Rintala RJ, Lindahl H. Is normal bowel function possible after repair of intermediate and high anorectal malformations? J Pediatr Surg 1995,30 3 :491-494.

79 Lin JN. Anorectal malformations--update 1998. Chang-Keng i Hsueh Tsa Chih 1998,21 3 :237-250.

80 Weber AM. The perspective of a gynecologist on treatment-related research for fecal incontinence in women. Gastroenterology 2004,126:S169-S171.

81 Lal M, Mann H, Callender R, Radley S. Does cesarean delivery prevent anal incontinence? Obstetrics & Gynecology 2003,101 2 :305-312.

82 Hofmeyr GJ, Hannah ME. Planned Caesarean section for term breech delivery. Cochrane Database of Systematic Reviews 2001,1 CD000166 .

83 Al-Mufti R, McCarthy A, Fisk NM. Obstetricians' personal choice and mode of delivery. Lancet 1996,347:544.

84 Tranquilli AL, Garzetti GG. A new ethical and clinical dilemma in obstetric practice: cesarean section "on maternal request". American Journal of Obstetrics & Gynecology 1997,177 1 :245-246.

85 Bewley S, Cockburn J. The unfacts of 'request' caesarean section. British Journal of Obstetrics & Gynaecology 2002,109 6 :597-605.

86 Hannah ME, Hannah WJ, Hodnett ED, Chalmers B, Kung R, Willan A et al. Outcomes at 3 months after planned cesarean vs planned vaginal delivery for breech presentation at term: the international randomized Term Breech Trial. JAMA 2002,287 14 :1822-1831.

87 Jorge JM, Wexner SD, Morgado PJ, James K, Nogueras JJ, Jagelman DG. Optimization of sphincter function after the ileoanal reservoir procedure. Dis Colon Rectum 1994,37:419-423.

88 MacArthur C, Bick DE, Keighley MRB. Faecal incontinence after childbirth. British Journal of Obstetrics & Gynaecology 1997,104:46-50.

89 Glazener CM, Herbison GP, Wilson PD, MacArthur C, Lang GD, Gee H et al. Conservative management of persistent postnatal urinary and faecal incontinence: randomised controlled trial. BMJ 2001,323 7313 :593-596.

90 Meyer S, Hohlfield P, Achtari C, De Grandi P. Pelvic floor education after vaginal delivery. Obstetrics & Gynecology 2001,97 5 :673-677.

91 Wilson PD, Herbison P. A randomised controlled trial of pelvic floor muscle exercises to treat postnatal urinary incontinence. International Urogynecology Journal 1998,9:257-264.

92 Sleep J, Grant A. Pelvic floor exercises in postnatal care. Midwifery 1987,3 4 :158-164.

93 Norton C. Nurses, Bowel Continence, Stigma and Taboos. Journal of Wound Ostomy and Continence Nursing 2004,31 2 :85-94.

94 Norton C, Chelvanayagam S. Bowel Continence Nursing. Beaconsfield: Beaconsfield Publishers, 2004.

95 Whitehead WE, Wald A, Norton N. Treatment options for fecal incontinence: consensus conference report. Dis Colon Rectum 2001,44:131-144.

96 Schnelle JF, Alessi C, Simmons SF, Al-Samarrai NR, Beck JG, Ouslander JG. Translating clinical research into practice: a randomized controlled trial of exercise and incontinence care with nursing home residents. Journal of the American Geriatrics Society 2002,50:1476-1483.

97 Bates-Jensen BM, Alessi C, Al-Samarrai NR, Schnelle JF. The effects of an exercise and incontinence intervention on skin health outcomes in nursing home residents. Journal of the American Geriatrics Society 2003,51:348-355.

98 Resende TL, Brocklehurst JC, O'Neill PA. A pilot study on the

effect of exercise and abdominal massage on bowel habit in continuing care patients. Clinical Rehabilitation 1993,7 3 :204-209.

99 Scott AM, Kellow JE, Eckersley GM, Nolan JM, Jones MP. Cigarette smoking and nicotine delay postprandial mouth-cecum transit time. Digestive Diseases & Sciences 1992,37 10 :1544-1547.

100 Rausch T, Beglinger C, Alam N, Meier R. Effect of transdermal application of nicotine on colonic transit in healthy nonsmoking volunteers. Neurogastroenterol Mot 1998,10:263-270.

101 Bump RC, Norton PA. Epidemiology and natural history of pelvic floor dysfunction. Obstetrics & Gynecology Clinics of North America 1998,25:723-746.

102 Chaliha C, Kalia V, Stanton SL, Monga AK, Sultan AH. Antenatal prediction of postpartum urinary and fecal incontinence. Obstetrics & Gynecology 1999,94 5 :689-694.

103 Heck AM, Calis KA, McDuffie JR, Carobene SE, Yanovski JA. Additive gastrointestinal effects with concomitant use of olestra and orlistat. Annals of Pharmacotherapy 2002,36 6 :1003-1005.

104 Fox M, Thumshirn M, Menne D, Stutz B, Fried M, Schwizer W. The pathophysiology of faecal spotting in obese patients during treatment with orlistat. Alimentary Pharmacology & Therapeutics 2004,19 3 :311-321.

