

## Risk factors in acquired faecal incontinence

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### SUMMARY

Acquired faecal incontinence arising in the non-elderly population is a common and often devastating condition. We conducted a retrospective cohort analysis in 629 patients (475 female) referred to a tertiary centre, to determine the relative importance of individual risk factors in the development of faecal incontinence, as demonstrated by abnormal results on physiological testing.

Potential risk factors were identified in all but 6% of patients (7 female, 32 male). In women, the principal risk factor was childbirth (91%), and in most cases at least one vaginal delivery had met with complications such as perineal injury or the need for forceps delivery. Of the males, half had undergone anal surgery and this was the only identified risk factor in 59%. In many instances, assignment of cause was hampered by a long interval between the supposed precipitating event and the development of symptoms. Abnormalities of anorectal physiology were identified in 76% of males and 96% of females (in whom they were more commonly multiple).

These findings add to evidence that occult damage to the continence mechanism, especially through vaginal delivery and anal surgery, can result in subsequent faecal incontinence, sometimes after an interval of many years.

### INTRODUCTION

Faecal incontinence, the involuntary loss of rectal contents at a socially inappropriate time or place, is an under-appreciated condition<sup>1</sup> which affects at least 2% of adults in the community.<sup>2</sup> The prevalence in elderly people is up to 15%, and higher still among those living in residential or nursing homes.<sup>3</sup> However, by comparison with urinary incontinence<sup>4,5</sup> the condition suffers neglect. This is surprising, given that the prevalence of the two conditions is similar, they frequently coexist,<sup>6</sup> and they may have common aetiologies;<sup>7,8</sup> moreover, the physical, psychological and social incapacitation related to faecal incontinence may be greater.<sup>8</sup>

Patients seeking help can now be referred to specialist units for comprehensive investigations of anorectal function, in the hope that an understanding of the individual pathophysiology will allow specific rather than empirical management. The results of interventions, however, whether conservative or surgical, are commonly disappointing. Consequently, we need to identify factors in the

histories of these patients that might allow preventive strategies.

Most individuals become faecally incontinent as a result of some form of insult—for example, obstetric trauma, anal surgery, neurological disease, pelvic surgery.<sup>2,8–13</sup> In some cases, the cause–effect relation is clear, in that a temporal relation is evident, the sufferer ascribes onset of symptoms to the event (e.g. 5–13% incidence of faecal incontinence after vaginal delivery in primiparous women<sup>14,15</sup>), and the pathophysiology is demonstrable on anorectal function testing.<sup>15</sup> Symptoms, however, may not develop until many years after the event,<sup>16</sup> and the relation between cause and effect may then be unclear. It is known that the incidence of occult anal sphincter damage following vaginal delivery (even those deemed ‘uneventful’) is much higher than the incidence of immediate post-partum incontinence,<sup>17</sup> and that unsuspected anal sphincter defects occur following various ‘minor’ anal surgical procedures.<sup>9</sup> Such pathophysiology provides the potential for subsequent development of incontinence in combination with other factors such as ageing.<sup>18</sup> Unfortunately, there have been no large and long-term prospective studies addressing eventual functional outcome. By performing a retrospective analysis of a large series of patients referred consecutively for investigation of faecal incontinence, we aimed to determine: the relative importance of individual proposed risk factors; the proportions of patients in whom the cause–effect relation was clear or unclear; and, in those patients

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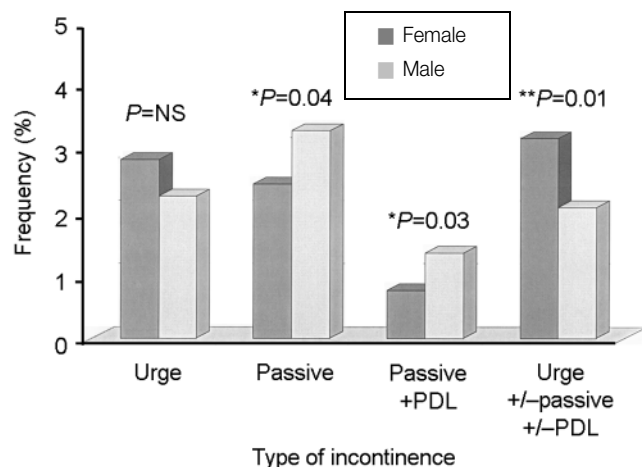


Figure 1 Prevalence of differing types of incontinence in female and male patients. PDL=post-defaecation leakage.

where the cause-effect relationship was unclear, whether an anorectal physiology was demonstrably abnormal.

