Original article

Symptoms and anal sphincter morphology following primary repair of third-degree tears

K. Davis[1], D. Kumar[1], S. L. Stanton[2], R. Thakar[2], M. Fynes[2] and J. Bland[1]

Departments of [1]Colorectal Surgery and [2]Urogynaecology, St George's Hospital, London, UK
Correspondence to: Mr D. Kumar, Department of Colorectal Surgery and Motility, 3rd Floor, St James Wing, St George's Hospital, London SW17 0RE, UK (e-mail: dkumar@sghms.ac.uk)

Background: Approximately 0·6–9 per cent of vaginal deliveries are complicated by third-degree tears. The precise impact of such injuries on future pelvic floor function remains unknown. The aim of this study was to define the extent of structural and physiological damage to the anal sphincter and to investigate anorectal function in women who sustained third-degree tears during vaginal delivery.

Methods: Fifty-six women who sustained a third-degree tear were investigated prospectively. All patients had a primary repair of the anal sphincter complex, and were assessed by anorectal physiology and endoanal ultrasonography at a mean of 3·6 months. Symptoms were assessed by direct personal interview and also by a self-completed questionnaire.

Results: Forty-four patients had a persistent anal sphincter defect on ultrasonography. The mean resting and squeeze anal canal pressures were significantly lower in patients with a combined defect than in those in whom the repair was intact ($P = 0·036$ and $P = 0·005$ respectively). At direct interview three patients volunteered current symptoms of faecal and/or urinary incontinence whereas 32 reported bothersome symptoms on the questionnaire ($P < 0·001$).

Conclusion: The anatomical and physiological damage sustained during third-degree tears appears to be much greater than is generally appreciated. Primary repair does not provide lasting integrity. A self-administered questionnaire appears to be more accurate in defining the symptomatology.

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Introduction

Childbirth injury is the main aetiological factor for faecal incontinence in women. Vaginal delivery carries inherent risks to the perineum, whereas structural damage to the anal sphincter complex during first delivery predisposes a significant number of women to the development of faecal incontinence at some stage during their life. The growing number of women presenting to colorectal departments, often many years after childbirth, with distressing, socially debilitating symptoms of faecal incontinence and who can recall a difficult birth, poses serious social and economic concerns to patients and healthcare providers[1].

Third-degree tears involve rupture of the anal sphincter complex without involvement of the rectal mucosa, whereas fourth-degree tears extend to involve the anal and rectal mucosa. Such sphincter injuries are a serious complication in approximately 0·6–9 per cent of vaginal deliveries. There is a 58 per cent risk of these patients subsequently developing faecal incontinence[2–4]. Although preventive measures can be taken to reduce the adverse influence of known risk factors associated with perineal tears at the time of delivery, they are not completely successful. It is often difficult to assess the severity of any perineal injury. After perineal injury and primary repair some 54–88 per cent of women have evidence of persistent morphological anal sphincter defects identifiable by endoanal ultrasonography up to 5 years postpartum[2,5,6]. However, it remains controversial whether these anal sphincter 'defects' are solely attributable to disruption of the primary repair despite symptomatic, physiological and morphological evidence of loss of sphincter muscle integrity. In addition, the clinical relevance of such structural abnormalities remains debatable. A proportion

of patients may hide or be unwilling to admit to symptoms, and the full range of symptoms and the associated degree of functional impairment remain undocumented.

Recent studies have found wide variations and significant differences in the clinical assessment, classification, management and follow-up of women who have sustained third- and fourth-degree tears[7,8]. This has led the Royal College of Obstetricians and Gynaecologists to develop new guidelines and recommendations, and to formalize training programmes for the multidisciplinary management of third- and fourth-degree tears following vaginal delivery[9]. Although many women adapt and may not be unduly bothered by symptoms of faecal incontinence up to 1 year after sustaining a third-degree tear, their long-term outcome is uncertain. The aim of this study was to define the extent of structural and physiological damage to the anal sphincter complex, and to investigate pelvic floor function in women who sustained third- and fourth-degree tears during vaginal delivery.

