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Asymmetric Sphincter Innervation is Associated With Fecal Incontinence After Anal Sphincter Trauma During Childbirth

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Aims: Functional asymmetry of pelvic floor innervation has been shown to exist in healthy subjects, and has been proposed to be a predictor of increased risk for fecal incontinence in case of trauma. However, this remains to be shown for different clinical conditions such as traumatic childbirth. **Methods:** A conventional surface EMG system was used to assess the innervation of the external anal sphincter. A symmetry index was used to define the relative EMG amplitude asymmetry of the external anal sphincter between 0 (symmetric) and 1 (asymmetric). Three cohorts were studied: 40 nulliparous women in the third trimester (Study 1), 15 primiparous women within 6 months following vaginal delivery without clinically apparent anal sphincter trauma (Study 2), and 50 women after childbirth-related third or fourth degree perineal tear 6–12 months postpartum (Study 3). Furthermore, all women underwent conventional anorectal manometry. **Results:** Sixteen or forty nulliparous women reported signs of fecal incontinence; however, relative asymmetry was not correlated to symptom severity ($P = 0.345$), and not to manometric measures (Study 1). In Study 2, Women who had suffered clinically apparent anal sphincter trauma ($P = 0.07$) tended to have a stronger association between incontinence and asymmetry. In Study 3, 19/50 women reported moderate to severe incontinence. Asymmetry and symptom severity were significantly correlated ($P < 0.001$). Patients with incontinence had a significantly higher asymmetry score than their continent counterparts. **Conclusion:** Functional asymmetry of anal sphincter innervation is significantly associated with incontinence symptoms, but only after childbirth-related sphincter injuries and therefore, should be regarded as an additional risk factor. *Neurourol. Urodynam. 26:134–139, 2007.* © 2006 Wiley-Liss, Inc.

Key words: anal incontinence; asymmetry; birth trauma; innervation; perineal laceration; vaginal delivery

INTRODUCTION

Fecal incontinence in woman is known to be related to childbirth. The reported frequency of incontinence of stool in primiparous women ranges from 2 to 6% [MacArthur et al., 1997; Donnelly et al., 1998], and incontinence for either stool or flatus from 13 to 25% [Zetterstrom et al., 1999; Signorello et al., 2000]. After severe perineal laceration, the rate of anal incontinence rises to between 17 and 62% [Crawford et al., 1993; Sultan et al., 1993; Eason et al., 2002].

The association of incontinence symptoms with specific obstetric factors is less well established. There is increasing evidence that instrumental delivery is a risk factor for the occurrence of muscle defects in the internal and external anal sphincter (IAS resp. EAS), depending on the type of instrument used—for example, forceps or vacuum extraction [Sultan et al., 1993; MacArthur et al., 1997]—or interventions such as an episiotomy [Coats et al., 1980; Sultan et al., 1993]. It could be shown that postpartal fecal incontinence was significantly more likely to be associated with instrumental delivery than with spontaneous vaginal delivery [MacArthur et al., 1997].

Functional asymmetry of pelvic floor innervation has been shown to exist in healthy subjects [Hamdy et al., 1999], and was demonstrated for the central and peripheral compartments of the motor pathways to the pelvic floor [Matzel et al., 1999; Turnbull et al., 1999]. It has been proposed to be a predictor of increased risk for fecal incontinence in case of trauma

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[Enck et al., 2004], but this remains to be shown for clinical conditions such as traumatic childbirth. In a large unselected cohort of patients with fecal incontinence, it was recently shown that asymmetric sphincter innervation is associated with decreased function of the external anal sphincter [Franz et al., 2005].

In patients undergoing sacral rhizotomy however, it was shown intra-operatively that the distribution of pudendal afferent fibers was asymmetrical with respect to level (S2–S4) and side (left, right) in the majority of cases and was furthermore confined to a single level in 18% or even to a single root in 8% of cases [Deletis et al., 1992; Huang et al., 1997]. Similarly, Matzel et al. [1999] showed intra-operatively in adult continent patients undergoing sacral pacemaker implantation for hypotonic bladder treatment, that approximately 20% showed asymmetry with respect to anal pudendal motor pathways. In these cases, saving of the dominant sensory root from rhizotomy or implantation of the pacemaker on the dominant motor pathways clearly demonstrated clinical relevance by reducing the risk of postoperative micturition disorders on the one hand, and improving the overall benefit of the procedure on the other.