**PATIENTS AND METHODS**

The study group consisted of 629 patients (475 females [median age 53, range 15–88], 154 males [median age 53, range 14–92]) referred to a tertiary centre for physiological assessment of their faecal incontinence, between January 1995 and June 2002, in whom records were complete. Each patient had undergone appropriate assessment to exclude organic disease before referral. Faecal incontinence was diagnosed in accordance with the Rome II diagnostic criteria for functional anal disorders,<sup>19</sup> and classified according to symptom profile:<sup>20,21</sup> passive faecal incontinence was defined as incontinence without the patients’ knowledge; post-defaecation leakage was defined as passive incontinence temporally related to defaecation; urge faecal incontinence was defined as incontinence occurring with the patients’ awareness, but against their will because of lack of voluntary control. All patients were evaluated by a thorough medical history and full anorectal physiological assessment. Collection of both clinical and physiological

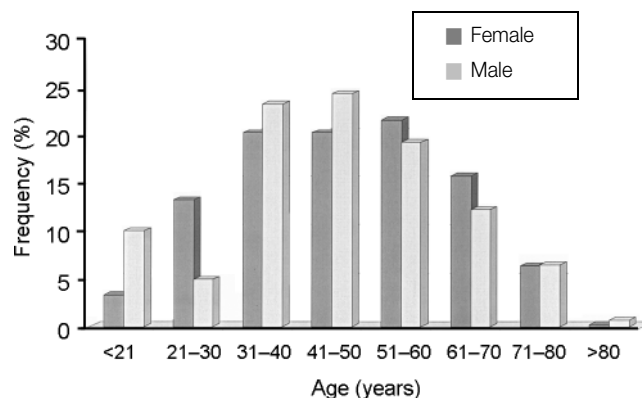


Figure 2 Age at onset of symptoms

data was standardized between different investigators within the department. Medical histories were scrutinized for the presence of four previously proposed risk factors<sup>2,8–13</sup> (obstetric events, anal surgery, pelvic surgery, neurological factors), either individually or in combination, the age of onset of symptoms and their duration, and whether the patient ascribed the symptoms to a particular event.

Measurements of anal pressures, rectal sensitivity, pudendal nerve terminal motor latencies, and anal sphincter integrity, with comparison with our normal ranges for each, as previously described,<sup>21,22</sup> allowed physiological findings to be classified as anatomical<sup>22</sup> (e.g. internal or external anal sphincter disruption), neurological (predominantly pudendal nerve motor dysfunction),<sup>22,23</sup> due to a sensory disturbance of the anorectum,<sup>21,24</sup> or a combination of these. Incontinence in the absence of any abnormal results was classified as idiopathic.

**Statistical analysis**

Data were expressed for grouped results as median and range. Fisher’s exact test was used to analyse contingency tables (Prism 3.02; Graph Pad Software Inc., San Diego, California, USA). A P value of <0.05 was taken as significant.

**RESULTS**

Of the 629 patients, 168 (27%) complained only of passive faecal incontinence, 170 (27%) only of urge faecal incontinence, and 43 (7%) only of post-defaecation leakage (Figure 1). Age at onset (females: median 47 years, range 0–88 years; males: 47, range 0–92) (Figure 2), and duration of symptoms (females: 26 months, range 2–502; males: 36 months, range 11–480) were similar for the two genders. 72% of patients developed symptoms before the age of 60.

**Clinical history**

Past clinical events of potential relevance had been recorded in 590 patients (94%) (Table 1). Only 7 females (1%) but 32 males (21%) had histories that contained no volunteered potential risk factor.

The overwhelming risk factor in females was childbirth (91%), with at least one vaginal delivery reported as complicated in 338 (78%). Complications relating to delivery included: perineal trauma (episiotomy/tear) in 259, the use of forceps in 107, and Ventouse extraction in 12. In 290 of these 338 parous females (86%), the complication occurred during the first delivery.