Patients and methods

Between May 2001 and October 2002, 65 women with a mean age of 29 (range 18–43) years who sustained a third-degree perineal tear during childbirth were invited to participate in this study. Nine women declined to attend for anorectal follow-up. Of the 56 women investigated, 53 (94·6 per cent) were primiparous and three multiparous. Twenty-two (39·3 per cent) had a spontaneous vaginal delivery. The remainder required instrumental assistance; 24 (42·9 per cent) had vacuum extraction (ventouse) and ten (17·9 per cent) a forceps-assisted delivery. Eighteen patients had a right mediolateral and two a midline episiotomy. In another two patients the type of episiotomy was not classified. Trauma to the anal sphincter was documented as partial (rupture of the perineum with partial disruption of the anal sphincter muscle fibres), complete (total separation of the anal sphincter muscle complex) or fourth degree (complete disruption of the anal sphincter complex with extension into the anal and rectal mucosa).

Delivery and primary repair

Labour and delivery were managed according to the standard protocol of active management of labour. Perineal tears in all patients were assessed, confirmed and sutured by the senior obstetric registrar or consultant on call. The type of repair chosen was at the discretion of the surgeon. Following dissection and identification of the muscle ends, either an overlapping or end-to-end apposition technique with absorbable sutures was used to restore sphincter integrity. The technique of overlap repair in the primary setting has been described previously[10]. A single dose of an intravenous antibiotic followed by 1 week of oral antibiotics and lactulose was routinely prescribed. A thorough clinical examination at the perineal clinic was performed at least 6 weeks after delivery and all patients were referred to the coloproctology and motility unit for anorectal physiological assessment and endoanal ultrasonography, performed at a mean(s.d.) of 3·6(1·0) (range 2–6) months after delivery. A fully trained operator performed all procedures and the results were stored on hard copy. A colorectal consultant, who was blinded to the patient's clinical profile, independently reported all the results. Obstetric variables analysed included mode of delivery, duration of labour, type of repair, classification of anal sphincter trauma, episiotomy, analgesia used during suturing, and baby weight and head circumference.

Symptom assessment

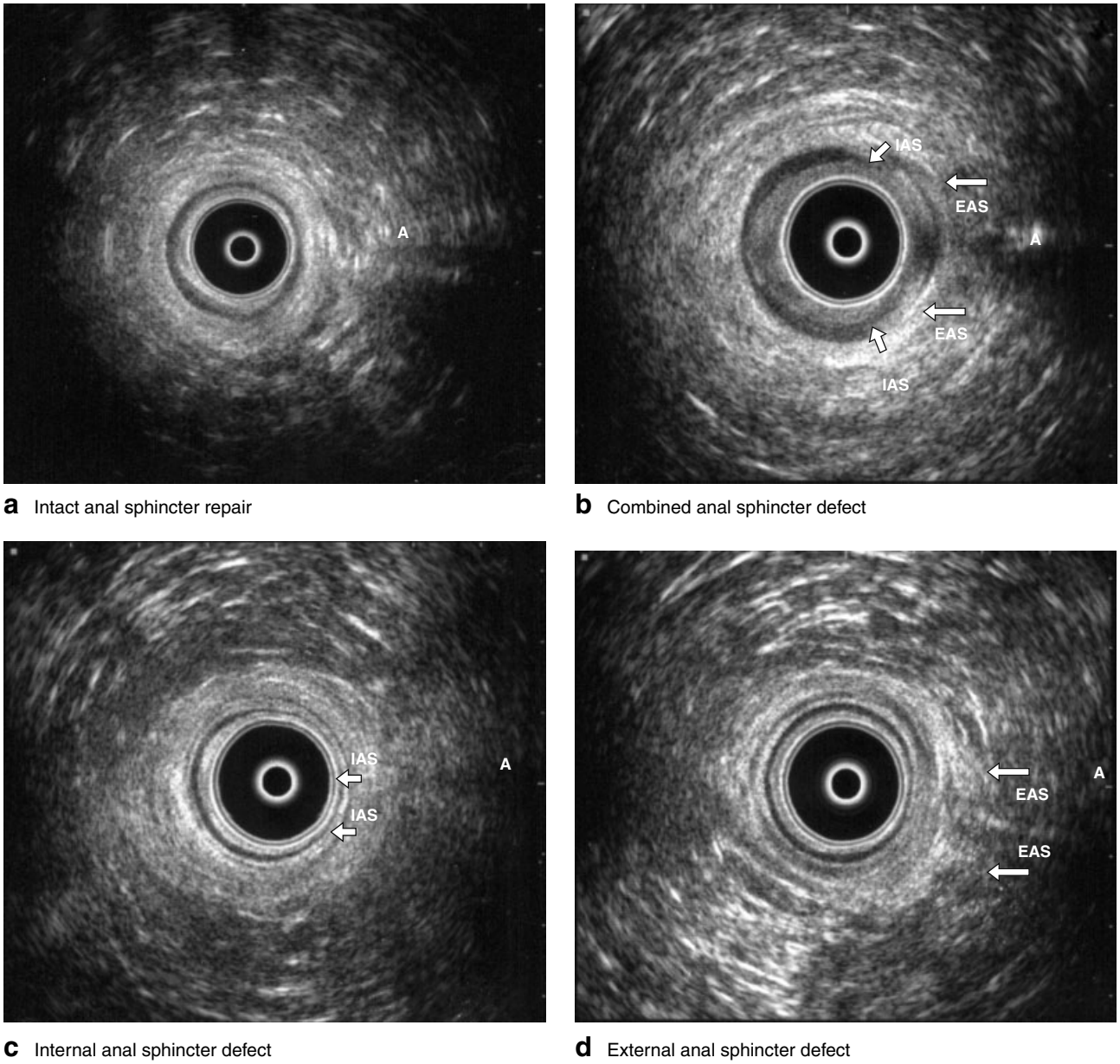
Clinical symptoms were investigated from the patient's perspective by two different methods. The first was by direct clinical interview routinely performed by the pelvic floor physician in the perineal clinic. The second was by a validated, structured and comprehensive pelvic floor symptom questionnaire that patients were asked to complete independently at home before attending for anorectal physiology and endoanal ultrasonography. The questionnaire incorporated elements of other established instruments in this field. It included questions related to urinary and faecal incontinence, stool characteristic and bowel function, prolapse and sexual function. The severity of 'bother factor' caused by individual symptoms, as perceived by the patient, was also assessed using a series of 10-cm visual analogue scales, with descriptions ranging from 'no bother at all' to 'intolerable'.

