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Anal Incontinence and Anal Sphincter Rupture during Childbirth – Prevalence, Diagnosis and Treatment

by Tarja Pinta

Academic dissertation

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to my family

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LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following original publications, which will be referred to by their Roman numerals.

- I Pinta T, Kylänpää-Bäck M-L, Salmi T, Järvinen HJ, Luukkonen P. Delayed sphincter repair for obstetric ruptures: Analysis of failure. Colorectal Disease 2003; 5: 73-78.
- II Pinta T, Kylänpää M-L, Salmi T, Teramo K, Luukkonen P. Primary sphincter repair: are the results good enough? Dis Colon Rectum 2004; 47: 18-23.
- III Pinta T, Kylänpää M-L, Teramo K, Luukkonen P. Sphincter rupture and anal incontinence after first vaginal delivery. Acta Obstet Gynecol Scand 2004; 83: 917-922.
- IV Pinta T, Kylänpää M-L, Luukkonen P, Tapani E, Kivisaari A, Kivisaari L. Anal incontinence: diagnosis by endoanal US or endovaginal MRI. Eur Radiol 2004; 14: 1472-1477.

ABBREVIATIONS

ABBREVIATIONS

EAUS	endoanal ultrasound
IAS	internal anal sphincter
EAS	external anal sphincter
PNTML	pudendal nerve terminal motor latency
MR	magnetic resonance
MRI	magnetic resonance imaging
PPV	positive predictive value
NPV	negative predictive value
EVMRI	endovaginal magnetic resonance imaging
EAMRI	endoanal magnetic resonance imaging
RAIR	rectoanal inhibitory reflex
SNS	sacral nerve stimulation
BMI	body mass index

ABSTRACT

It is estimated that anal incontinence affects up to 18 per cent of all people and its prevalence increases with age. Sphincter rupture during vaginal delivery is an important factor for its development, while another mechanism is damage to the pudendal nerve during vaginal delivery.

The aim of the present study was to examine operation results after anterior anal sphincter repair or primary sphincter repair in patients with obstetric trauma. Another primary object was to investigate the incidence of occult anal sphincter defects and symptoms of anal incontinence among primiparous women before and after the first vaginal delivery. We also assessed possible risk factors for anal sphincter rupture during vaginal delivery. Endoanal ultrasound (EAUS) examination has been found to be worthwhile for diagnosing anal sphincter defects, and we investigated here whether endovaginal magnetic resonance imaging (EVMRI) might also be useful for diagnosing anal sphincter defects.

Anterior anal sphincter repair for anal incontinence was performed on 39 women with a history of childbirth at Helsinki University Central Hospital. The results of follow-up questionnaires administered were good for 12 patients (31%), acceptable for 15 (38%) and poor for 12 (31%). Postoperative EAUS showed sphincter overlap in 28 patients (72%), but a defect was still found in 11 (28%). A defect in postoperative EAUS correlated with a poor clinical result according to Parks' evaluation scheme (R=0.8, P<0.01) and the patients' questionnaire results (R=0.7, P<0.01). The patients with poor clinical results (Parks III/IV) were statistically significantly older (median 63 years, range 26-73) than those with favourable results (Parks I/II) (median 45 years, range 27-79) (P<0.05). Furthermore, the duration of incontinence symptoms correlated with poor functional results (R=0.4, P<0.05).

To assess the results of primary sphincter repair, a total of 52 females with a thirddegree or fourth-degree perineal laceration were examined. The median follow-up time was 15 (range 2-144) months after the primary sphincter repair. The control group consisted of 51 primiparous females with no clinically detectable perineal laceration after vaginal delivery. After primary sphincter repair, 61 per cent had symptoms of anal incontinence, of which 20 per cent were cases of fecal incontinence. The patients had more severe symptoms of anal incontinence according to Parks' and Wexner's classifications than did the control group (P<0.001 with both classifications). A persistent defect in the external anal sphincter (EAS) was found in EAUS in 75 per cent of the rupture group compared with 20 per cent of the control group. Abnormal fetal presentation at birth was the only risk factor for anal sphincter rupture during vaginal delivery.

To find out the incidence of anal incontinence and an occult anal sphincter defect after the first vaginal delivery, 99 women were examined before and after delivery. In the end 75 of these had a vaginal delivery and 24 a caesarean section. Vacuum extraction was necessary in 20 cases. Symptoms of anal incontinence, mainly gas incontinence, increased after vaginal delivery more than after caesarean section (p<0.032). Occult anal sphincter defects were noted by EAUS in 17 of the 75 women (23%) after vaginal delivery and in nine out of the 20 women (45%) after vacuum extraction, but no new sphincter defects were found in the caesarean section group. Mean squeeze pressures were significantly decreased in the patients with EAS defects (P=0.0025). Vacuum extraction is a risk factor for anal sphincter defects but does not significantly increase anal incontinence symptoms or reduce mean anal sphincter pressures.

To evaluate the possibility of using EVMRI for detecting anal sphincter defects, we examined 19 women prospectively by preoperative EAUS and EVMRI. The sphincter defects were validated at operation. EAUS and EVMRI showed almost similar agreement with the surgical findings; 12 out of 19 (63%) vs. 11 out of 19 (58%). Internal anal sphincter (IAS) defects were equally detected by EAUS and EVMRI in relation to surgical diagnosis.

In conclusion, the results suggest that anterior anal repair gives acceptable shortterm clinical results. Advanced age, preoperative signs of perineal descent, long-lasting severe incontinence symptoms and a persistent defect in postoperative EAUS seem to be related to a poor clinical result. A persistent EAS defect and symptoms of anal incontinence are common after primary sphincter repair. The first vaginal delivery may result in occult sphincter defects, especially if vacuum extraction is used. EAUS and EVMRI are of equal value in diagnosing anal sphincter defects.

INTRODUCTION

Anal incontinence, involuntary loss of flatus and / or feces, is an awkward symptom which often limits the individual's social life. It is also underreported, under-recognized and poorly understood. According to Parks, symptoms of anal incontinence can be classified into four grades. Grade I implies full continence, Grade II incontinence to flatus, Grade III incontinence to liquid stools and Grade IV incontinence to solid stools (Hardcastle and Parks 1970).

The exact prevalence of anal incontinence is unknown, but it is estimated that up to 18% of all the people suffer from it and that its prevalence increases with age (Johanson and Lafferty 1996, Perry et al. 2002). In a previous study by Sultan et al. (1993a), fecal urgency or anal incontinence occurred in about 13 per cent of primiparous women. Macarthur et al. (1997) showed that new symptoms of anal incontinence occur in about 4 to 5 per cent of women after vaginal delivery. Thus vaginal delivery seems to be an important risk factor for anal incontinence (Kamm MA 1994, Pollack et al. 2004).

The most common cause for symptoms of anal incontinence is rupture of the external and /or internal anal sphincter during traumatic vaginal delivery. The rupture site is always the anterior side of the muscles. In their prospective study of primiparous women who had undergone vaginal delivery, Sultan et al. (1993a) showed that up to one in three women may sustain occult anal sphincter trauma visible in EAUS. A third of these women had new symptoms of anal incontinence. The symptoms are often mild in young women, but might become worse with age.