105 White H. Making toilets more accessible for individuals with a disability. In: Norton C, Chelvanayagam S, editors. Bowel Continence Nursing. Beaconsfield, UK: Beaconsfield Publishers, 2004.

106 Chiarelli P, Markwell S. Let's get things moving: overcoming constipation. East Dereham: Neen Healthcare, 1992.

107 Norton C, Chelvanayagam S, Wilson-Barnett J, Redfern S, Kamm MA. Randomized controlled trial of biofeedback for fecal incontinence. Gastroenterology 2003,125:1320-1329.

108 Heymen S, Jones KR, Ringel Y, Scarlett Y, Drossman DA, Whitehead WE. Biofeedback for fecal incontinence and constipation: the role of medical management and education. Gastroenterology 2001,120 Suppl 1 :A397.

109 Harari D, Norton C, Lockwood L, Swift C. Treatment of constipation and faecal incontinence in stroke patients: randomised controlled trial. Stroke 2004: 35, 2549-2555.

110 Harari D, Norton C, Lockwood L, Swift C. Treatment of constipation and faecal incontinence in stroke patients: randomised controlled trial. Age and Ageing 2004,abstract.

111 Clemesha L, Davies E. Educting home carers on faecal incontinence in people with dementia. Nursing Standard 2004,18 34:33.

112 Freeman RM, Baxby K. Hypnotherapy for incontinence caused by the unstable detrusor. British Medical Journal 1982,284:1831-1834.

113 Taitz LS, Wales JKH, Urwin OM, Molnar D. Factors associated with outcome in management of defecation disorders. Archives of Disease in Childhood 1986,61:472-477.

114 Bliss DZ, McLaughlin J, Jung H, Savik K, Jensen L, Lowry AC. Comparison of the nutritional composition of diets of persons with fecal incontinence and that of age and gender-matched controls. Journal of Wound Ostomy and Continence Nursing 2000,27 2 :90-97.

115 Chrysos E, Athanasakis E, Tsiaoussis J, Zoros O, Nickolopoulos A, Vassilakis JS et al. Rectoanal motility in Crohn's disease patients. Dis Colon Rectum 2001,44 10 :1509-1513.

116 Kangas E, Hiltunen KM, Matikainen M. Anorectal function in Crohn's disease. Annales Chirurgiae et Gynaecologiae 1992,81:43-47.

117 Buchmann P, Mogg GAG, Alexander-Williams J, Allan RN, Keighley MRB. Relationship of proctitis and rectal capacity in Crohn's disease. Gut 1980,21:137-140.

118 Farthing MJG, Lennard-Jones JE. Sensibility of the rectum to distension and the anorectal distension reflex in ulcerative colitis. Gut 1978,19:64-69.

119 Drossman DA, Li Z, Andruzzi E, Temple RD, Talley NJ, Thompson WG. U.S. householder survey of functional gastrointestinal disorders. Digestive Diseases and Sciences 1993,38 9 :1569-1580.

120 Bliss DZ, Fischer LR, Savik K. Self-care practices for managing fecal incontinence by elders at managed care clinics [Abstract No. 169]. The Gerontologist 2001,41:188.

121 Chassagne P, Jego A, Gloc P, Capet C, Trivalle C, Doucet J et al. Does treatment of constipation improve faecal incontinence in institutionalized elderly patients? Age & Ageing 2000,29 2:159-164.

122 Read NW, Abouzekry L, Read M, Howell P, Ottewell D, Donnelly TC. Anorectal function in elderly patients with faecal impaction. Gastroenterology 1985,89:959-966.

123 Bengmark S. Pre- pro- and synbiotics. Current Opinion in Clinical Nutrition & Metabolic Care 2001,4:571-579.

124 Eherer AJ, Santa Ana C, Poston J, Fordtran JS. Effect of psyllium, calcium polycarbophil, and wheat bran on secretory diarrhea induced by phenolphthalein. Gastroenterology 1992, 104:1007-1012.

125 Norton C, Chelvanayagam S. Conservative management of faecal incontinence in adults. In: Norton C, Chelvanayagam S, editors. Bowel continence nursing. Beaconsfield: Beaconsfield Publishers, 2004: 114-131.

126 Ardron ME, Main ANH. Management of constipation. British Medical Journal 1990,300:1400.

127 Aurisicchio LN, Pitchumoni CS. Lactose intolerance. Recognizing the link between diet and discomfort. Postgraduate Medicine 1994,95:113-120.

128 Martini MC, Kukielka D, Savaiano DA. Lactose digestion form yogurt: Influence of a meal and additional lactose. American Journal of Clinical Nutrition 1991,53:1253-1258.

129 Inman-Felton AE. Overview of lactose maldigestionlactase nonpersistence . Journal of the American Dietetic Association 1999,99 4 :481-489.

130 Phillips SF, Greenberger NJ. The diverse spectrum of irritable bowel syndrome. Hospital Practice 1995,30:69-78.

131 Ledochowski M, Widner B, Bair H, Probst T, Fuchs D. Fructose and sorbitol reduced diet improves mood and gastrointestinal disturbances in fructose malabsorbers. Scandinavian Journal of Gastroenterology 2000,35:1048-1052.