With the present study, we wished to determine as to whether functional asymmetry of pelvic floor innervation can be found in continent and incontinent women prior and post-childbirth, both with and without pelvic floor trauma, as well as to investigate its association with symptoms of fecal incontinence.

METHODS

The studies were conducted as part of research collaboration between the Departments of General Surgery and Gynecology of the University Hospital Tübingen, Germany between January 1999 and December 2000. The study protocols had previously been approved by the Ethical Committee of the Medical Faculty to be in accordance with the Declaration of Helsinki. All patients and volunteer subjects gave written informed consent prior to the investigations.

Three cohorts of female patients were studied:

Group I

Forty pregnant women volunteers during their 29–39 gestational week without a previous vaginal birth (mean age ± SD: 34.5 ± 4.2 years, range: 25–44 years). Five women had previously had a delivery by elective cesarean section. These volunteers were recruited with the aid of a physical therapist during prenatal pelvic floor training.

Group II

Fifteen women (35.4 ± 3.5 years, range: 30–40 years) after vaginal delivery recruited from Group 1, who were, upon request, willing to participate in a second investigation between 3 and 6 weeks postpartum.

Group III

Consecutive nulliparous women (or primiparous women after a first elective cesarean section delivery) who experienced a perineal tear of third or fourth degree during vaginal delivery at the maternity unit of the University Hospital, Tübingen. All were sent an information letter regarding the aims of this study. Of 91 women contacted between July 1999 and June 2000, 50 (55%) women (31.9 ± 4.3 years, range: 18–40) agreed to participate in the study.

Clinical Assessment

Prior to further diagnostic and technical procedures, all women completed a standardized questionnaire with questions regarding the obstetric and medical history, general health, daily defecatory patterns and complaints of flatus incontinence, fecal soiling, fecal and urinary incontinence, and/or urgency as well as perineal pain and sexual dysfunction [Franz et al., 1998]. Urinary stress incontinence was graded according to international standards (Ingelman-Sundberg) into grade I (incontinence while coughing), grade II (incontinence during lifting and carrying weight), and grade III (incontinence while standing) [Schussler and Alloussi, 1983]. The women were also asked whether they had experienced urinary and/or fecal incontinence before or during pregnancy or after delivery. All respective urinary and fecal incontinence symptoms were added and scored based on severity.

Perineal trauma was determined according to Clinical Green Top Guidelines [Sultan, 1999], and was classified into four groups: first degree lacerations involved sutured injury to the perineal or vaginal skin but not the underlying muscle; second-degree injury involved, in addition, fascia and perineal muscles but not the anal sphincter. A third degree tear was defined as partial or complete rupture of the anal sphincter complex (EAS and IAS) with intact anal mucosa, and a fourth-degree tear as complete rupture of the anal sphincters and of the anorectal mucosa [Sultan, 1999].

None of the women reported a history of previous pelvic floor surgery, nor known inflammatory or other bowel or bladder diseases.

Surface EMG

Left and right surface EMG (S-EMG) recording by means of a modified anal plug electrode with one pair of electrodes located on each side of the probe and positioned in the left and right side of the anal canal, and a conventional mass EMG system (Pelvimeter, Standard Instruments, Karlsruhe, Germany) (s. Fig. 1) was used to non-invasively assess the overall innervation of the external anal sphincter—and eventually of the levator ani muscles such as the puborectalis muscle that is in close proximity—during maximal voluntary anal sphincter contraction for 10 sec. The peak value of the root

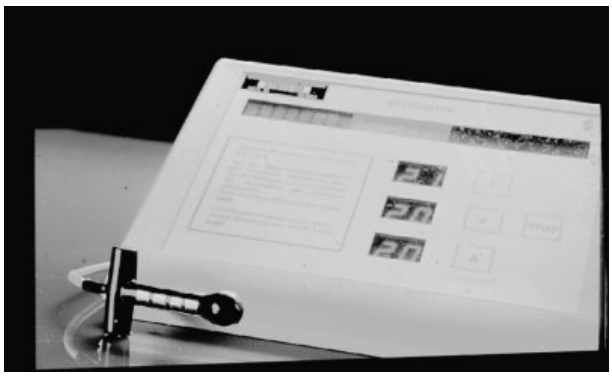

Fig. 1. Left and right surface EMG recording by means of a modified anal plug electrode with four electrodes located on each side of the probe and a conventional mass EMG system (Pelvimeter, Standard Instruments, Karlsruhe, Germany) to non-invasively assess the overall innervation of the external anal sphincter and adjacent muscles during maximal voluntary anal sphincter contraction. Note that on each side of the probe, the outer two of the four electrodes were used for bipolar recording of surface EMG.

mean square (RMS) value of the 120 Hz component of the EMG spectrum is detected by the system and displayed as a result for the time window. This was repeated three times to assure reproducibility.