According to earlier studies, the risk factors for anal sphincter rupture during vaginal delivery include primiparous status (Walsh et al. 1996), instrumental delivery (Sultan et al. 1994a, Macarthur et al. 1997, Donnelly et al. 1998), prolonged second-stage labour (Donnelly et al. 1998, Groutz et al. 1999), infant birth weight exceeding 4 kg (Walsh et al. 1996), and occipitoposterior presentation (Fitzpatric et al. 2001). The role of mediolateral episiotomy is not clear (Groutz et al. 1999, de Leeuw et al. 2001a).

Another mechanism causing anal incontinence is damage to the pudendal nerve during vaginal delivery (Snooks et al. 1984a, 1984b, Sultan et al. 1994b) or progressive denervation of the anal sphincter muscles caused by chronic straining (Snooks et al. 1990). This denervation is often reversible, but might be cumulative with subsequent deliveries (Snooks et al. 1990, Ryhammer et al. 1995).

EAUS allows accurate imaging of the external and internal anal sphincters (Law et al. 1991, Nielsen et al. 1992, Sultan et al. 1993b, 1994c, Bartram and Sultan 1995), and with the development of the magnetic resonance imaging (MRI) technique, these can also be visualized using endoanal MRI (Rociu et al. 1999a).

INTRODUCTION

The most important consideration after anal sphincter rupture caused by delivery is to achieve good primary sphincter repair as soon as possible, as inadequate primary repair can lead to early anal incontinence (Nielsen et al. 1992, Sultan et al. 1994a, Poen et al. 1998, Davis et al. 2003, Zetterström et al. 2003). If there are still symptoms of anal incontinence present and an anal sphincter rupture is found in the EAUS examination, a new operation can be performed. The most common surgical procedure for a secondary or delayed anal sphincter repair is the anterior overlapping technique as described by Parks and McPartlin in 1971 and later modified by Slade et al. (1977).

One aim of the present study was to determine the factors related to obstetric anal incontinence. The results of delayed sphincter repair and primary sphincter repair in women with a history of childbirth were studied and the prevalence of occult anal sphincter defects and symptoms of anal incontinence after the first vaginal delivery were assessed. The effectiveness of EVMRI vs. EAUS for diagnosing the anal sphincter defects was also determined.

REVIEW OF THE LITERATURE

1. History of anal incontinence

Anal incontinence was thought at first to be caused by a neurogenic dysfunction of the anal complex, and diagnostic techniques were focused mostly on functional information. The development of imaging techniques that involve the use of endoanal devices - EAUS and endoanal MRI - resulted in good visualization of the anal sphincters, however, and this made it possible to obtain a better understanding of anal incontinence by showing that anal sphincter tears are the main cause (Law et al. 1991, Sultan et al. 1994c). Consequently, detailed imaging of the anal sphincter became important for the diagnosis and treatment of symptoms of anal incontinence.

2. Epidemiology of anal incontinence

2.1. General

The exact incidence and prevalence of anal incontinence are unknown because it is often a hidden problem. It is a common condition, however, especially in older individuals, as Tobin and Brocklehurst (1986) found that as many as 10 per cent of their patients were incontinent for feces at least once a week. An epidemiological study by Nelson et al. (1995) to identify the community-based prevalence of fecal incontinence, showed it to be 2.2 per cent of the general population.

In an epidemiological prospective study in Rockford, Illinois (Johanson and Lafferty 1996), the overall prevalence of anal incontinence was 18.4 per cent. Stratified by the frequency of occurrence - daily, weekly or once per month or less, the prevalence rates were 2.7, 4.5 and 7.1 per cent, respectively.

A recent large postal questionnaire study of the prevalence of fecal incontinence in adults aged 40 years or more living in the community (Perry et al. 2002) found that 1.4 per cent reported major fecal incontinence and 0.7 per cent major fecal incontinence with bowel symptoms that had an impact on the quality of life, incontinence being more prevalent and more severe in older people. There was no significant difference between men and women.

2.2. Obstetric anal incontinence

Anal incontinence as an immediate consequence of childbirth is more common than was previously believed, the main risk factors being an anal sphincter tear during delivery and subsequent childbirth (Pollack et al. 2004). Up to 44 per cent of women

REVIEW OF THE LITERATURE

with a clinically diagnosed anal sphincter tear in the report referred to had symptoms of anal incontinence, as also did some women without a clinically diagnosed sphincter tear (25 per cent), and the symptoms increased significantly over a five-year follow-up in both groups. Symptoms of anal incontinence without a clinically diagnosed sphincter tear may be due to neurological impairment or occult anal sphincter defects.

The incidence of anal incontinence following vaginal delivery has varied between 4 and 44 per cent (Sultan et al. 1993a, Crawford et al. 1993, Kamm MA 1994, Varma et al. 1999, Pollack et al. 2004) and the prevalence of anal sphincter defects in EAUS in patients with anal incontinence varies between 65 and 87 per cent (Law et al. 1991, Deen et al. 1993, Karoui et al. 1999).

2.2.1. Incontinence after third or fourth-degree anal sphincter rupture

Anal sphincter rupture during vaginal delivery is a risk factor for anal incontinence (Sultan et al. 1993a, Kamm MA 1994, Pollack et al. 2004), and inadequate primary repair of these anal sphincter injuries can lead to early anal incontinence.

Clinically diagnosed anal sphincter tears are rare, as the incidence of anal sphincter damage due to a third-degree or fourth-degree tear in women undergoing vaginal delivery varies between 0.4 and 2.4 per cent (Haadem et al. 1988, Sultan et al. 1993a, Nielsen et al. 1992, Walsh et al. 1996, Fornell et al. 1996, Pirhonen et al. 1998, Cook and Mortensen 1998, de Leeuw et al. 2001b).

Up to 85 per cent of women have persistent sphincter defects after a third-degree rupture, and up to 50 per cent have anorectal complaints, despite apparently adequate repair (Nielsen et al. 1992, Crawford et al. 1993, Sultan et al. 1994a, Tetzschner et al. 1996, Haadem and Gudmundsson 1997, Poen et al. 1998). The most common type of repair employs an end-to-end technique.

Kairaluoma et al. (2004) have recently published promising medium-term results of primary sphincter repair using the overlapping technique instead of the end-toend technique, noting that occasional incontinence to flatus and stools occurred in 17 and 7 per cent of the patients and that an EAS overlap was found in up to 94 per cent.

2.2.2. After an occult anal sphincter defect

One of the reasons for anal incontinence in women is unrecognized damage to the anal sphincter during childbirth. Occult anal sphincter defects are common after vaginal delivery, and are often associated with symptoms of anal incontinence.