58% of females had also undergone pelvic surgery, most commonly hysterectomy (153), and 90 (19%) had undergone anal surgery. The frequency of multiple risk factors in females (reflecting the high prevalence of childbirth) was

**Table 1 Potential risk factors identified from patient histories**

<b>Risk factors</b>	<b>Female*</b>	<b>Male*</b>
Obstetric	<b>91</b>	—
Complicated	78	—
Perineal tear	77	—
Episiotomy	36	—
Forceps	32	—
Ventouse	4	—
Uncomplicated	18	—
Caesarean section	4	—
Abdominopelvic surgery	<b>58</b>	<b>23</b>
Hysterectomy	56	—
Ovarian surgery	11	—
Anterior compartment prolapse	7	—
Sterilization	7	—
Laparoscopy	6	—
Appendicectomy	17	28
Cholecystectomy	12	3
Rectal prolapse surgery	9	33
Bowel/anterior resection	3	25
Adhesiolysis	2	0
Anal surgery	<b>19</b>	<b>50</b>
Haemorrhoidectomy	34	35
Banding/sclerotherapy	12	17
Fistula surgery	20	17
Sphincterotomy	19	20
Anal stretch	12	14
Neurological	<b>13</b>	<b>17</b>
Back injury/surgery	53	62
Diabetes	28	31
Multiple sclerosis	10	0
Other	<b>8</b>	<b>23</b>
Gastrointestinal infection	13	17
Medication	5	11
None	<b>1</b>	<b>21</b>

\*Numbers represent percentages. Those in bold relate to the cohort of incontinent males and females; those in normal type the percentage within each risk category

higher than in males (67% versus 30%). A single risk factor was present in 49% of males and 32% of females (Figure 3).

Among the 150 females in whom a single risk factor was present, obstetric factors (complicated vaginal deliveries in 105, uncomplicated in 16) were reported in 124 (82%). 28 females had delivered a single child. The frequencies of anal surgery, abdomino-pelvic surgery and neurological factors as isolated risk factors between the other 26 (nulliparous) females were similar.

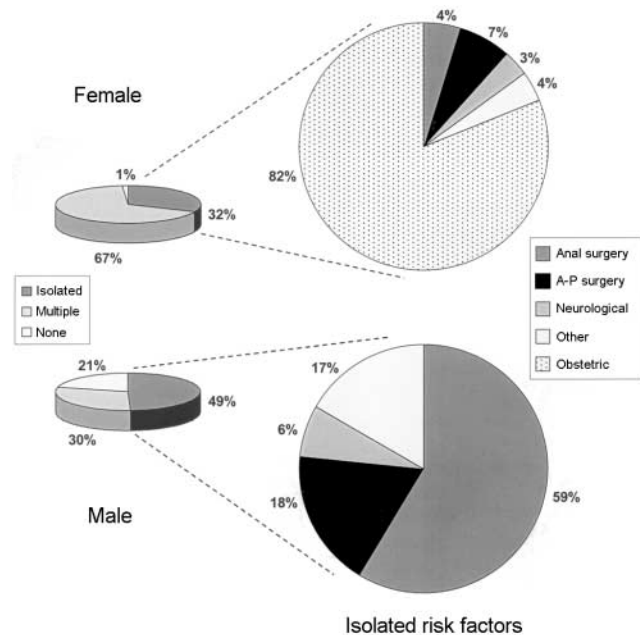
The most commonly reported risk factor in males was anal surgery (50%). In the two sexes, the incidence of anal surgical procedures was almost identical (see Table 1), with haemorrhoidectomy the most frequent procedure reported,

followed by fistula surgery and sphincterotomy for anal fissure. 23% of males had also undergone pelvic surgery, most commonly procedures involving mobilization of the rectum, and appendicectomy.

Of the 76 males in whom a single risk factor was evident, anal surgery was reported in 45 (59%). 13 males (17%) had single risk factors other than the four of major interest, of varying types (e.g. anal assault, pelvic trauma, radiotherapy).

**Cause-effect relations in those with single risk factors**

Overall, 268 patients (43%) ascribed the onset of symptoms to a particular event in their medical histories, more frequently in males than in females (51% versus 40%). In those patients with isolated risk factors, again males more frequently ascribed the onset of symptoms to the particular event (63% versus 39% in females). Only 48 of the 124 females with obstetric factors as a single risk ascribed the onset of their incontinence to this event (Table 2). The median age of onset of symptoms in these patients was 26 years lower than that of the 76 females in whom obstetric factors were the only risk but in whom no association had been made with symptoms. The median time lag before onset of symptoms in such females was 18.5 years (range 2–55 years). There was no difference in duration of symptoms between those who ascribed their incontinence to an obstetric event and those who had not.