EMG system amplitude readings (μV) were averaged across the three trials for each side. A symmetry index (SI) was defined as the absolute value of the EMG-amplitude difference between both sides, divided by the higher value of the two:

$$\frac{|\text{right} - \text{left}|}{\max(\text{right, left})} = \text{SI}$$

This allowed assessment of relative asymmetry between 0 (symmetric) and 1 (asymmetric); this SI has not previously been described but is used the first time here to test its validity.

Anorectal Manometry

Furthermore, all women underwent conventional anorectal manometry by means of a high-pressure, low-compliance perfusion system and a multi-lumen water-perfused catheter with eight recording sites spaced 5 mm apart and oriented circumferentially (external diameter 4.8 mm; perfusions rate 3.0 ml/min (Arndorfer Medical Specialties Inc., Greendale, WI). Manometry includes among others, the recording of anal resting pressure (as a measure of the function of the IAS) and maximal and sustained squeeze pressure (as a function of the EAS) using a computerized program (Polygramm Lower GI®, Medtronic GmbH, Düsseldorf, Germany) for data acquisition. Anorectal manometry was performed according to internationally standardized procedures [Azpiroz et al., 2002; Rao et al., 2002].

Statistical Analysis

Data are presented as mean ± standard deviation or percentage, depending on the variable. The results of S-EMG recording (symmetry index, SI) were compared between subgroups of patients (with/without incontinence symptoms) by means of *t*-test, and were correlated to anorectal manometry and clinical data by means of Pearson's correlation coefficient. A *P*-value of $P < 0.05$ was considered indicating significant differences. Analyses were performed with the Statistical Package for Social Sciences, version 11 for Windows (SPSS Inc., Chicago IL).

RESULTS

Table I shows the overall prevalence of urinary and fecal incontinence symptoms in all three cohorts.

Group I

Among the 40 nulliparous pregnant women (Group I), incontinence for flatus at some time during pregnancy was

TABLE I. Overall Prevalence of Incontinence Symptoms in all Three Cohorts

Symptoms	Group I	Group II	Group III
Number of patients	N = 40	N = 15	N = 50
Symptoms	Before/during pregnancy	Before/during/after pregnancy	3/6 months postpartum
No discrimination between gas, solid or liquid stool	4/6	3/4/0	13/5
Incontinence for gas	5/16	2/5/4	24/20
Incontinence for liquid stool	2/2	2/2/2	5/6
Incontinence for solid stool	1/0	1/0/0	1/0
Incomplete evacuation	10/20	2/6/0	11/15
Urinary incontinence			
I°	5/24	0/8/2	20/11
II°	1/5	0/0/0	3/2
III°	1/1	0/0/0	1/1

Numbers are absolute numbers for each condition, for example, before, during and after pregnancy.

reported by 16/40 (40%) but gross fecal incontinence of stool was reported by only two (5%). Thirty women (75%) complained about urinary incontinence. Fifteen women considered hygiene arrangements necessary such as wearing pads at least some time during pregnancy.

The SI ranged between 0.0 and 0.39 (0.113 ± 0.113). It was neither significantly correlated to symptom occurrence or severity, nor to any of the manometric measures (data not shown). Subgroups of women with or without incontinence symptoms (18/40 vs. 22/40) did not differ significantly (*t*-test) in relation to the SI (data not shown).

Group II

Six of the fifteen women had episiotomies performed during delivery, and two of these 15 developed severe fecal incontinence; in addition, 2 women without an episiotomy also reported symptoms of fecal incontinence after delivery.

Their SI ranged between 0 and 0.4 (0.156 ± 0.133). The association between asymmetry and incontinence severity did not reach significance level ($r = 0.20$, $P = 0.07$); however, correlation of pre- and postpartal measures of S-EMG showed high reproducibility of the asymmetry measure within subjects ($r = 0.83$, $P < 0.001$) (Table II).