132 Brown SR, Cann PA, Read NW. Effect of coffee on distal colon function. Gut 1990,31:450-453.

133 Dennish GW, Castell DO. Caffeine and the lower esophageal sphincter. American Journal of Digestive Diseases 1972,17:993-996.

134 Debas HT, Cohen MM, Holbitsky IB, Harrison RC. Caffeine stimulated acid & pepsin secretion: Dose response studies. Scandinavian Journal of Gastroenterology 1971,6:453-457.

135 Wald A, Back C, Bayliss TM. Effect of caffeine in the human small intestine. Gastroenterology 1976,71:738-742.

136 Acquaviva F, DeFrancesco A, Andriulli A, Piantino P, Arrigoni A, Massarenti P et al. Effects of regular and decaffeinated coffee on serum gastrin levels. J Clin Gastroenterol 1986,8:150-153.

137 Izbeki F, Wyttmann T, Csati S, Jeszenszky E, Lonovics J. Izbeki, F., Wyttmann, T., Csati, S., Jeszenszky, E., & Lonovics, J.2001 . Opposite effects of acute and chronic administration of alcohol on gastric emptying and small bowel transit in rat. Alcohol & Alcoholism, 36, 304-308. Alcohol & Alcoholism 2001,36:304-308.

138 Bujanda L. The effects of alcohol consumption upon the gastrointestinal tract. Am J Gastroenterol 2000,95:3374-3382.

139 Bode C, Bode JC. Effect of alcohol consumption on the gut. Best Practice & Research in Clinical Gastroenterology 2003,17:575-592.

140 Bliss DZ, Jung H, Savik K, Lowry AC, LeMoine M, Jensen L et al. Supplementation with dietary fiber improves fecal incontinence. Nursing Research 2001,50 4 :203-213.

141. Korula, J. EBM reviews: dietary fiber supplements with psyllium or gum arabic reduced fecal incontinence in community-living adults. American College of Physicians Journal Club 2002, 136 (1): 23-24.

142 Doughty D. A physiologic approach to bowel training. Journal of Wound, Ostomy, & Continence Nursing 1996,23 1 :46-56.

143 Narducci F, Bassotti G, Bagurri M, Morelli A. Twenty-four hour manometric recording of colonic motor activity in healthy man. Gut 1987,28:17-25.

144 Bassotti G, Crowell MD, Cheskin LJ, Chami TN, Schuster MM, Whitehead WE. Physiological correlates of colonic motility in patients with irritable bowel syndrome. Zeitschr Gastroenterol 1998,36:811-817.

145 Snooks SJ, Barnes PRH, Swash M, Henry MM. Damage to the innervation of the pelvic floor musculature in chronic constipation. Gastroenterology 1985,89:977-981.

146 Lubowski DZ, Swash M, Nicholls RJ. Increases in pudendal nerve terminal motor latency with defecation straining. British Journal of Surgery 1988,75:1095-1097.

147 Spence-Jones C, Kamm MA, Henry MM. Bowel dysfunction: a pathogenic factor in uterovaginal prolapse and urinary stress incontinence. British Journal of Obstetrics & Gynaecology 1994,101:147-152.

148 Barrett JA. Faecal incontinence and related problems in the older adult. London: Edward Arnold, 1993.

149 Brazzelli M, Griffiths P. Behavioural and cognitive interventions with or without other treatments for defaecation disorders in children. Cochrane Library John Wiley & Sons Ltd, Chichester, UK 2002,Issue 2.

150 Stokes G. Psychological approaches to bowel care in older people with dementia. In: Potter J, Norton C, Cottenden A, editors. Bowel care in older people. London: Royal College of Physicians, 2002: 97-109.

151 Dalrymple N, Rubie LA. Toilet training. Journal of Autism and Developmental Disabilities 1992,22:265-270.

152 Smith L, Smith P. Bowerl control and intellectual disability. In: Norton C, Chelvanayagam S, editors. Bowel continence nursing. Beaconsfield UK: Beaconsfield Publishers, 2004: 204-216.

153 Norton C, Chelvanayagam S. Methodology of biofeedback for adults with fecal incontinence - a program of care. Journal of Wound, Ostomy, & Continence Nursing 2001,28:156-168.

154 Doughty D. Urinary and fecal incontinence: nursing management2nd edition . St. Louis: Mosby, 2000.

155 Gauderer MWL, Decou JM, Boyle JT. Sigmoid irrigation tube for the management of chronic evacuation disorders. J Pediatr Surg 2002,37 3 :348-351.

156 Bruce RG, El-Galley RE, Wells J, Galloway NT. Antegrade continence enema for the treatment of fecal incontinence in adults: use of gastric tube for catheterizable access to the descending colon. Journal of Urology 1999,161 6 :1813-1816.

157 Shandling B, Chait PG, Richards HF. Percutaneous cecostomy: a new technique in the management of faecal incontinence. J Pediatr Surg 1996,31 4 :1-5.

158 Eltringham MT, Watson C, Bain IM, Green SE, Yiannakou JY. Percutaneous endoscopic colostomy PEC- role in recurrent sigmoid volvulus and chronic constiption Abstract 071. Gut 2004,53 supplement 111 :A19.