**Figure 3 Risk factors identified from patient histories.** The smaller pie charts depict the proportions of male and female patients with isolated, multiple or no risk factors in their histories. The larger pie charts illustrate the relative proportions of isolated risk factors. A-P=abdomino-pelvic

Table 2 Isolated risk factors: patients in whom development of their symptoms was ascribed to a given event are compared with non-ascribers

	Risk factor									
	Obstetric		Anal surgery		A-P surgery		Neuro		Other	
	F	M	F	M	F	M	F	M	F	M
	124	—	7	45	11	13	5	5	6	13
Ascribed (A) <sup>†</sup>	48 (39%)	—	4	33 (73%)	3	6	1	2	4	7
Non-ascribed (NA)	76 (61%)	—	3	12 (27%)	8	7	4	3	2	6
Demonstrable pathophysiology	119 (96%)	—	6	37 (82%)	10	9	5	4	4	12
Age of onset (A) (years)	30.5 <sup>‡</sup> (20–43)	—	—	46 (0–76)						
Age of onset (NA) (years)	56 (27–79)	—	—	38 (0–73)						
Symptom duration (A) (months)	24 (2–502)	—	—	54 (6–372)						
Symptom duration (NA) (months)	24 (2–192)	—	—	36 (3–240)						
Lag to symptoms (NA) (years)	18.5* (2–55)	—	—	7.5 (2–16)						

\* $P=0.02$  first obstetric event in females versus anal surgery in males; <sup>†</sup> $P=0.001$  male versus female; <sup>‡</sup> $P<0.0001$  ascribers versus non-ascribers; A-P=abdomino-pelvic; neuro=neurological

In those males in whom anal surgery was the only risk factor, and in whom no association had been made with subsequent symptoms, the delay between event and onset was 7.5 years, range 2–16. This was significantly shorter than the equivalent lag to symptom onset in females following childbirth ( $P=0.02$ ).

**Physiology**

Overall, the results of physiological investigations were abnormal in 573 patients (91%). The finding of abnormal physiology was more frequent in incontinent females than males (96% versus 76%;  $P=0.0001$ ). Incontinent females were found to have greater than one physiological abnormality more frequently than males (64% versus 32%;  $P=0.0001$ ) (Figure 4). Of the 425 patients who had a structural abnormality of the anal sphincter, females were more likely to have external anal sphincter defects (87% versus 43%, respectively;  $P<0.0001$ ), and males internal anal sphincter defects (57% versus 11%;  $P<0.0001$ ). A purely sensory cause of incontinence was observed more frequently in males.

Of the 432 incontinent parous women, 329 had evidence of structural damage (159 combined external/internal sphincter defects, 139 external sphincter alone, 31 internal sphincter alone). Of the remaining 98 parous females, 79 had resting or squeeze pressures below the normal range (74 reduced squeeze pressure alone, 25 reduced resting pressure alone), 76 showed evidence of

neurogenic injury with or without rectal sensory disturbance, and 8 showed isolated sensory disturbance of the rectum. Of the 124 women in whom childbirth was the only risk factor identified from the clinical history, 95 (77%) had ultrasonographic evidence of sphincter disruption, 24 had a neurogenic and/or sensory abnormality, and the remaining 5 had reduced squeeze or resting pressures on manometry. 6 of the 7 females and 18 of the 32 males with no risk factor for incontinence identified in their histories had demonstrably abnormal anorectal physiology, most commonly solitary abnormalities.