Group III

Twenty of the fifty women reported flatus incontinence while 6/50 reported incontinence for liquid stool. Only one woman was incontinent to solid feces in the first 3 months postdelivery. These symptoms improved after 6 months. Altogether 25/50 (50%) women also had concomitant urinary incontinence 3 months postdelivery, whereas after 6 months 14 women complained of urinary incontinence (Table I).

The asymmetry index ranged between 0 and 0.57 (0.214 ± 0.175). Relative asymmetry and symptom severity were significantly and positively correlated ($r = 0.43$, $P < 0.001$) (Fig. 2). Compared to the asymptomatic group, women with incontinence symptoms showed a significantly higher asymmetry index (0.14 ± 0.17 vs. 0.27 ± 0.16, $t = -2.7$, $P < 0.005$).

Manometry revealed a significant but low negative correlation between relative asymmetry and the EAS squeeze pressure ($r = -0.28$, $P < 0.05$), and women with asymmetry exhibited significantly lower EAS squeeze pressures than women with symmetry sphincter innervation (94.1 ± 7.5 mm Hg vs. 114.2 ± 7.6, $t = 1.9$, $P = 0.033$). No association was found between relative asymmetry and IAS resting pressure ($r = 0.20$, $P < 0.15$) and no group differences with respect to IAS functions (135.3 ± 5.7 vs. 127.0 ± 6.2 mm Hg).

TABLE II. Individual Symmetry Data From Group II in 15 Women Before and After Vaginal Delivery (Mean of Three Measures)

During pregnancy (EMG amplitude (μv))		Perineal trauma	Postpartum (EMG amplitude (μv))	
Right	Left		Right	Left
8.00	8.00	No	6.00	6.33
7.00	9.67	Yes	7.00	10.00
8.00	6.67	No	7.67	7.00
10.00	10.00	No	9.00	9.00
12.00	12.33	No	9.00	9.33
12.00	12.00	Yes	12.00	12.00
8.67	8.33	No	12.00	10.00
10.33	6.00	Yes	8.67	6.33
7.00	9.00	Yes	6.67	9.00
7.67	9.00	No	8.00	8.00
5.00	7.00	No	5.00	3.00
7.67	11.00	Yes	9.00	12.33
11.33	7.67	No	14.00	10.33
15.00	17.33	No	15.00	17.00
8.00	8.00	No	6.00	6.33

Their symmetry index ranged between 0 and 0.4 (0.156 ± 0.133). Pre- and postpartal measures of S-EMG (average amplitude in μv) showed high reproducibility of the asymmetry measure within subjects ($r = 0.83$, $P < 0.001$).

DISCUSSION

It is well established that major obstetric risk factors for postpartum anal incontinence are, among others, prolonged first and second stages of labor, operative vaginal delivery, and the use of episiotomy. After severe perineal laceration, the rate of anal incontinence increases to between 17 and

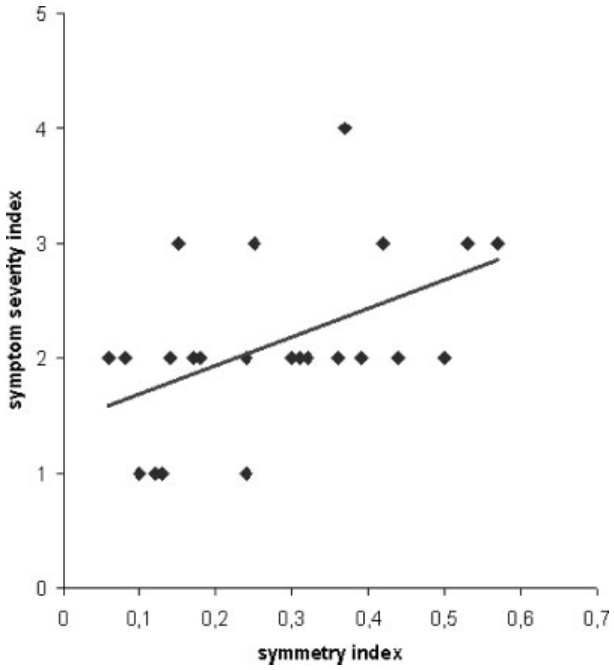

Fig. 2. Significant positive correlation ($r = 0.43$, $P < 0.001$) between severity of incontinence symptoms and degree of asymmetry in Group III.

62% [Crawford et al., 1993; Sultan et al., 1993; Eason et al., 2002]. However, not all women who experience one or more of these risk factors will suffer of incontinence after delivery. Additional factors are needed to explain the occurrence of these symptoms in some but not in other women.