Sultan et al. (1993a) studied 202 women prospectively six weeks before delivery; 150 of them six weeks after delivery and 32 with abnormal findings six months after delivery and found that 13 per cent of the primiparous women and 23 per cent of the multiparous ones who had delivered vaginally had anal incontinence or fecal urgency six weeks afterwards and up to 35 per cent of the primiparous women had a sphincter defect visible in EAUS at at that point. Twenty-two women with anal sphincter defects were also studied six months after delivery, and all of them had a persistent defect. Of the multiparous women, 40 per cent had an anal sphincter defect before delivery and 44 per cent afterwards. There was a close association between sphincter defects and the development of symptoms of anal incontinence.

Occult anal sphincter defects have been reported in several other connections: Donnelly et al. (1998) 35%, Rieger et al. (1998) 41%, Zetterström et al. (1999) 20%, Varma et al. (1999) 12.2 %, Faltin et al. (2000) 28%, Belmonte-Montes et al. (2001) 29%, Williams et al. (2001) 29%, and Nazir et al. (2002) 19%.

2.2.3. After caesarean section

Caesarean section protects the anal sphincters from occult injury but not from symptoms of anal incontinence (Zetterström et al. 2003). Sultan et al. (1993a) found that none of their 23 women who had undergone caesarean section had an occult anal sphincter defect identifiable in EAUS examination, although Fynes et al. (1998) noted that caesarean delivery performed in late labour does not protect the anal sphincter mechanism, probably because of neurological injury. There is some evidence that the routine use of caesarean section does not prevent anal incontinence (Lal et al. 2003, Harkin et al. 2003).

3. Pathophysiology of anal incontinence

Normal continence depends on many factors: the volume and consistency of stools, colonic transit time, rectal distensibility, anal sphincter structure and function, anorectal sensation, anorectal reflexes and mental function. Abnormalities of any of these factors, alone or in combination, can lead to incontinence. A high volume of liquid stool or diarrhoea, for example, can lead to incontinence even if the anal sphincter is normal. Inflammatory bowel disease or radiation proctitis can cause anal incontinence because of a poorly distensible rectum and inadequate reservoir function.

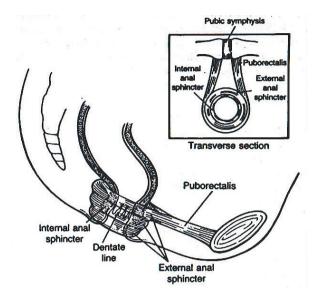
Adequate rectal sensation is necessary for normal continence (Swash M 1985, Sun et al. 1990a). Patients with fecal impaction and overflow incontinence, diabetes mellitus or spinal disease have diminished rectal sensation (Wald and Tunuguntla 1984, Read and Abouzekry 1986, Sun et al. 1990b), and patients with traumatic or idiopathic incontinence may have abnormal sensation of the anal canal (Rogers et al. 1988, Miller et al. 1989).

4. Structure of the normal anal canal

The anal sphincter mechanism comprises the IAS, EAS and puborectalis muscles (Figure1). The internal sphincter, the thickened, circular smooth-muscle layer of the distal rectal wall that is under autonomic control, accounts for 80 per cent of the resting pressure (Frenckner and Euler 1975, Schweiger M 1979). EAS, puborectalis muscles and other muscles of the levator ani, which form a skeletal muscle complex and function voluntarily, behave as a functional unit, in spite of independent innervation, the external sphincter by the pudendal nerves and the puborectalis by the pelvic branches S-3 and S-4 (Percy et al. 1981, Wunderlich and Swash 1983).

REVIEW OF THE LITERATURE

Figure 1. Diagram of the rectum, anal canal and surrounding muscles (Basic Provisions of Grant of Permission).



A disruption of the IAS will lead to passive fecal incontinence (loss of feces without the patient's awareness), while dysfunction of the EAS will result in urge incontinence due to the patient's inability to suppress defecation (Gee and Durdey 1995). Voluntary sphincter contraction normally doubles the pressure in the anal canal for a few minutes (Pemberton and Kelly 1986). A spinal reflex causes the striated sphincter to contract during sudden increases in intra-abdominal pressure, such as coughing (Pemberton and Kelly 1986, Sun et al. 1990a).

5. Etiology of anal incontinence

An anal sphincter rupture during vaginal delivery can lead to anal incontinence. Pudendal neuropathy, also known as idiopathic incontinence, may be cumulative with subsequent deliveries (Ryhammer et al 1995). Other common reasons for anal incontinence include previous anoperineal surgery, chronic diarrhoea and neurological diseases.

5.1. Obstetric causes of anal incontinence

5.1.1. Anal sphincter rupture

An anal sphincter rupture during vaginal delivery can lead to anal incontinence (Crawford et al. 1993, Sultan et al. 1993a, Sorensen et al. 1993, Sultan et al. 1994a, Walsh et al. 1996, Tetzschner et al. 1996, Haadem and Gudmundsson 1997, Poen et al. 1998, de Leeuw et al. 2001a), and any subsequent vaginal delivery will further in-

creases the risk (Bek and Laurberg 1992, Ryhammer et al. 1995, Fynes et al. 1999a, Pollack et al. 2004).

Perineal tears are classified into four degrees according to the international classification of diseases (Franz and Hirsch 1996): a first-degree tear involves the perineal skin and vaginal epithelium, a second-degree tear also involves the underlying fascia, a third-degree tear causes defects in the anal sphincters, and a fourth-degree rupture also involves the anorectal mucosa. Clinically recognized sphincter ruptures are rare, their incidence varying between 0.4 and 2.4 per cent of women delivering vaginally (Haadem et al. 1988, Moller and Laurberg 1992, Nielsen et al. 1992, Sultan et al. 1993a, Fornell et al. 1996, Walsh et al. 1996, Cook and Mortensen 1998, Pirhonen et al. 1998, de Leeuw et al. 2001b).

Zettersröm et al. (2003) have shown recently that up to 83 per cent of women sustaining a clinical sphincter tear at delivery and /or developing symptoms of anal incontinence after delivery have abnormalities in follow-up by EAUS, anorectal manometry or electrophysiology.

Developments in EAUS examination techniques have improved the detection of anal sphincter tears and occult anal sphincter injuries. Sultan et al. (1993a) showed in a prospective study that three per cent of primiparous women had clinically diagnosed third-degree or fourth-degree tears and up to 35 per cent of those who delivered vaginally developed a sphincter defect visible in the EAUS examination. Only four per cent of multiparous women developed new defects visible in EAUS, although 40 per cent had an existing defect before delivery. EAS damage was detected only in the presence of a tear or episiotomy, which suggests that it occurs as part of a direct continuation of perineal disruption. The IAS was injured more frequently than the EAS, and was sometimes damaged when the perineum remained intact.

The significance of these occult injuries has not been fully established. Although a defect may be found in an EAUS examination, not all of these women have symptoms of anal incontinence, although the occult injury may predispose them to incontinence later in life, after the menopause. Occult anal sphincter defects have been reported in several more recent studies (Donnelly et al. 1998, Rieger et al. 1998, Zetterström et al. 1999, Varma et al. 1999, Faltin et al. 2000, Belmonte-Montes et al. 2001, Williams et al. 2001, and Nazir et al. 2002).