Endoanal ultrasonography

Endoanal ultrasonography was performed with a 7·5-MHz rotating endoprobe covered with a hard water-filled sonolucent cone (Bruel and Kjer, Naerum, Denmark). This provides a 360° view of the anal canal and sphincter complex. Anal sphincter images were obtained at 0·5-cm intervals starting at the anal verge. Integrity of the primary repair and the presence of anal sphincter defects were documented (*Fig. 1a–d*).

Anorectal physiological assessment

All patients underwent anal manometry using a water-filled microballoon system. Resting and squeeze pressures



a Intact anal sphincter repair

b Combined anal sphincter defect

c Internal anal sphincter defect

d External anal sphincter defect

Fig. 1 Ultrasonographs showing **a** an intact anal sphincter complex following primary repair of a third-degree tear, **b** an internal anal sphincter (IAS) defect (small arrows) and an external anal sphincter (EAS) defect (large arrows), **c** an isolated IAS defect (small arrows) and **d** an isolated EAS defect (large arrows). A, anterior

were measured with the patient in the left lateral position. A station pull-through technique was used. The normal reference values for this laboratory are 8 cmH_2O or greater and 120 cmH_2O or greater for mean resting and squeeze anal canal pressures respectively.

Rectal volume sensation at threshold, constant and maximum tolerated volume was measured using a latex balloon assembly positioned in the rectum and connected to a 60-ml syringe. The rectal balloon was filled with increasing measured volumes of air. The first sensation observed by the patient represented the threshold volume. Constant sensation in the rectum and the urge to evacuate the rectum represented the constant volume sensation and maximum tolerated volume respectively. Up to 40 ml, 150–160 ml and 200–220 ml were used as normal reference values for

threshold, constant sensation and maximum tolerated values respectively.

Anal canal mucosal electrosensitivity was measured in the upper, mid and lower anal canal by passing a direct current through an electrode positioned in the anal canal. A reference electrode was applied to the lateral aspect of the patient's thigh. Up to 16 mA was used as a normal reference value.