Among those factors mostly unexplored, functional asymmetry of pelvic floor and sphincter innervation has received the least attention. This may be due to the fact that only recently evidence has been gathered indicating that "unilateral pudendal neuropathy" as occasionally found in patients with incontinence [Lubowski et al., 1988; Sangwan et al., 1996] may not be the consequence of sphincter or pudendal nerve damage but may also be found under physiological conditions [Hamdy et al., 1999]. This evidence derives from three classes of investigations: studies on the cortical sensory and motor representation of the pelvic floor, studies on the peripheral sensory and motor pathways in healthy volunteers, and intraoperative studies of sensory and/or motor pathways during selective sacral root monitoring.

During topographic mapping of both cortical hemispheres [Turnbull et al., 1999] in healthy subjects following transcranial magnetic stimulation, it was found that the anal responses were bilaterally represented on the superior motor cortex but showed consistent differences in the degree of bilaterality between individuals. A similar study by another group [Witscher et al., 1998] showed comparable results. When the sensory representation of the pelvic floor over the cortex was investigated by means of functional MRI and other techniques, it was found that at least in a subgroup of volunteers this representation was furthermore asymmetrical [Stottrop et al., 1998].

Hamdy et al. [1999] found that right or left pudendal nerve stimulation evoked anal responses of similar latencies, but asymmetric amplitudes in a subgroup of subjects. Cortical stimulation proceeded by pudendal nerve stimulation facilitated the response, but induced greater facilitation on the dominant side than on the non-dominant side. Needle EMG of the anal sphincter in healthy volunteers also revealed significant intra-individual asymmetries of EMG amplitudes in about 1/3 of cases [Middelsdorf et al., 1998].

Finally, during intraoperative monitoring in patient undergoing sacral rhizotomies, it was shown that the distribution of pudendal afferent fibers was asymmetrical with respect to level (S2–S4) and side (left, right) in the majority of cases and was confined to a single root in 8% of cases [Huang et al., 1997]. Dissociation and "independent" asymmetry can also exist between anal and clitoral sensory pathways [Deletis et al., 2000]. Similarly, Matzel et al. [1999] showed, intraoperatively in adult continent patients undergoing sacral pacemaker implantation for hypotonic bladder treatment, that about 20% showed asymmetry with respect to anal pudendal motor pathways. In both cases, identification of the dominant side or root has been shown to be of clinical relevance.

With a non-invasive tool—surface EMG via anal plug electrodes—we were able to demonstrate for the first time that women who have undergone traumatic childbirth and suffer from incontinence postpartially show asymmetry of sphincter innervation to a higher degree than was the case during pregnancy in primiparous women. This result cannot be attributed to technical measurement errors, as asymmetry was highly reproducible within subjects at two occasions (Groups I and II), although a test for reproducibility of our technique in a healthy control cohort or in a patient group without a pelvic floor trauma would be superior for proof of validity. However, asymmetry can also not 'just' be due to childbirth: there may be pre-existing factors that contribute to symptom severity, and pre-existing asymmetry may be one of these. This hypothesis, however, needs confirmation by further data in women antepartum and after traumatic childbirth.

It is postulated here that asymmetry of sphincter innervation is a major risk factor for postpartal incontinence in those cases in which the trauma occurs on the dominant side of innervation in case of significant asymmetry. As the number of cases in which we were able to prospectively compare pre- and postpartal symmetry functions (Groups I and II) was rather small in the present study, further studies with a larger number of patients will be needed to verify this hypothesis. The fact that asymmetry correlated significantly with the sphincter function of the EAS but not the IAS supports this hypothesis, despite the fact that the association was rather weak. It has been shown that other factors such as age also contribute significantly to anal sphincter function [Enck et al., 1989].

CONCLUSION

Functional asymmetry of anal sphincter innervation was associated significantly with incontinence symptoms in postpartal women after anal sphincter injury. This may be due to intrapartum pudendal nerve trauma or pre-existing asymmetry of innervation.

REFERENCES

Azpiroz F, Enck P, Whitehead WE. 2002. Anorectal functional testing: Review of collective experience. Am J Gastroenterol 97:232–40.

Coats PM, Chan KK, Wilkins M, et al. 1980. A comparison between midline and mediolateral episiotomies. Br J Obstet Gynaecol 87:408–12.