5.1.2. Nerve injury

Injury to the muscles of the pelvic floor and anal sphincters can be mechanical or neurological.

Before the development of EAUS, injury to the nerves of the pelvic floor was thought to be one of the main causes of incontinence (Snooks et al. 1984a). Parks et al. (1977) showed increased fibrous connective tissue and degenerative changes in EAS muscle fibres in 24 women with fecal incontinence and suggested that the incontinence occurred as a result of denervation through nerve injury after childbirth, or as a result of entrapment or stretch injury in the pudendal nerve during repeated straining to defecate. Later, Snooks et al. (1990) reported a 5-year follow-up study of the effect of childbirth on the pelvic floor musculature, providing direct evidence

REVIEW OF THE LITERATURE

for the hypothesis that pudendal neuropathy due to vaginal delivery persists and may worsen with time. Jameson et al. (1994) suggested that PNTML increases with age and that the IAS generates a lower pressure with increasing age.

Speakman et al. (1991, 1995) demonstrated that the IAS is also affected by denervation in patients with incontinence, and later idiopathic degeneration of the IAS was described by Vaizey et al. (1997), who suggest that it may be one of the most common causes of passive fecal incontinence in the community.

In the majority of women suffering from incontinence the symptoms are manifested after the menopause, probably because of ageing and hypo-estrogenic status of the nerves, muscles and ligaments of the pelvis and of the anorectal supporting tissue. Cumulative obstetric injury can be a risk factor for incontinence, together with a low level of estrogen (Snooks et al. 1990, Ryhammer et al. 1995, 1996, Donnelly et al. 1997).

Many other studies aimed at assessing the correlation between pudendal neuropathy and symptoms of anal incontinence after delivery have been published (Sultan et al. 1994b, Tetzschner et al. 1997, Frudinger et al. 1999).

Sultan et al. (1994b) showed in a prospective study of 128 women during pregnancy and after delivery that PNTML was slightly prolonged after delivery, indicating a degree of nerve damage. Sixteen per cent of the women had a prolonged PNTML after delivery, but in only one third of these did it remain prolonged at 6 months. They suggest that nerve injury manifested as prolonged pudendal latency may result in anal incontinence later in life and caesarean section may not protect such patients from nerve damage.

Tetzschner et al. (1997) showed that PNTML does not increase significantly during pregnancy but does increase significantly after delivery, while Frudinger et al. (1999) showed in a prospective observational study that women without any apparent sphincter trauma can undergo generalized anal canal trauma during vaginal delivery (anterior thinning and lateral thickening), which can lead to symptoms of anal incontinence.

The recently developed sacral nerve stimulation (SNS) technique has given promising results in the treatment of idiopathic anal incontinence (Malouf et al. 2000a).

5.2. Non-obstetric causes of anal incontinence

5.2.1. Incontinence with normal anal sphincters

Severe chronic diarrhoea can lead to symptoms of fecal incontinence. The causes of diarrhoeal states are infectious, inflammatory bowel disease, short-gut syndrome, laxative abuse or radiation enteritis. Fecal impaction and overflow incontinence occurs as a result of anatomical or functional outlet obstruction. Poor rectal compliance (inflammatory bowel disease, rectal tumours, outside compression) can also lead to incontinence, as can fistula, dementia or learning impairment (Madoff et al. 1992, Mavrantonis and Wexner 1998).

5.2.2. Incontinence with abnormal sphincters

Anal incontinence can be the cause or effect of direct anal sphincter traumas after anorectal surgery (anal-fistula surgery or complications after haemorroidectomy) and accidental injuries.

Sphincter neuropathy can lead to anal incontinence and abnormal function at any level of the nervous system. Upper motor neuron lesions are cerebral (tumour, stroke, trauma) or spinal (demyelization, tumour), while lower motor neuron lesions can be caused by cauda equine lesions, diabetes, pelvic tumour or demyelization (Madoff et al. 1992, Mavrantonis and Wexner 1998).

5.2.3. Other reasons

Rectal prolapse and / or the descending perineum syndrome can be found to lie behind symptoms of anal incontinence. These symptoms can also be attributed to congenital anorectal malformations, although these are rare (Madoff et al. 1992).

6. Risk factors for anal sphincter rupture during childbirth

The risk factors for anal sphincter rupture during delivery have been shown to include primiparous status, instrumental delivery (forceps-assisted delivery or vacuum extraction), high birth weight (more than 4 kg), episiotomy, occipitoposterior presentation and prolonged second stage of labour.

6.1. Instrumental delivery

In earlier studies forceps delivery was considered a major risk factor for anal sphincter rupture during vaginal delivery (Sultan et al. 1993a, 1994a, Walsh et al. 1996, Riegel et al. 1998, Donnelly et al. 1998, Varma et al. 1999, Groutz et al. 1999, Abramowitz et al. 2000, Belmonte-Montes et al. 2001, de Leeuw et al. 2001b, Riskin-Mashiah et al. 2002). MacArthur et al. (1997) found that 4 per cent developed new symptoms of fecal incontinence after childbirth, whereas forceps and vacuum extraction were the only independent risk factors. In a prospective study, Donnelly and colleagues (1998) found that instrumental vaginal delivery was associated with an 8.1-fold risk of anal sphincter injury. In a large study by de Leeuw et al. (2001b), the relative risk of anal sphincter rupture if forceps were used was 2.73 per cent.

There are also some opposite results. In a recent study, de Parades et al. (2004) showed that in experienced hands, forceps delivery cannot be considered to be as important a risk factor as was suggested earlier: anal sphincter injury was detected in less than 13 per cent of the patients with previous forceps delivery.

Vacuum extraction has also proved to be a risk factor for anal sphincter rupture and symptoms of anal incontinence (Macarthur et al. 1997, Groutz et al. 1999, de Leeuw et al. 2001b). According to de Leeuw et al. (2001b), the relative risk of anal sphincter rupture if vacuum extraction was used was 1.79 per cent. In a prospective study by Groutz et al. (1999), the incidence of anal incontinence three months after delivery was seven per cent, of which 0.7 per cent had incontinence to solid feces. In that study the incidence of postpartum anal incontinence was significantly higher after vacuum extraction than after normal spontaneous delivery (25 per cent versus 3.8 per cent, respectively).

6.2. Primiparous status

A first vaginal delivery has been shown to be a risk factor for anal sphincter rupture. Sultan et al. (1993a) found that up to 35 per cent of primiparous women had an anal sphincter defect visible in the EAUS examination after delivery and 13 per cent of these women became symptomatic. Many studies support the idea that primiparous status is a risk factor for anal sphincter rupture during delivery and for symptoms of anal incontinence afterwards (Bek and Laurberg 1992, Sorensen et al. 1993, Walsh et al. 1996, Varma et al. 1999, Belmonte-Montes et al. 2001, Nazir et al. 2002, Riskin-Mashiah et al. 2002, Pollack et al. 2004).