159 Krogh K, Kvitzau B, Jorgensen TM, Laurberg S.Treatment of anal incontinence and constipation with transanal irrigation . Ugeskrift for Laeger 1999,161 3 :253-256.

160 Briel JW, Schouten WR, Vlot EA, Smits S, van K, I. Clinical value of colonic irrigation in patients with continence disturbances. Dis Colon Rectum 1997,40 7 :802-805.

161 Crawshaw A. How to establish a rectal irigation service. Gastrointestinal Nursing 2004,2 2 :29-31.

162 Puet TA, Phen L, Hurst DL. Pulsed irrigation enhanced evacuation: new method for treating faecal impaction. Archives of Physical Medicine & Rehabilitation 1991,72:935-936.

163 Shandling B, Gilmour RF. The enema continence catheter in spina bifida: successful bowel management. J Pediatr Surg 1987,22:271-273.

164 Lipak GS, Revell GM. Management of bowel dysfunction in children with spinal cord disease or injury by means of the enema continence catheter. Journal of Pediatrics 1992,120:190-194.

165 Scholler-Gyure M, Nesselaar CH, van Wieringen H, Van Gool JD. Treatment of defaecation disorders by colonic enemas in children with spina bifida. European Journal of Pediatric Surgery 1996,6 Suppl1 :32-34.

166 Christensen P, Olsen N, Krogh K, Bacher T, Laurberg S. Scintigraphic assessment of retrograde colonic washout in fecal incontinence and constipation. Dis Colon Rectum 2003,46 1 :68-76.

167 Gattuso JM, Kamm MA, Myers C, Saunders B, Roy A. Effect of different infusion regimens on colonic motility and efficacy of colostomy irrigation. British Journal of Surgery 1996,83:1459-1462.

168 Gramlich T, Puet TA. Long-term saftey of pulsed irrigation evacuationPIEuse with chronic bowel conditions. Digestive Diseases & Sciences 1998,43 8 :1831-1834.

169 Yerkes EB, Rink RC, King S, Cain MP, Kaefer M, Casale AJ. Tap water and the Malone antegrade continenc enema: a safe combintion? Journal of Urology 2001,166 4 :1476-1478.

170 Chertow GM. Hyponatraemia from tap-water enema. Lancet 1994,344:748.

171 Biberstein M, Parker BA. Enema-induced hyperphosphatemia. American Journal of Medicine 1985,79:645-646.

172 Hunter MF, Ashton MR, Griffiths DM, Ilangovan P, Roberts JP, Walker V. Hyperphosphataemia after enemas in childhood: prevention and treatment. Archives of Disease in Childhood 1993,68:233-234.

173 Bell AM. Colonic perforation with a phosphate enema. Journal of the Royal Society of Medicine 1990,83:54-55.

174 Addison R, Smith M. Digital rectal examination and manual removal of faeces. London: Royal College of Nursing, 2000.

175 Wiesel P, Bell S. Bowel dysfunction: assessment and management in the neurological patient. In: Norton C, Chelvanayagam S, editors. Bowel continence nursing. Beaconsfield: Beaconsfield Publishers, 2004.

176 Coggrave M, Wiesel P, Norton C, Brazzelli M. Bowel management for adults with neurological disease or injury. Cochrane review . The Cochrane Library, John Wiley & Sons, Chichester, UK 2003,Issue 2, 2004.

177 Cheetham M, Brazzelli M, Norton C, Glazener CM. Drug treatment for faecal incontinence in adults Cochrane review . The Cochrane Library, John Wiley & Sons, Chichester, UK 2003,Issue 2, 2004.

178 Read M, Read NW, Barber DC, Duthie HL. Effects of loperamide on anal sphincter function in patients complaining of chronic diarrhoea with faecal incontinence and urgency. Digestive Diseases and Sciences 1982,27:807-814.

179 Hallgren T, Fasth S, Delbro DS, Nordgren S, Oresland T, Hulten L. Loperamide improves anal sphincter function and continence after restorative proctocolectomy. Digestive Diseases & Sciences 1994,39 12 :2612-2618.

180 Sun WM, Read NW, Verlinden M. Effects of loperamide oxide in gastrointestinal transit time and anorectal function in patients with chronic diarrhoea and faecal incontinence. Scandinavian Journal of Gastroenterology 1997,32:34-38.

181 Kekomaki M, Vikki P, Gordin A, Salo H. Loperamide as a symptomatic treatment in pediatric surgery: a double-blind cross-over study. Z Kinderchir 1981,32:237-243.

182 Palmer KR, Corbett CL, Holdsworth CD. Double blind cross over study comparing loperamide codeine and diphenoxylate in the treatment of chronic diarrhoea. Gastroenterology 1980,79:1272-1275.

183 Martenson JA, Bollinger JW, Sloan JA, Novotny PJ, Urias RE, Shanahan TG et al. Sucralfate in the prevention of treatment-induced diarrhea in patients receiving pelvic radiation therapy: A North Central Cancer Treatment Group phase III double-blind placebo-controlled trial. Journal of Clinical Oncology 2000,18:1239-1245.