Crawford LA, Quint EH, Pearl ML, et al. 1993. Incontinance following rupture of the anal sphincter during delivery. Obstet Gynecol 82: 527–31.

Deletis V, Vodusek DB, Abott R, et al. 1992. Intraoperative monitoring of the dorsal sacral roots: Minimizing the risk of iatrogenic mictrition disorders. Neurosurgery 30:72–5.

Deletis V, Krzan MJ, Bueno de Camargo A, 2000. et al. Functional anatomical asymmetry of püudendal nerve sensorty fibres. In: Bruch HP, Köcherling F, Bouchard R, Schug-Pass C, editors. New Aspects of High Technology in Medicine. Monduzzi Editore, Milano, Italy 153–8.

Donnelly V, Fynes M, Cambell D, et al. 1998. Obstetric events leading to anal spincter demage. Obstet Gynecol 92:955–61.
Eason EL, Labrecque M, Marcoux S, et al. 2002. Anal incontinence after childbirth. CMAJ 166:326–30.
Enck P, Kuhlbusch R, Lübke H, et al. 1989. Age and sex and anorectal manometry in fecal incontinence. Dis Colon Rectum 32:1026–3.
Enck P, Hinninghofen H, Wietek B, et al. 2004. Functional asymmetry of pelvic floor innervation and its role in the pathogenesis of fecal incontinence. Digestion 69:102–11.
Franz HB, Benda N, Gonser M, et al. 1998. Clinical effects of childbirth with median episiotomy and anal sphincter injury on fecal incontinence of primiparous women. Zentralbl Chir 123:218–22.
Franz H, Stellmacher A, Azpiroz F, et al. 2005. On asymmetry in sphincters (OASIS): First evidence that innervation asymmetry is relevant for sphincter performance. Gastroenterology 128:A38.
Hamdy S, Enck P, Aziz Q, et al. 1999. Laterality effects of pudendal nerve stimulation on human cortico-anal pathways: Evidence for functional asymmetry. Gut 45:58–63.
Huang JC, Deletis V, Vodusek DB, et al. 1997. Preservation of pudendal afferents in sacral rhizotomies. Neurosurgery 41:411–5.
Lubowski DZ, Jones PN, Swash M, et al. 1988. Asymmetrical pudendal nerve damage in pelvic floor disorders. Int J Colorect Dis 3: 158–60.
MacArthur C, Bick DE, Keigly MRB. 1997. Fecal incontinence after childbirth. Br J Obstet Gynecol 104:46–50.
Matzel KE, Stadelmaier U, Hohenfellner M, et al. 1999. Asymmetry of the pudendal motor function assessed during intraoperative monitoring. Gastroenterology 116:A1937.
Middelsdorf P, von Giesen HJ, Schneider J, et al. 1998. Extend of functional asymmetry of the external anal sphincter muscle in healthy volunteers. Neurogastroenterology 10:439.
Rao SSC, Azpiroz F, Diamant N, et al. 2002. Minimal standards of anorectal manometry. Neurogastroenterol Motil 14:553–9.
Sangwan YP, Coller JA, Barrett RC, et al. 1996. Unilateral pudendal neuropathy. Impact on outcome of anal sphincter repair. Dis Colon Rectum 39: 686–9.
Schussler B, Alloussi S. 1983. Ingelman-Sundberg classification of stress incontinence. Gynakol Rundsch 23:166–74.
Signorello LB, Harlow BL, Chekos AK, et al. 2000. Midline episiotomy and anal incontinence: Retrospective cohort study. BMJ 320: 86–90.
Stottrop K, Schnitzler A, Witte OW, et al. 1998. Cortical representation of the anal canal. Gastroenterology 114:A167.
Sultan AH. 1999. Obsterical perineal injury and anal incontinence. Clin Risk 5:193–6.
Sultan AH, Kamm MA, Hudson CN, et al. 1993. Anal sphincter trauman during instrumental delivery. Int J Gynaecol Obstet 43: 263–70.
Turnbull GK, Hamdy S, Aziz Q, et al. 1999. The cortical topography of human anorectal musculature. Gastroenterology 117:32–9.
Witscher K, Greving I, Liepert J, et al. 1998. A bihemispheric representation of the anal sphincter on the human motor cortex. Gastroenterology 114: A1130.
Zetterstrom J, Lopez A, Anzen B, et al. 1999. Anal sphincter tears at vaginal delivery: Risk factors and clinical outcome of primary repair. Obstet Gynecol 94:21–8.