6.3. Episiotomy

The role of mediolateral episiotomy in protecting the patient from anal sphincter rupture is still unknown. Mediolateral episiotomy has proved to be a risk factor for anal sphincter rupture in some studies (Bek and Laurberg 1992, Henriksen et al. 1992, Sultan et al. 1993a, Walsh et al. 1996, Groutz et al. 1999), but there are also data suggesting the opposite (Poen et al. 1997, de Leeuw et al. 2001b). Walsh et al. (1996) found that episiotomy did not prevent anal sphincter tears, as 74 per cent of the women with a tear had had episiotomy as compared with 28 per cent of those without a tear.

Compared with mediolateral episiotomy, midline episiotomy is associated with an increased risk of sphincter damage (Coats et al. 1980, Klein et al. 1994, Riskin-Mashiah at al. 2002). If episiotomy is necessary, mediolateral episiotomy is therefore recommended.

6.4. Other risk factors

High fetal birth weight and long duration of the second stage of delivery were associated with an elevated risk of anal sphincter rupture (de Leeuw et al. 2001b), while other risk factors have been found to include occipitoposterior presentation (Sultan et al. 1994a, Fitzpatric et al. 2001), prolongation of the second stage of labour by epidural analgesia (Donnelly et al. 1998, Groutz et al. 1999) and large infant head circumference (Nazir et al. 2002). Riskin-Mashiah et al. (2002) has recently suggested on the basis of a retrospective study that the use of pudendal block analgesia may also be a risk factor for severe tears during delivery.

7. Classification of symptoms of anal incontinence

Various classifications can be used for grading the symptoms of anal incontinence. These establish the degree and frequency of incontinence and help assess the patient's quality of life. The difficulty of symptoms of anal incontinence depends on the patient's situation and subjective experiences, so that incontinence may mean a great deal of embarrassment and psychological suffering (in social situations) to some women. There are some quality of life scales that have been shown in psychometric evaluations to be both reliable and valid (Rockwood et al. 2000).

A scoring system for symptoms of anal incontinence allows objective evaluation of the functioning of the anal sphincters and is also an indicator of the severity of incontinence, permitting comparison with other series. A number of scales have been published: Browning and Parks (1983), Pescatori et al. (1992), Jorge and Wexner (1993a) and American Medical Systems (AMS).

According to Parks' classification, gradus I implies normal continence, gradus II incontinence to flatus, gradus III incontinence to flatus and liquid stools, and gradus IV incontinence to solid stools (Hardcastle and Parks 1970).

Jorge and Wexner (1993a) developed the first system that took account of the use of pads and life-style alterations, so that it has higher clinical applicability than Parks' scale. Scores in this system vary between 0 and 20, a score of 0 corresponding to normal continence and a score of 20 to total anal incontinence.

In a prospective study, Vaizey et al. (1999b) showed that the existing scales for the assessment of fecal incontinence (Pescatori, Wexner and AMS) correlated well with the clinical impression of severity, but they also developed a new scale which also takes into account the need for constipation medicine and the lack of ability to defer defecation for 15 minutes.

8. Diagnostic evaluation of anal incontinence

8.1. Anamnesis

A careful clinical history is essential. Other potential causes of incontinence should be excluded, previous vaginal deliveries, anoperineal surgery or possible systematic diseases have to be asked about, and it is important to establish the degree and frequency of incontinence and its effect on quality of life. The use of pads and the need to change underwear are also indicators of the severity of incontinence. The use of a standard questionnaire (e.g. Wexner's classification) will ensure that all the necessary data are taken into consideration.

8.2. Clinical examination

An examination of the perineum will identify scars and allow assessment of the anal margin. Possible perineal soiling should be noted. Patients should be asked to strain in order to evaluate the presence of perineal descent, rectocele, or cystocele. The physician should perform a digital examination when the patient is both resting and squeezing, which is necessary for estimating possible sphincter defects and the functioning of the anal sphincters. If an organic condition such as neoplasia or proctitis is suspected, sigmoidoscopy should be carried out.

8.3. Endoanal ultrasound examination

EAUS is the accepted gold standard for the examination of anal sphincter anatomy and it is a routine examination in patients with anal incontinence. It is easy to carry out, requires no preparation of the patient and causes minimal discomfort. EAUS is performed with a rotating 360° endoprobe 1.7 cm in diameter (Bartram and Sultan 1995). The probe is covered by a hard plastic cone filled with degassed water and is withdrawn downwards in the anal canal so that pictures can be obtained. This provides a clear image of the IAS and EAS, enabling structural abnormalities in either muscle to be detected.

An EAUS examination is also useful in the follow-up of primary sutured sphincter ruptures (Nielsen et al. 1992). The IAS is well visualized in EAUS, because the hypoechogenic smooth muscle is clearly differentiated from the echogenic subepithelial tissues medially and the longitudinal muscle laterally. In contrast, the EAS is of mixed and variable echogenicity, so that its boundaries are more difficult to define. Sphincter defects are seen as breaks in the normal texture of these muscle rings. Several studies have confirmed the value of EAUS for detecting sphincter defects in cases of anal incontinence (Felt-Bersma et al. 1992, Cuesta et al. 1992, Nielsen et al. 1993, Falk et al. 1994, Vaizey et al. 1997, Karoui et al. 1999, Buhr and Kroesen 2003).

The development of three-dimensional ultrasound has clarified the EAUS anatomy of the anal sphincters (Gold et al. 1999), as it enables the EAS can be distinguished better from other closely related structures, and once acquired, the data can be reviewed later. It also allows imaging of the sphincter in the coronal plane. In a recent comparison of three-dimensional EAUS with endoanal MRI, Williams et al. (2002) suggested that it should be possible to recognize EAS atrophy in EAUS, which may be clinically important in assessing a patient's suitability for sphincter repair.

8.4. Anal manometry

The standard manometric evaluation enables measurement of the maximal and mean resting and squeeze pressures and the length of the anal canal (HPZ, high pressure) (Jorge and Wexner 1993b). A low resting pressure indicates IAS dysfunction, whereas a low voluntary contraction (squeeze) pressure indicates EAS dysfunction. Unconscious fecal soiling may be a result of low resting anal canal pressure (passive incontinence) (Law et al. 1991). Urge incontinence may be the result of rupture of the EAS.

A correlation between functioning of the EAS and squeeze pressure has been reported previously by Frenckner et al. (1975, 1976) and Sultan et al. (1993a), although there are also studies in which no such a correlation has been established (Nielsen et al. 1992, Nazir et al. 2002).

The most widely used method is measurement using a multichannel catheter perfused with water (Perdersen and Christiansen 1989). In the case of total anal sphincter rupture after vaginal delivery, the resting and squeeze pressures are usually reduced (Fynes et al. 2000, Jorge and Habr-Gama 2000, Damon et al. 2002). Williams et al. (2001) showed recently that an EAS defect may be associated with a significant decrease in squeeze pressure and an increase in the incontinence score.