184 Kusunoki M, Shoji Y, Ikeuchi H, Yamagata K, Yamamura T, Utsunomiya J. Usefulness of valporate sodium for treatment of incontinence after ileoanal anastomosis. Surgery 1990,107:311-315.

185 Santoro GA, Eitan BZ, Pryde A, Bartolo DC. Open study of low-dose amitriptyline in the treatment of patients with idiopathic fecal incontinence. Dis Colon Rectum 2000,43:1676-1681.

186 Carapeti EA, Kamm MA, Evans BK, Phillips RK. Topical phenylephrine increases anal sphincter resting pressure. British Journal of Surgery 1999,86 2 :267-270.

187 Carapeti EA, Kamm MA, Phillips RK. Randomized controlled trial of topical phenylephrine in the treatment of faecal incontinence. British Journal of Surgery 2000,87:38-42.

188 Cheetham M, Kamm MA, Phillips RK. Topical phenylephrine increases anal canal resting pressure in patients with faecal incontinence. Gut 2001,48:356-359.

189 Rutter M, Seeley WW, Ritchey ML, McGuire EJ. New York: Robert E. Krieber Publications, 1981.

190 Bloom DA, Seeley WW, Ritchey ML, McGuire EJ. Toilet habits and continence in children: an opportunity sampling in search of normal parameters. J Urol 1993,149 5 :1087-1090.

191 Fishman L, Rappaport L, Schonwald A, Nurko S. Trends in referral to a single encopresis clinic over 20 years. Pediatrics 2003,111 5 Pt 1 :e604-e607.

192 Dey AN. Characteristics of elderly nursing home residents: Data from the 1995 National Nursing Home Survey. Advance data from vital and health statistics,no. 289. Hyattsville, Maryland: National Center for Health Statistics, 1997.

193 Kinnunen O. Study of constipation in a geriatric hospital, day hospital, old people's home and at home. Aging 1991,3 2 :161-170.

194 Ryan D, Wilson A, Muir TS, Judge TG. The reduction of faecal incontinence by the use of "Duphalac" in geriatric patients. Curr Med Res Opin 1974,2:329-333.

195 Nolan T, Debelle G, Oberklaid F, Coffey C. Randomised trial of laxatives in treatment of childhood encopresis. Lancet 1991,338:523-527.

196 Berg I, Forsythe I, Holt P, Watts J. A controlled trial of 'Senokot' in faecal soiling treated by behavioural methods. J Child Psychol Psychiatry 1983,24:543-549.

197 Nurko S, Garcia-Aranda JA, Worona LB, Zlochisty O. Cisapride for the treatment of constipation in children: A double-blind study. Journal of Pediatrics 2000,136:35-40.

198 Wald A, Chandra R, Gabel S, Chiponis D. Evaluation of biofeedback in childhood encopresis. Journal of Pediatric Gastroenterology and Nutrition 1987,6 4 :554-558.

199 Loening-Baucke V. Biofeedback treatment for chronic constipation and encopresis in childhood: long-term outcome. Pediatrics 1995,96 1 Pt 1 :105-110.

200 van der Plas RN, Benninga MA, Redekop WK, Taminiau JA, Buller HA. Randomised trial of biofeedback training for encopresis. Archives of Disease in Childhood 1996,75 5 :367-374.

201 Miller NE. Biofeedback and visceral learning. Annual Review Psychophysiology 1978,29:373-404.

202 Kohlenberg RJ. Operant conditioning of human anal sphincter pressure. Journal of Applied Behavior Analysis 1973,6:201-208.

203 Kegel AH. Progressive resistance exercise in the functional restoration of the perineal muscles. American Journal of Obstetrics & Gynecology 1948,56:238-248.

204 Engel BT, Nikoomanesh P, Schuster MM. Operant conditioning of rectosphincteric responses in the treatment of faecal incontinence. New England Journal of Medicine 1974,290:646-649.

205 Cerulli MA, Nikoomanesh P, Schuster MM. Progress in biofeedback conditioning for faecal incontinence. Gastroenterology 1979,76:742-746.

206 Wald A. Biofeedback therapy for faecal incontinence. Annals of Internal Medicine 1981,95:146-149.

207 Goldenberg DA, Hodges K, Hersh T, Jinich H. Biofeedback therapy for faecal incontinence. Am J Gastroenterol 1980,74 4 :342-345.

208 Berti Riboli E, Frascio M, Pitto G, Reboa G, Zanolla R. Biofeedback conditioning for faecal incontinence. Archives of Physical & Medical Rehabilitation 1988,69:29-31.

209 Rao SS, Welcher KD, Happel J. Can biofeedback therapy improve anorectal function in fecal incontinence? Am J Gastroenterol 1996,91 11 :2360-2366.

210 Glia A, Gylin M, Akerlund JE, Lindfors U, Lindberg G. Biofeedback training in patients with fecal incontinence. Dis Colon Rectum 1998,41 3 :359-364.

211 Guillemot F, Bouche B, Gower-Rousseau C, Chartier M, Wolschies E, Lamblin MD et al. Biofeedback for the treatment of fecal incontinence. Long-term clinical results. Dis Colon Rectum 1995,38 4 :393-397.