Statistical analysis

Summary statistics were expressed as mean(s.d.). One-way analysis of variance and *post hoc* pair-wise comparison with the Scheffé test were used to identify differences between resting and squeeze pressures. Correlation and regression were used to analyse the relationships between frequency and severity of symptoms on direct interview and questionnaire by means of a commercially available statistics program, SPSS® version 10.0 (SPSS, Chicago, Illinois, USA).

Results

Delivery and primary repair

During the study interval the annual rate of third-degree tears was 1·4 per cent of all deliveries. Fifty-six women of mean age 29 (range 18–43) years with third-degree tears were studied. The mean(s.d.) follow-up was 3·6(0·9) (range 2–6) months. The obstetric details are summarized in *Table 1*.

Thirty-eight of the 56 patients underwent an overlapping sphincter repair following a previously published protocol[10], five had additional interrupted suturing of the internal anal sphincter, and 12 had an end-to-end approximation using a monofilament absorbable suture. One patient at the time of suturing had only perineal reconstruction and no sphincter repair but she was subsequently found to have a combined defect (internal and external anal sphincters) on endoanal ultrasonography. The majority of repairs were performed under either epidural (33 of 56) or spinal (20) analgesia. In one patient suturing was performed under local anaesthesia and two women had intravenous pethidine sedation.

Table 1 Obstetric details

Parity*	
Primiparous	53 (94·6)
Multiparous	3 (5·4)
Duration of labour (h)†	
Total duration	12·1(8·2) (1·3–53·5)
Second stage	2·0(2·1) (0·1–3·5)
Mode of delivery*	
Spontaneous vaginal	22 (39·3)
Ventouse	24 (42·9)
Forceps	10 (17·9)
Episiotomy*	
None	34 (60·7)
Routine	20 (35·7)
Extended	2 (3·6)
Baby head circumference (cm)†	34·8(1·6) (32·0–38·5)
Baby weight (g)†	3647(541) (2300–5200)
Anal sphincter injury during labour*	
Partial	45 (80·4)
Complete	7 (12·5)
Fourth-degree tear	4 (7·1)
Analgesia used during suturing*	
Epidural	33 (58·9)
Spinal	20 (35·7)
Local anaesthetic	1 (1·8)
Intravenous pethidine sedation	2 (3·6)
Repair following delivery*	
Overlapping repair	38 (67·9)
Overlapping repair plus interrupted suturing of internal anal sphincter	5 (8·9)
End-to-end approximation	12 (21·4)
Perineal reconstruction with no sphincter repair described	1 (1·8)

*Values in parentheses are percentages; †values are mean(s.d.) (range).

Endoanal ultrasonography

In only 12 patients (21·4 per cent) was an intact primary anal sphincter repair observed on endoanal ultrasonography. The most common defect identified after primary repair in the remaining 44 women was a combined anterior defect of both internal and external anal sphincters (35 patients, 62·5 per cent); four women (7·1 per cent) had a defect or non-uniformity of the internal anal sphincter only and five (8·9 per cent) had disruption of the external anal sphincter alone.

Anorectal physiology

The mean maximal resting and squeeze anal canal pressures for the entire group were in the normal range. When the anal canal pressures were divided into subsets according to the type of defect, it was found that patients with a combined anal sphincter defect had significantly lower resting and squeeze anal canal pressures than women with an intact anal sphincter ($P = 0·036$ and $P = 0·005$ respectively) (*Fig. 2*). In those with an isolated defect of the internal or external anal sphincter, the mean resting and squeeze anal canal pressures were not significantly different from those in other groups. The overall mean rectal volume was reduced; constant and maximum tolerated volumes were 119·6 and 165·8 ml respectively. Anal mucosal

electrosensitivity for the whole group was normal in the lower and mid anal canal (11·0 and 14·3 mA) and slightly impaired in the upper anal canal (18·8 mA).