**DISCUSSION**

This study has shown that the overwhelming risk factor for the development of faecal incontinence in women is childbirth, and in males anal surgery. In women incontinence is predominantly combined (passive and urge) whereas in males it is predominantly passive in nature. In the great majority, tests of anorectal function were abnormal, reflecting the nature of the incontinence.<sup>20</sup>

Are these results likely to represent faecal incontinence in the general population? The substantial number of young and middle-aged patients, with an age peak in the fifth decade, may reflect referral patterns. Referrals to the Gastrointestinal Physiology Unit are from secondary rather than primary sources, and the unit is part of a surgical department. It may be, therefore, that some (especially

elderly) patients are not referred on the assumption that they would not be candidates for surgical interventions. The reason for the female predominance, in contrast to findings in some population studies,<sup>2,25</sup> may be that there is a true gender difference in incidence, or that males with symptoms do not seek help, or that males are not referred for specialist investigation. The advantage of this study design over questionnaire-based investigations<sup>2,26</sup> is that the association between event and symptoms is explored further by investigation of anorectal physiology; but a disadvantage, in contrast to prospective studies, is that the attribution of symptoms and demonstrable pathophysiology have to be assumed to relate to the volunteered event.

**Major risk factors in females**

Vaginal childbirth can impair the continence mechanism in several ways,<sup>8,10,15,16,27</sup> reflecting the combined symptoms (urge and passive faecal incontinence) and multiple pathophysiological abnormalities observed in the majority of parous females in this study. Although most of the parous women had experienced a complicated delivery, it is important to note that symptoms may develop after apparently uncomplicated parturition.<sup>16,17</sup>

Of those females with a single risk factor in their histories, nearly 40% ascribed their symptoms to childbirth. Of those who did not ascribe incontinence to delivery, there was a median delay of 18.5 years to onset of symptoms. Pathophysiological abnormalities were evident in all but 4% of these 124 females. This study supports the

findings of Sultan *et al.*, who demonstrated prospectively an incidence of 30% occult anal sphincter defects in women immediately after their first delivery and only a 4% rise in incidence of sphincter disruption consequent upon subsequent deliveries.<sup>15,17</sup> Long-term clinical and physiological follow-up of childbearing women is unfortunately lacking,<sup>27,28</sup> but the results of this study are highly suggestive that covert damage to the continence mechanism does eventually become clinically overt in a proportion, perhaps several decades later, when the aetiology is more likely to be multifactorial.<sup>27</sup>

This study has also demonstrated the relative importance of anal surgery as a risk factor to continence in females,<sup>9</sup> proctological intervention being the commonest cause in those who had not borne children vaginally and those who had had clinically uncomplicated deliveries.

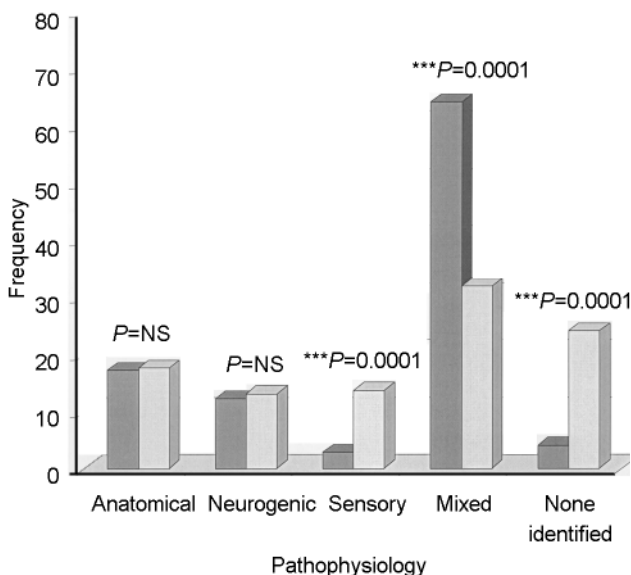
**Major risk factors in males**

In incontinent males, usually a single risk factor was volunteered, the commonest being anal surgery. In such procedures it is primarily the internal (rather than external) anal sphincter that is susceptible to disruption, either deliberately (e.g. lateral sphincterotomy) or as a complication (e.g. haemorrhoidectomy).<sup>9</sup> This is consistent with the predominance of passive faecal incontinence in association with isolated internal anal sphincter defects observed in the majority of males.

It must be stressed that, in contrast to females, one-fifth of the males had volunteered no risk factor, and that in nearly one-quarter no abnormality was observed on physiological testing. An apparent lack of risk factors may be due to inadequate history taking (for example, they were not asked about the practice of anoreceptive intercourse<sup>29</sup> or a history of abuse in childhood, although 8 males with no history of anal surgery had internal sphincter defects). Similarly, physiological testing may not have been sufficiently comprehensive: the complex mechanics of continence involve also colonic transit and rectal evacuation,<sup>30</sup> which was not measured.