212 Whitehead WE, Orr WC, Engel BT, Schuster MM. External anal sphincter response to rectal distension: learned response or reflex. Psychophysiology 1981,19 1 :57-62.

213 MacLeod JH. Management of anal incontinence by biofeedback. Gastroenterology 1987,93:291-294.

214 Patankar SK, Ferrara A, Larach SW, Williamson PR, Perozo SE, Levy JR et al. Electromyographic assessment of biofeedback training for faecal incontinence and chronic constipation. Dis Colon Rectum 1997,40:907-911.

215 van Tets WF, Kuijpers JH, Bleijenberg G. Biofeedback treatment is ineffective in neurogenic fecal incontinence. Dis Colon Rectum 1996,39 9 :992-994.

216 Rieger NA, Wattchow DA, Sarre RG, Cooper SJ, Rich CA, Saccone GTP et al. Prospective trial of pelvic floor retraining in patients with faecal incontinence. Dis Colon Rectum 1997,40:821-826.

217 Ryn A-K, Morren GL, Hallbook O, Sjodahl R. Long-term results of electromyographic biofeedback training for faecal incontinence. Dis Colon Rectum 2000,43:1262-1266.

218 Jensen LL, Lowry AC. Biofeedback improves functional outcome after sphincteroplasty. Dis Colon Rectum 1997,40:197-200.

219 Jensen LL, Lowry AC. Biofeedback: a viable treatment option for anal incontinence. Dis Colon Rectum 1991,34 supplement :P6.

220 Buser WD, Miner PB. Delayed rectal sensation with faecal incontinence. Successful treatment using anorectal manometry. Gastroenterology 1986,91:1186-1191.

221 Miner PB, Donnelly TC, Read NW. Investigation of the mode of

action of biofeedback in the treatment of faecal incontinence. Digestive Diseases & Sciences 1990,35 10 :1291-1298.

222 Chiarioni G, Scattolini C, Bonfante F, Vantini I. Liquid stool incontinence with severe urgency: anorectal function and effective biofeedback treatment. Gut 1993,34:1576-1580.

223 Whitehead WE, Burgio KL, Engel BT. Biofeedback treatment of faecal incontinence in geriatric patients. Journal of the American Geriatrics Society 1985,33:320-324.

224 Oresland T, Fasth S, Nordgren S, Swenson L, Akervall S. Does balloon dilatation and anal sphincter training improve ileo- anal pouch function? International Journal of Colorectal Disaese 1988,3:153-157.

225 Norton C, Kamm MA. Outcome of biofeedback for faecal incontinence. British Journal of Surgery 1999,86:1159-1163.

226 Solomon MJ, Pager CK, Rex J, Roberts R, Manning J. Randomised, controlled trial of biofeedback with anal manometry, transanal ultrasound, or pelvic floor retraining with digital guidance alone in the treatment of mild to moderate fecal incontinence. Dis Colon Rectum 2003,46 6 :703-710.

227 Solomon MJ, Rex J, Eyers AA, Stewart P, Roberts R. Biofeedback for fecal incontinence using transanal ultrasonography: novel approach. Dis Colon Rectum 2000,43 6 :788-792.

228 Tries J, Eisman E. Fecal Incontinence. In: Schwartz MS, Andrasik F, editors. Biofeedback: A Practitioner's Guide. New York: Guilford Press, 2003: 622-645.

229 Workman DE, Cassisi JE, Dougherty MC. Validation of surface EMG as a measure of intravaginal and intra-abdominal activity: implications for biofeedback-assisted Kegel exercises. Psychophysiology 1992,30:88-93.

230 Pinho M, Hoaiw K, Bielecki K, Keighley MR. Assessment of noninvasive intra-anal electromyography to evaluate sphincter function. Dis Colon Rectum 1991,34:69-71.

231 Norton C, Kamm MA. Anal sphincter biofeedback and pelvic floor exercises for faecal incontinence in adults - a systematic review. Alimentary Pharmacology & Therapeutics 2001, 15:1147-1154.

232 Heymen S, Jones KR, Ringel Y, Scarlett Y, Whitehead WE. Biofeedback treatment of fecal incontinence: a critical review. Dis Colon Rectum 2001,44:728-736.

233 Heymen S, Pikarsky AJ, Weiss EG, Vickers D, Nogueras JJ, Wexner S. A prospective randomised trial comparing four biofeedback techniques for patients with faecal incontinence. Colorectal Disease 2000,2 :88-92.

234 Choi JS, Hwang YH, Salum MR, Weiss EG, Pikarsky AJ, Nogueras JJ et al. Outcome and management of patients with large rectoanal intussusception. Am J Gastroenterol 2001.

235 Chiarioni G, Bassotti G, Stranganini S, Vantini I, Whitehead WE, Stegagnini S. Sensory retraining is key to biofeedback therapy for formed stool incontinence. Am J Gastroenterol 2002,97:109-117.

236 Coffey SW, Wilder E, Majsak MJ, Stolove R, Quinn L. The effects of a progressive exercise program with surface electromyographic biofeedback on an adult with fecal incontinence. Physical Therapy 2002,82:798-811.