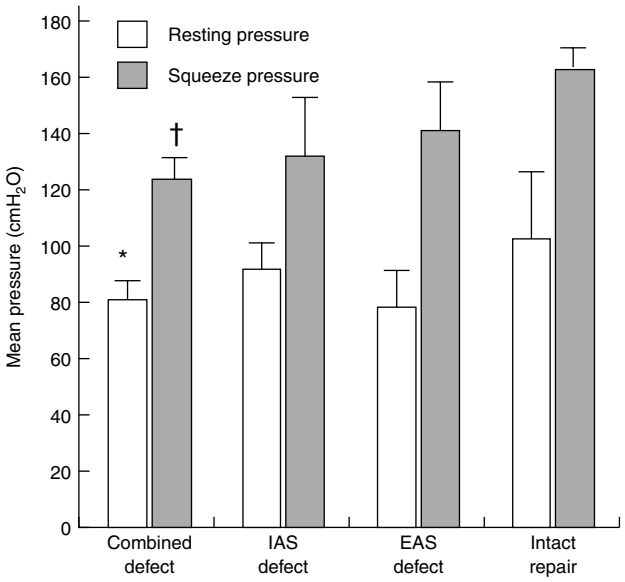


Fig. 2 Mean(s.d.) resting and squeeze anal canal pressures in relation to sphincter status following repair of a third-degree tear. IAS, internal anal sphincter; EAS, external anal sphincter. *$P = 0{\cdot}036$ *versus* resting pressure with intact repair (ANOVA); †$P = 0{\cdot}005$ *versus* squeeze pressure with intact repair (*post hoc* Scheffé test)

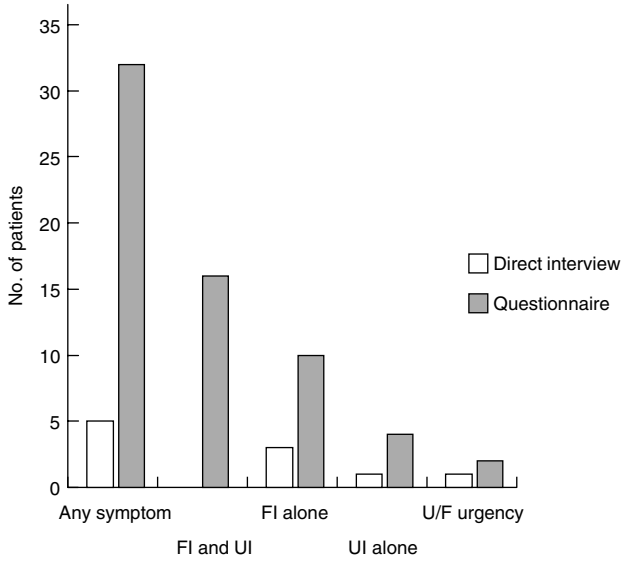


Fig. 3 Symptoms described by patients at direct interview and questionnaire. FI, faecal incontinence; UI, urinary incontinence; U/F, urinary or faecal urgency. $P = 0{\cdot}049$ (regression analysis)

Symptom assessment

Fifty-two patients completed the questionnaire. Five (9·6 per cent) of 52 women admitted to pelvic floor symptoms at direct interview compared with 32 (61·5 per cent) of 52 on the symptom questionnaire ($P < 0{\cdot}001$, Sign test) (*Fig. 3*). Only three patients volunteered the same symptoms on both direct interview and questionnaire. At direct interview three patients admitted to symptoms of faecal incontinence (flatus, liquid and/or solid stool) and one to urinary incontinence. In contrast, 26 of 52 patients reported current symptoms of faecal incontinence in the questionnaire survey. Ten had faecal incontinence alone and 16 had a combination of urinary and faecal incontinence. Four patients reported symptoms of urinary incontinence alone and one had urinary urgency but no incontinence. Although a large proportion of the patients who reported faecal incontinence had only mild to moderate symptoms including involuntary loss of flatus, seven patients reported daily, ten weekly and five monthly episodes of incontinence. Four patients who suffered loss of liquid and solid stool reported leakage twice or more per week. Fourteen of the 20 patients who reported involuntary leakage of urine had two or more episodes weekly, and the remainder reported one or more episodes daily.