**Other considerations**

The association between neurological disorders (thoracolumbar spinal trauma or surgery, diabetes mellitus, etc.) and disturbed continence is well documented.<sup>10,13,31,32</sup> No prospective studies have been conducted on the long-term effects of pelvic surgery, especially hysterectomy, on faecal continence; however, studies of such interventions on urinary function,<sup>33</sup> the recent identification of autonomic nerves in the supporting ligaments of the uterus<sup>34</sup> and the results of the present study, lend weight to the possibility that pelvic interventions are causally associated with faecal incontinence.



**Figure 4 Pathophysiological basis for faecal incontinence, as revealed by objective anorectal physiological testing.** 'Mixed' represents any combination of the other three physiological abnormalities (anatomical; sensory; neurogenic). 'None identified' signifies that none of the three other pathophysiologicals were demonstrated; however, 41% of these patients had reduced anal pressures on manometry. ■ Female; □ male

### Clinical implications for the promotion of continence

The degree of individual suffering rendered by symptoms and the inability of current practice to restore to the patient completely normal function mean that, in obstetric, gynaecological and colorectal practice, full consideration must be given to avoidance of those interventions known to be potentially injurious. At present, for example, both obstetricians and pelvic surgeons may underrate the risks of certain procedures because faecal incontinence, when it develops, tends to be dealt with by gastroenterologists and proctological surgeons. The excess incidence of incontinence after obstetric interventions, especially forceps delivery, should not be used to justify an increase in the use of caesarean section;<sup>35</sup> nevertheless, obstetricians should consider the risks of sphincter damage when advising patients. Furthermore, the general surgeons who contemplate doing a haemorrhoidectomy or other supposedly 'minor' procedure should consider whether the patient might better be referred to a specialist.