8.5. Electromyography

Before the advent of EAUS, concentric-needle electromyography (EMG) was used to detect EAS defects (Swash M 1985, Wexner et al. 1991b). EMG is used for sphincter mapping, especially in patients with an ectopic anus, congenital anomalies or severe disruption (traumatic or obstetric injury), and to assess the degree of denervation by means of measurements of fibre density and evidence of conduction defects in terms of nerve latency. This assessment also provides a measure of striated muscle function, which can be used in biofeedback retraining.

The EMG examination is painful and laborious to do, and has nowadays been replaced by EAUS for diagnosing anal sphincter defects (Tjandra et al. 1993). EAUS abnormalities have been shown to correlate with electrical defects in EMG (Law et al. 1991, Cuesta et al. 1992, Nielsen et al. 1993).

8.6. Pudendal nerve terminal motor latency measurements

The measurement of PNTML is an assessment of pudendal nerve function. Damage to this nerve is one of the reasons for anal incontinence. PNTML is a measure of the length of time required for a fixed electric stimulus to travel along the pudendal nerve between the ipsilateral ischial spine and the anal verge. The normal latency is considered to be 2.1+- 0.2ms. PNTML can be measured transrectally using a disposable pudendal nerve stimulator mounted over a glove on the index finger. The electrode at the tip of the finger stimulates the pudendal nerve at the level of the ischial spine, while the electrodes at the base of the finger record the action muscle potential of the external anal sphincter (Snooks et al. 1985, Rogers et al. 1988). Pudendal nerve latency time and perineal descent increase with age.

Sultan et al. (1994b) found that vaginal delivery, particularly the first, can lead to pudendal nerve damage and stretching of the pelvic floor tissue. Caesarean section does not always protect the patient from this. According to some studies, a prolonged value is a prognostic indicator of poor long-term function after surgery (Laurberg et al. 1988, Tetzschner et al. 1995). Normal latencies do not exclude nerve damage, because only the fastest conducting fibres are recorded. Also, anal canal sensation has been shown to be impaired in women with anal sphincter damage at delivery (Cornes et al. 1991).

8.7. Magnetic resonance imaging

8.7.1. Endoanal magnetic resonance imaging

The recently developed technique of endoanal MRI with an endoanal coil allows detailed visualization of the normal anatomy and pathologic conditions of the anal sphincters (deSouza et al. 1995a, Hussain et al. 1995). Aroson et al. (1990) were the first to quantify the anal sphincter muscles by MRI.

This imaging technique accurately defines the site and extent of sphincter tears. The hyperintense IAS appears as a continuation of the smooth muscle of the rectum, while the hypointense EAS surrounds the lower part of the IAS. The puborectal muscle swings around the upper part of the external sphincter and is continued cranially by the levator ani muscle. A sphincter defect is seen as a discontinuity in the muscle ring.

MRI also enables the detection of EAS atrophy (deSouza et al. 1995b, 1996, Briel et al. 1999, Beets-Tan et al. 2001), and initial work suggests that endoanal MRI may be superior to EAUS for the diagnosis of anal sphincter defects (deSouza et al. 1996, Rociu et al. 1999b).

The main disadvantages of this method are the fact that its use has been restricted to specialized centres because the required endoanal coil is not yet available with every MR machine, the time required by examination and its cost. In their prospective study, Malouf et al. (2000b) found that EAUS and endoanal MRI are equal in diagnosing EAS defects but MRI is inferior in diagnosing IAS defects.

8.7.2. Endovaginal magnetic resonance imaging

Tan et al. (1998) have shown that the anatomy of the female pelvic floor and urethra can be clearly demonstrated by endovaginal MRI. Ten healthy nulliparous volunteers underwent MRI with an endovaginal coil and the findings were correlated with those from EVMRI and cross-sectional anatomical slices obtained from three cadavers. Another study by Tan et al. (1997) showed that, compared with the body coil, endovaginal MRI is excellent for demonstrating the anatomy of the pelvic floor and urethra.

9. Treatment

The treatment of anal incontinence should always be directed at the cause. Many individuals can be treated adequately by conservative therapy, but surgical treatment should be offered to patients with EAS rupture.

9.1. Conservative

Conservative treatment should be used as the first and second lines of therapy, and it can also be used as an adjunct to surgical treatment (Norton et al. 2003). Idiopathic degeneration of the IAS has recently been described (Vaizey et al. 1997), and this may be one of the most common causes of anal incontinence in the community. Defects of the IAS are not amenable to direct surgical repair and are usually treated non-surgically (Leroi et al. 1997). The majority of these patients can be managed conservatively with pads, dietary modification, biofeedback, sphincter exercises, an anal plug and /or electrical sphincter stimulation, yielding satisfactory results.

9.1.1. Dietary manipulation and fibre supplements

In dietary manipulation, fibre supplements are often prescribed in an attempt to increase stool bulk. This may lead to an increase in stool volume, however, and thereby to further episodes of incontinence. Others recommend patients to reduce dietary fibre intake in order to produce a smaller and firmer stool. No published controlled data exist to justify either approach. Caffeine stimulates colonic motility and may worsen the symptoms of incontinence (Cheetham et al. 2001).

9.1.2. Medical

Antidiarrhoeal medicines such as loperamide and codeine phosphate have a constipating effect, and these are valuable agents for the management of anal incontinence. Loperamide is the favoured drug because of its low side-effect profile (Gattuso and Kamm, 1994). It reduces stool weight and small and large bowel motility while increasing IAS tone to a minimal extent (Sun et al. 1997). Only small doses (1-4 mg daily) are titrated according to the response in terms of clinical symptoms. Codeine phosphate is used in the same way, but its usefulness may sometimes be limited by its side effects. A combination of loperamide and codeine phosphate can be used in resistant cases.

There may be an association between the menopause and the onset of anal incontinence. According to a pilot study by Donnely et al. (1997), hormone replacement therapy may alleviate the symptoms of anal incontinence, but this needs further investigation. Further help can be obtained by maintaining an empty rectum with the use of regular suppositories (such as those containing glycerin) or enemas (sodium citrate or, occasionally, sodium phosphate) and executing planned bowel movements with laxatives.

9.1.3. Biofeedback therapy

Biofeedback therapy involves the use of an auditory or visual representation of a biological measurement (anal canal pressure). Electrical stimulation can be used either alone or as an adjunct to biofeedback therapy. An electrode is inserted into the anal canal to stimulate the anal sphincters (Pescatori et al. 1991).

The results of biofeedback therapy depend on factors such as motivation, ability to understand instructions, the presence of some rectal sensation and the ability to contract the EAS voluntarily. In practice, biofeedback is only part of a package of care which includes dietary advice, sphincter exercises and careful titration of antidiarrhoeal agents. There is no strong evidence for the effect of biofeedback therapy, as there are studies supporting its effectiveness and studies suggesting the opposite.