237 Bouchoucha M, Devroede G, Faye A, Arsac M. Importance of colonic transit evaluation in the management of fecal incontinence. International Journal of Colorectal Disaese 2002,17:412-417.

238 Pager CK, Solomon MJ, Rex J, Roberts RA. Long-term outcomes of pelvic floor exercise and biofeedback treatment for patients with fecal incontinence. Dis Colon Rectum 2002,45:997-1003.

239 Ferrara A, De Jesus S, Gallagher JT, Williamson PR, Larach SW, Pappas D et al. Time-related decay of the benefits of biofeedback therapy. Tech Coloproctol 2001,53:131-135.

240 Fernandez-Fraga X, Azpiroz F, Aparici A, Casaus M, Malagelada JR. Predictors of response to biofeedback treatment in anal incontinence. Dis Colon Rectum 2003,46:1218-1225.

241 Kienle P, Weitz J, Koch M, Benner A, Herfath C, Schmidt J. Biofeedback versus electrostimulation in treatment of anal sphincter insufficiency. Digestive Diseases & Sciences 2003,48 8:1607-1613.

242 Martinez-Puente MM, Pascual-Montero JA, Garcia-Olmo D. Customized biofeedback therapy improves results in fecal incontinence. International Journal of Colorectal Disaese 2003.

243 Munoz Yague T, Alvarez Sanchez V, Ibanez Pinto A, Solis-Herruzo JA. Clinical, anorectal manometry and surface electromyography in the study of patients with fecal incontinence. Rev Esp Enferm Dig 2003,95:629-634.

244 Pucciani F, Iozzi L, Masi A, Cianchi F, Cortesini C. Multimodal rehabilitation for faecal incontinence: experience of an Italian centre devoted to faecal disorder rehabilitation. Tech Coloproctol 2003,7:139-147.

245 Beddy P, Neary P, Eguare EI, McCollum R, Crosbie J, Conlon KC et al. Electromyographic biofeedback can improve subjective and objective measures of fecal incontinence in the short term. Journal of Gastrointestinal Surgery 2004,8:64-72.

246 Fynes MM, Marshall K, Cassidy M, Behan M, Walsh D, O'Connell PR et al. A prospective, randomized study comparing the effect of augmented biofeedback with sensory biofeedback alone on fecal incontinence after obstetric trauma. Dis Colon Rectum 1999,42 6 :753-758.

247 Sander P, Bjarnesen J, Mouritsen L, Fuglsang-Frederiksen A. Anal incontinence after obstetric third- /fourth-degree laceration. One-year follow-up after pelvic floor exercises. International Urogynecology Journal & Pelvic Floor Dysfunction 1999,10 3 :177-181.

248 Hwang YH, Choi JS, Nam YS, Salum M, Weiss EG, Nogueras JJ et al. Biofeedback therapy after perineal rectosigmoidectomy or J pouch operation. Colorectal Disease 1999,1 Suppl 1 :54.

249 Ho YH, Chiang JM, Tan M, Low JY. Biofeedback therapy for excessive stool frequency and incontinence following anterior resection or total colectomy. Dis Colon Rectum 1996,39 11:1289-1292.

250 Wiesel PH, Norton C, Roy AJ, Storrie JB, Bowers J, Kamm MA. Gut focused behavioural treatmentbiofeedbackfor constipation and faecal incontinence in multiple sclerosis. Journal of Neurology, Neurosurgery & Psychiatry 2000,69 2 :240-243.

251 Enck P, Daublin G, Lubke HJ, Strohmeyer G. Long-term efficacy of biofeedback training for faecal incontinence. Dis Colon Rectum 1994,37:997-1001.

252 Fox J, Sylvestre L, Freeman JB. Rectal incontinence: a team approach. Physiotherapy 1991,77 10 :665-672.

253 Hamalainen KJ, Raivio P, Antila S, Palmu A, Mecklin JP. Biofeedback therapy in rectal incontinence, long-term clinical results. Dis Colon Rectum 1995,38:393-397.

254 Ko CY, Tong J, Lehman RE, Shelton AA, Schrock TR, Welton ML. Biofeedback is effective therapy for fecal incontinence and constipation. Arch Surg 1997,132 8 :829-833.

255 McIntosh LJ, Frahm JD, Mallett VT, Richardson DA. Pelvic floor rehabilitation in the treatment of incontinence. Journal of Reproductive Medicine 1993,38:662-666.

256 Nicastro A, La Torre NA. Faecal incontinence: non surgical therapies rehabilitation in colonproctology. International Journal of the Proctological and Perineal Diseases 1997,1 1 :165-169.

257 Patankar SK, Ferrara A, Levy JR, Larach SW, Williamson PR, Perozo SE. Biofeedback in colorectal practice. Dis Colon Rectum 1997,40:827-831.

258 Schmidbaur W, Barnert J, Wienbeck M. Anal incontinence: evaluation and biofeedback therapy. Materia Medica Polona 1992,24 3 :181-184.

259 Latimer PR, Campbell D, Kasperski J. A components analysis of biofeedback in the treatment of faecal incontinence. Biofeedback and Self-Regulation 1984,9 3 :311-324.