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### REFERENCES

- 1 Leigh RJ, Turnberg LA. Faecal incontinence: the unvoiced symptom. *Lancet* 1982;**i**:1349-51
- 2 Walter S, Hallbook O, Gotthard R, Bergmark M, Sjodahl R. A population-based study on bowel habits in a Swedish community: prevalence of faecal incontinence and constipation. *Scand J Gastroenterol* 2002;**37**:911-16
- 3 Peet SM, Castleden CM, McGrother CW. Prevalence of urinary and faecal incontinence in hospitals and residential and nursing homes for older people. *BMJ* 1995;**311**:1063-4
- 4 Perry S, Shaw C, Assassa P, et al. An epidemiological study to establish the prevalence of urinary symptoms and felt need in the community: the Leicestershire MRC Incontinence Study. Leicestershire MRC Incontinence Study Team. *J Publ Health Med* 2000;**22**:427-34
- 5 Department of Health. *Good Practice in Continence Services*. London: DoH, 2000
- 6 Meschia M, Buonaguidi A, Pifarotti P, Somigliana E, Spennacchio M, Amicarelli F. Prevalence of anal incontinence in women with symptoms of urinary incontinence and genital prolapse. *Obstet Gynecol* 2002;**100**:719-23
- 7 Brubaker L. Postpartum urinary incontinence. *BMJ* 2002;**324**:1227-8
- 8 O'Boyle AL, Davis GD, Calhoun BC. Informed consent with birth: protecting the pelvic floor and ourselves. *Am J Obstet Gynecol* 2002;**187**:981-3
- 9 Felt-Bersma RJ, van Baren R, Koorevaar M, Strijers RL, Cuesta MA. Unsuspected sphincter defects shown by anal endosonography after anorectal surgery. A prospective study. *Dis Colon Rectum* 1995;**38**:249-53
- 10 Kamm MA. Faecal incontinence. *BMJ* 1998;**316**:528-32
- 11 Sood AK, Nygaard I, Shahin MS, Sorosky JI, Lutgendorf SK, Rao SS. Anorectal dysfunction after surgical treatment for cervical cancer. *J Am Coll Surg* 2002;**195**:513-19
- 12 Ho YH, Low D, Goh HS. Bowel function survey after segmental colorectal resections. *Dis Colon Rectum* 1996;**39**:307-10
- 13 Jorge JM, Wexner SD. Etiology and management of fecal incontinence. *Dis Colon Rectum* 1993;**36**:77-97
- 14 Chaliha C, Kalia V, Stanton SL, Monga A, Sultan AH. Antenatal prediction of postpartum urinary and fecal incontinence. *Obstet Gynecol* 1999;**94**:689-94
- 15 Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. *N Engl J Med* 1993;**329**:1905-11
- 16 Fitzpatrick M, O'Herlihy C. The effects of labour and delivery on the pelvic floor. *Best Pract Res Clin Obstet Gynaecol* 2001;**15**:63-79
- 17 Sultan AH, Stanton SL. Occult obstetric trauma and anal incontinence. *Eur J Gastroenterol Hepatol* 1997;**9**:423-7
- 18 Jameson JS, Chia YW, Kamm MA, Speakman CT, Chye YH, Henry MM. Effect of age, sex and parity on anorectal function. *Br J Surg* 1994;**81**:1689-92
- 19 Whitehead WE, Wald A, Diamant NE, Enck P, Pemberton JH, Rao SS. Functional disorders of the anus and rectum. *Gut* 1999;**45**(suppl. 2):1155-9
- 20 Engel AF, Kamm MA, Bartram CI, Nicholls RJ. Relationship of symptoms in faecal incontinence to specific sphincter abnormalities. *Int J Colorectal Dis* 1995;**10**:152-5
- 21 Gladman MA, Scott SM, Chan CL, Williams NS, Lunniss PJ. Rectal hyposensitivity: prevalence and clinical impact in patients with intractable constipation and fecal incontinence. *Dis Colon Rectum* 2003;**46**:238-46
- 22 Buchanan GN, Nicholls T, Solanki D, Kamm MA. Investigation of faecal incontinence. *Hosp Med* 2001;**62**:533-7
- 23 Azpiroz F, Enck P, Whitehead WE. Anorectal functional testing: review of collective experience. *Am J Gastroenterol* 2002;**97**:232-40
- 24 Chan CL, Facer P, Davis JB, et al. Sensory fibres expressing capsaicin receptor TRPV1 in patients with rectal hypersensitivity and faecal urgency. *Lancet* 2003;**361**:385-91
- 25 Perry S, Shaw C, McGrother C, et al. Prevalence of faecal incontinence in adults aged 40 years or more living in the community. *Gut* 2002;**50**:480-4
- 26 Nygaard IE, Rao SS, Dawson JD. Anal incontinence after anal sphincter disruption: a 30-year retrospective cohort study. *Obstet Gynecol* 1997;**89**:896-901
- 27 Chaliha C, Sultan AH, Bland JM, Monga AK, Stanton SL. Anal function: effect of pregnancy and delivery. *Am J Obstet Gynecol* 2001;**185**:427-32
- 28 MacArthur C, Glazener CM, Wilson PD, Herbison GP, Gee H, Lang GD, et al. Obstetric practice and faecal incontinence three months after delivery. *Br J Obstet Gynaecol* 2001;**108**:678-83
- 29 Miles AJ, Allen-Mersh TG, Wastell C. Effect of anoreceptive intercourse on anorectal function. *J R Soc Med* 1993;**86**:144-7
- 30 Sagar PM, Pemberton JH. Anorectal and pelvic floor function. Relevance of continence, incontinence, and constipation. *Gastroenterol Clin N Am* 1996;**25**:163-82
- 31 Longo WE, Ballantyne GH, Modlin IM. The colon, anorectum, and spinal cord patient. A review of the functional alterations of the denervated hindgut. *Dis Colon Rectum* 1989;**32**:261-7
- 32 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. *Arch Intern Med* 2001;**161**:1989-96
- 33 Brown JS, Sawaya G, Thom DH, Grady D. Hysterectomy and urinary incontinence: a systematic review. *Lancet* 2000;**356**:535-9
- 34 Butler-Manuel SA, Buttery LD, A'Hern RP, Polak JM, Barton DP. Pelvic nerve plexus trauma at radical hysterectomy and simple hysterectomy: the nerve content of the uterine supporting ligaments. *Cancer* 2000;**89**:834-41
- 35 Rortveit G, Daltveit AK, Hannestad YS, Hunskaar S, Norwegian Epidemiology of Incontinence in the County of Nord-Trøndelag Study. Urinary incontinence after vaginal delivery or cesarean section. *N Engl J Med* 2003;**348**:900-7