In a prospective study by Norton and Kamm (1999), two thirds of the cases had been subjectively cured or had improved by the end of the treatment, patients with intact sphincters being the most likely to benefit. Patients with urge incontinence alone fared better than those with passive incontinence alone (55 vs. 23 per cent). Jensen and Lowry (1997) found that biofeedback improves the functional outcome after sphincter repair and is a reasonable option for patients with a less than optimal outcome after sphincter repair alone, while Kairaluoma et al. (2004) support this idea and also suggest that biofeedback does not improve the symptoms in the case of idiopathic incontinence. A prospective, randomized study by Fynes et al. (1999b) proved that augmented biofeedback is superior to sensory biofeedback alone for the treatment of impaired fecal incontinence after obstetric trauma. By contrast, a recent randomized controlled trial on the usefulness of biofeedback for treating fecal incontinence showed that it is not superior to standard care supplemented with advice and education (Norton et al. 2003).

9.1.4. Anal plug

An anal plug can be used with patients who are not suitable for surgery or when surgery has not been of help in the treatment of symptoms of anal incontinence (Mortensen and Humphreys 1991, Christiansen and Roed-Petersen 1993, Norton and Kamm 2001). It is inserted into the anal canal, after which it expands, creating a watertight seal. The results are reasonable in those patients who tolerate the plug without complications. Failures tend to occur because of either discomfort or inability to retain the device. The anal plug has been useful in the case of patients with neurological disease and/or patients with impaired anorectal sensation (pudendal neuropathy).

9.2. Operative treatment

Surgical reconstruction is usually performed if an EAS defect can be shown in the EAUS examination (Browning and Motson 1984, Christiansen and Pedersen 1987a, Wexner et al. 1991a).

The results of the operation depend on the extent to which the remaining sphincter retains the ability to contract. Primary sphincter repair is done soon after delivery if a third or fourth-degree anal sphincter tear is found. The most recent studies suggest that the overlapping technique is better than the end-to-end technique. Afterwards, a secondary or delayed anterior anal sphincter repair can be performed using the overlapping technique.

9.2.1. Primary anal sphincter repair

Primary anal sphincter repair operation is used immediately after traumatic delivery, when anal sphincter injury is detected. The most common type of repair is an endto-end approximation. Two or three figure-of-eight sutures can be inserted into the approximate torn ends of the sphincter. According to earlier studies, the results of the operation are not good enough (Sultan et al. 1994a, Poen et al. 1998, Kammerer-Doak et al. 1999, Zetterström et al. 2003, Davis et al. 2003), possibly because it is often performed by junior medical staff with an insufficient understanding of the anal anatomy. The operation is often difficult because of swollen tissue, bleeding and contamination with liquid or solid feces, which makes it difficult to identify the muscle.

Sultan et al. (1999) have reported an improved outcome using an overlapping technique for primary sphincter repair. In a randomized study by Fitzpatrick et al. (2000), end-to end approximation and the overlap technique were found to be equal and the symptomatic outcome was good, although residual anal sphincter defects

were found in EAUS in two-thirds of the patients irrespective of the method. Kairaluoma et al. (2004), in their recent report of results obtained with the overlap technique after anal sphincter rupture, maintain that the median outcome was good in the case of an experienced operator, 77 per cent of patients having no subsequent symptoms of anal incontinence and the overlap of the EAS being found in EAUS examination in up to 97 per cent of cases.

More randomized controlled studies are needed to compare these two surgical methods.

There is no evidence as to the best suture material for sphincter repair, but monofilament materials such as polydioxanone (PDS) are often used because of their long half-life (Sultan et al. 1999). A prophylactic antibiotic is recommended, but covering colostomy is not usually necessary (Fernando et al. 2002). Laxatives are usually used to soften the stool. The most common complication after surgery is wound infection, and fistulas (rectovaginal, anovaginal) can sometimes be seen afterwards (Sultan et al. 1994a).

Some authors have demonstrated that there may be a role for a colorectal team in the management of acute severe vaginal tears if these involve the rectal or anal mucosa (Cooke et al. 1999).

9.2.2. Secondary overlapping anal sphincter repair

Secondary or delayed overlapping sphincter repair is the operation of choice for patients with anal sphincter rupture and symptoms of anal incontinence. This technique was originally described by Parks and McPartlin (1971) and later modified by Slade et al. (1977). It has since become widely accepted.

In this technique the scar tissue is preserved and dissection continued back until a viable EAS is identified. The overlap is created with the fibromuscular ends. Suturation of the IAS may be performed separately, but this is often difficult, due to fragmentation of the muscle. Covering colostomy is not needed. Prophylactic antibiotics are used, but bowel preparation before surgery is not necessary. The most common complication after surgery is wound infection. Good results imply continence to solid and liquid stools, but the control of flatus is more difficult.

Short-term success rates for the operation vary between 47 and 90 per cent (Parks and McPartlin 1971, Browning and Motson 1984, Fang et al. 1984, Christiansen and Pedersen 1987a, Pezim et al. 1987, Laurberg et al. 1988, Wexner et al. 1991a, Fleshman et al. 1991, Engel et al. 1994, Sitzler and Thomson 1996, Felt-Bersma et al. 1996, Kammerrer-Doak et al. 1998), but long-term results have not proved to be satisfactory, and total continence cannot usually be achieved (Karoui et al. 2000, Malouf et al. 2000c).

Poor results of the operation are often related to prolonged PNTML, the length of the preoperative symptoms, gross perineal descent, obesity and increased age (Laurberg et al. 1988, Engel et al.1994, Nikiteas et al. 1996, Gilliland et al. 1998). In contrast, Simmang et al. (1994) and Oliveira et al. (1996) suggest that anal sphincter reconstruction can be performed on elderly patients with improvements in the majority of cases.

Rociu et al. (1999a) indicate that EAS atrophy (seen in endoanal MRI) could be predictive of a poor outcome of anterior anal sphincter repair. Patients with a poor outcome may experience a significant improvement after repeat anal sphincter repair (Pinedo et al. 1999).

9.2.3. Other surgical methods

Post-anal repair was proposed earlier as a standard method for treating idiopathic anal incontinence (Parks AG 1975, Keighley and Fielding 1983, Browning and Parks 1983). Because of the theoretical importance of an adequate anorectal angle in the maintenance of continence, Parks et al. (1966) devised an operation to restore this angle in incontinent patients. Posterior levatorplasty was thought to improve continence both by turning the anorectal angle upwards and forwards and by lengthening the anal canal. The short-term results were promising (Keighley and Fielding 1983, Browning and Parks 1983) but the long-term results were poor (Orrom et al. 1991).

External anal sphincter repair combined with anterior levatorplasty was designed to improve EAS function and lengthen the functional anal canal. The results were satisfactory, up to 71 per cent of the patients in the traumatic group being satisfied and 62 per cent in the idiopathic group (Miller et al. 1989). A recent study has shown that anterior levatorplasty combined with EAS plication reduced the symptoms of incontinence in the majority of the patients, but complete continence was seldom achieved in treatment of patients with either idiopathic or traumatic anal incontinence (Aitola et al. 2000).

Total pelvic floor repair, performed occasionally on patients with idiopathic incontinence in earlier times, includes both anterior and posterior levatorplasty. The long-term results in cases of obstetric neuropathic anal incontinence published by Korsgen et al. (1997) showed a great improvement in symptoms in 49 per cent of cases and a mild improvement in 23 per cent.