260 Tries J. Protocol- and therapist-related variables affecting outcomes of behavioral interventions for urinary and fecal incontinence. Gastroenterology 2004,126 S152 :S158.

261 Norton C, Hosker G, Brazzelli M. Biofeedback and/or sphincter exercises for the treatment of faecal incontinence in adultsCochrane review . The Cochrane Library, John Wiley & Sons Ltd, Chichester, UK 2002,Issue 2, 2004.

262 McHugh S, Walma K, Diamant NE. Faecal incontinence: a controlled trial of biofeedback. Gastroenterology 1986,90:1545.

263 Riboli EB, Frascio M, Pitto G, Reboa G, Zanolla R. Biofeedback conditioning for faecal incontinence. Archives of Physical & Medical Rehabilitation 1988,69:29-31.

264 McHugh S, Kersey K, Diamant NE. Biofeedback training for faecal incontinence: outcome according to physiological parameters. Gastroenterology 1988,94:A295.

265 Magrini P, Pallotta L, Koch M, Capurso L. Manometric biofeedback in the management of faecal incontinence and pelvic floor dyssynergia. International Journal of the Proctological and Perineal Diseases 1997,1 1 :269-270.

266 Loening-Baucke V. Efficacy of biofeedback training in improving faecal incontinence and anorectal physiologic function. Gut 1990,31:1395-1402.

267 Leroi AM, Dorival MP, Lecouturier MF, Saiter C, Welter ML, Touchais et al. Pudendal neuropathy and severity of incontinence but not presence of an anal sphincter defect may determine the response to biofeedback therapy in fecal incontinence. Dis Colon Rectum 1999,42 6 :762-769.

268 Marcello PW, Barrett RC, Coller JA, Schoetz DJ, Roberts PL, Murray JJ et al. Fatigue rate index as a new measure of external sphincter function. Dis Colon Rectum 1998,41:336-343.

269 Sangwan YP, Coller JA, Barrett RC, Roberts PL, Murray JJ, Schoetz DJ, Jr. Can manometric parameters predict response to biofeedback therapy in fecal incontinence? Dis Colon Rectum 1995,38 10 :1021-1025.

270 Enck P. Biofeedback training in disordered defecation. A critical review. Digestive Diseases & Sciences 1993,38 11 :1953-1960.

271 Fernandez-Fraga X, Azpiroz F, Malagelada JR. Significance of pelvic floor muscles in anal incontinence. Gastroenterology 2002,1234:1441-1450.

272 Stiener SS, Dince WM. Biofeedback efficacy studies: a critique of critiques. Biofeedback & Self-Regulation 1981,6:275-288.

273 Caldwell KPS. The electrical control of sphincter incompetence. Lancet 1963,ii:174-175.

274 Haskell B, Rovner H. Electromyography in the management of the incompetent anal sphincter. Unknown 1967, 81-84.

275 Schussler B, Laycock J, Norton P, Stanton S. Pelvic floor re-education: principles and practice. London: Springer-Verlag, 1994.

276 Fall M, Lindtstrom S. Electrical stimulation: a physiologic approach to the treatment of incontinence. Urol Clin 1991, 18:393-407.

277 Hobday DI. A study of the cortical processing of ano-rectal sensation using functional MRI. Brain 2001,124 2 :361-368.

278 Salvioli B. Rectal compliance,capacity,and rectoanal sensation in fecal incontinence. Am J Gastroenterol 2001,96 7 :2158-2168.

279 Hamdy S. Spinal and pudendal nerve modulation of human corticoanal motor pathways. Am J Physiol Gastrointest Livr Physiol 1998,274 2 :G419-G423.

280 Peticca L. Combined biofeedback,physiotherapy and electrostimulation for fecal incontinence. Tech Coloproctol 2000,4:157-161.

281 Baker L, Wederich CL, McNeal DR, Newsam C, Waters RL. Neuromuscular Electrical Stimulation. Los Amigos Research & Education Institute Inc, 2000.

282 Larpent JL, Cuer JC, Da Poigny M. Clinical and manometric results of electrical stimulation in patients with anal incontinence. Coloproctology 1987,3:183-184.

283 Leroi AM, Karoui S, Touchais JY, Berkelmans I, Denis P. Electrostimulation is not a clinically effective treatment of anal incontinence. Eur J Gastroenterol Hepatol 1999,11 9 :1045-1047.

284 Scheuer M, Kuijpers HC, Bleijenberg G. Effect of electrostimulation on sphincter function in neurogenic fecal continence. Dis Colon Rectum 1994,37 6 :590-593.

285 Sylvester KL, Keilty SEJ. A pilot study to investigate the use of interferential in the treatment of ano-rectal incontinence. Physiotherapy 1987,73 4 :207-208.

286 Hosker G, Norton C, Brazzelli M. Electrical stimulation for faecal incontinence in adultsCochrane review . The Cochrane Library, John Wiley & Sons Ltd, Chichester, UK 2002,Issue 2, 2004.

287 Gladman A, Scott SM, Chan CLH, Willis NS, Lunniss PJ. Rectal hyposensitivity is common in patients with constipation and/or fecal incontinence and may thus be important in the etiology of such conditions. Dis Colon Rectum 2003,46 2 .