Regression analysis revealed a significant relationship between any symptoms and overall 'bother factor' ($P = 0{\cdot}049$) determined by questionnaire; however, using data from direct interview only the relationship between urinary symptoms and overall trouble associated with pelvic floor symptoms approached significance ($P = 0{\cdot}060$). When the individual symptoms and the overall 'bother factor' were correlated, symptoms of urge urinary incontinence ($r = 0{\cdot}665$, $P < 0{\cdot}001$), stress urinary incontinence ($r = 0{\cdot}466$, $P = 0{\cdot}001$) and incontinence to flatus ($r = 0{\cdot}364$, $P = 0{\cdot}009$) reached significance (correlation significant at two-tailed $P = 0{\cdot}010$). There was no correlation between the presence of symptoms and an anal sphincter defect on endoanal ultrasonography ($r = 0{\cdot}171$, $P = 0{\cdot}225$). However, there was an inverse correlation between the presence of a defect and mean squeeze ($r = -0{\cdot}445$, $P = 0{\cdot}001$) and resting ($r = -0{\cdot}384$, $P = 0{\cdot}004$) anal canal pressures.

Discussion

Third-degree tears occur relatively infrequently. The incidence of 1·4 per cent in this study period falls within the published range[1,3]. It is higher in units in which a midline episiotomy is used routinely[11]. Although the majority of these injuries are recognized and repaired at the time of delivery, the precise mechanism by which anal sphincter

injury occurs during a traumatic labour remains elusive. In agreement with previous reports, the authors found that overt injury to the anal sphincter complex following either a spontaneous uncontrolled tear or extension of an episiotomy was greatest amongst primiparous women.

There is increasing evidence that instrument-assisted delivery and episiotomy, particularly midline episiotomy, increase the risk of direct mechanical damage to the anal sphincters[12–15]. Although successful vacuum extraction results in less maternal trauma there is little difference in postpartum symptoms compared with those after forceps-assisted delivery[16–19]. In the present study 34 patients (in whom second-stage labour was failing to proceed despite oxytocin stimulation) required instrumental assistance, 24 of whom had a successful ventouse delivery. Nineteen of these 24 patients were subsequently found to have anal sphincter defects on endoanal ultrasonography.

These findings are at variance with those reported by Sultan *et al.*[2], who noted that none of their patients who sustained a third-degree tear had undergone vacuum extraction. It is possible that anal sphincter damage in the present patients actually occurred during a prolonged second stage of labour. An alternative explanation may be the use of epidural analgesia. It has been suggested previously that there is a significant relationship between epidural blockade and pelvic floor dysfunction, including anal sphincter muscle disruption[20]. In this study 33 of 56 patients had epidural analgesia, the majority of whom had either a combined internal and external anal sphincter defect or an isolated defect of one of the anal sphincter muscles.

In keeping with data from previous reports[2,3,21], residual anal sphincter defects after repair were identified by endoanal ultrasonography in 44 patients in this study. The majority of these patients had a combined defect of the internal and external anal sphincter muscles. There are two possible explanations for these findings: first, that the anal sphincter repair became disrupted in the postoperative period owing to either sepsis or tissue factors and, second, that the injury was classified incorrectly at the time of repair. In the presence of gross perineal trauma it is not easy to identify the structures accurately so an incorrect classification may be documented and an injury missed[8]. The second explanation seems more plausible because four-fifths of patients in this study were reported to have partial injury to the anal sphincter muscle and yet an overlapping anal sphincter repair was achieved in more than two-thirds. This suggests that the injury was more severe than initially recognized and, more importantly, that a complete repair may not have been performed as part of the primary procedure.

The high proportion of persistent anal sphincter defects found in women who had a non-assisted delivery and did not have an episiotomy suggests that, regardless of the mode of delivery, primary repair of a severe perineal injury remains unsatisfactory and staging of third-degree tears is variable. Primary repair may be difficult at the time of delivery owing to bleeding, oedema and unrecognizable tissue damage.

The observed discrepancy between direct interview and questionnaire may be explained by social factors, social taboo and preconceived ideas of what may be expected after delivery, the clinical environment of a busy noisy clinic, or the circumstances of a multiprofessional consultation (with midwife, physician, physiotherapist and clinic nurse, and new baby present). It has been shown previously that the frequency and severity of faecal incontinence following a third-degree tear varies from 19 to 58 per cent[2–5,22], with the majority of patients reporting mild symptoms even in the presence of significant sphincter damage. This may be explained largely in terms of compensation by other factors that contribute to the continence mechanism. Although relevant in the long term, such a structural abnormality in the presence of minor symptoms is often neglected in the short term, particularly when considering future modes of delivery.

Acknowledgements

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