9.2.4. Dynamic graciloplasty

The gracilis muscle is the most superficial adductor on the medial side of the thigh, but it has no important function in humans. The possibility of using this muscle for sphincterplasty was first explored by Pickrell in 1952 to treat children with neurogenic fecal incontinence (spina bifida, meningocele). Because of the superficial position of the gracilis muscle and the proximal neurovascular supply, it is easily accessible and can be freely dissected distally without damage.

The technique involves wrapping the gracilis muscle around the anal canal and suturing it to the contralateral ischial tuberosity. Segments of the gluteus maximus may also be used, and successful results associated with increased squeeze pressures have been reported. Stimulation of the gracilis muscle by electrons to convert the muscle fibres from type II fast-twitch fibres into fatigue-resistant type I slow-twitch fibres may improve the outcome. This technique, in which the electrode is fixed directly on the nerve, was developed independently by Williams et al. (1991). The indications for a dynamic graciloplasty are a residual EAS defect after sphincter reconstruction, severe neurological damage and congenital disease such as anal atresia or spina bifida. The contraindications are inflammatory bowel disease and poor motivation or physical or mental incapacity.

Dynamic graciloplasty is a technically demanding and expensive operation involving a high morbidity rate and also mortality. The results range from 42 to 85 per cent achieving satisfactory continence, but unfortunately most studies report success rates of 60 per cent or less (Williams et al. 1991, Baeten et al. 1995, 2000, Chapman et al. 2002).

9.2.5. Artificial bowel sphincter

Artificial sphincter implants have been used for urinary incontinence for many years. Christiansen and Lorentzen (1987b) have reported a successful outcome with the device in a man with neurogenic fecal incontinence. The elements of the device are an inflatable cuff with a pressure-regulating balloon and a pump.

The indications and contraindications for the use of this technique are the same as those described for stimulated graciloplasty.

Associated morbidity is high, the presence of complications ranging between 31 and 43 per cent (Lehur et al. 1996, 2000, Wong et al. 1996, Vaizey et al. 1998). An artificial bowel sphincter is easy to implant, but removal has been necessary in about 20 per cent of cases owing to pressure necrosis or infection.

The early results were encouraging, with success in 50 to 71 per cent of cases (Christiansen and Sparso 1992, Lehur et al. 1996, 2000, Wong et al. 1996, Vaizey et al. 1998), but a multi-centre prospective, non-randomized clinical trial to evaluate the safety, efficacy and impact on quality of life carried out recently by Wong et al. (2002) showed that morbidity and the need for revisional surgery were high, up to 25 per cent, although a satisfactory outcome was achieved in 85 per cent of cases with a functioning device. Thus an artificial bowel sphincter can improve symptoms of anal incontinence and quality of life in selected patients with fecal incontinence.

9.2.6. Sacral nerve stimulation

Permanent sacral nerve stimulation (SNS) is a new method to treat symptoms of anal incontinence. SNS can be offered to patients with primary degeneration and weakness of the anal sphincters and pelvic floor damage, spinal injury or some other neurological disorder. Permanent SNS has also been used recently after failed sphincter repair, whereas the treatment options in this group had previously been limited (Malouf et al. 2000a, Kenefick et al. 2002).

Sacral nerve stimulation is a minimally invasive surgical technique that uses lowlevel electrical stimulation applied via electrodes though the sacral foramina to the nerves of the sacral plexus. This technique has been used safely and successfully for years to treat urinary disorders (Tanagho and Schmidt 1988).

The major advantages of the technique are that temporary percutaneous stimulation can be performed under local anaesthesia and that it is an accurate predictor

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of success before surgical intervention. There is an effect on rectal and IAS smooth muscle activity in addition to the facilitation of EAS striated muscle function. Implantation of a permanent sacral nerve stimulator is a relatively minor procedure, especially compared with the alternative surgical methods. The use of SNS is indicated in patients who have failed with maximum conservative therapies and who have severe incontinence.

Matzel et al. (1995) were the first to report that this method can also be used for patients with symptoms of anal incontinence, and preliminary reports showed promising results (Matzel et al. 1995, Vaizey et al. 1999a, Malouf et al. 2000a, Rosen et al. 2001, Kenefick et al. 2002).

Recently, Matzel et al. (2004) found in a multi-centre prospective trial (n=37) that SNS greatly improved continence and the quality of life in selected patients with morphologically intact or repaired anal sphincters.

9.2.7. Colostomy

A permanent end colostomy may be indicated if none of the above treatment modalities is successful. Although considered as the last option in the surgical strategy, many find that a colostomy can be more easily managed than an incontinent anal sphincter, allowing a dramatic improvement in quality of life.

PRESENT RESEARCH

The present research was undertaken to obtain more information about anal incontinence in women with previous vaginal delivery. The following specific questions were addressed:

- 1. What are the results of delayed sphincter repair for obstetric ruptures, and what are the reasons for failure?
- 2. Are the results of primary sphincter repair good enough?
- 3. What is the incidence of anal sphincter rupture and anal incontinence after the first vaginal delivery?
- 4. Which is better for diagnosing anal sphincter rupture in the case of anal incontinence, endoanal US or endovaginal MRI?

PATIENTS

The present work was carried out during the years 1998 to 2004 at the Department of Surgery, Division of Gastroenterology, Helsinki University Central Hospital, and between the years 2001 and 2004 at the Department of Surgery, Division of Gastroenterology, Seinäjoki Central Hospital. All the patients included were examined at Helsinki University Central Hospital during the years 1998 to 2000, while the analysis of the data and writing of the papers took place at Seinäjoki Central Hospital between 2001 and 2004. The research was done in co-operation with the Departments of Gynecology and Radiology at Helsinki University Central Hospital. The population was limited to cases of obstetric anal incontinence. The patients and methods are shown in Table 1.

	patients	controls	methods
Paper I	39	-	results of anterior overlapping sphincter repair, retrospective study
Paper II	52	51	results of primary sphincter repair, retrospective study
Paper III	75	24	prospective study be- fore and after the first delivery vaginal group vs. sec- tion group
Paper IV	19	-	prospective study, en- doanal US vs. EVMRI for diagnosing anal sphincter defects

Table 1. Patients and methods.

1. Patient selection

Paper I

To assess the results of anterior overlapping sphincter repair in women with a history of childbirth. A total of 39 women who had been operated on between 1990 and

1999 were invited by letter for a clinical control examination, interview, EAUS and PNTML measurement. The clinical data were retrieved from the patient records.

The median number of deliveries was 2 (range 2 to 5), and the symptoms of anal incontinence had started directly after delivery in 19 cases and some years later in 20. Nine patients had been incontinent for gas or liquids and 30 for feces. The median duration of incontinence before repair had been 7.5 years (range 0.5 to 40 years) and the median age 53 years (26 to 79 years). The median follow-up time was 22 months (2 to 99 months).

The symptoms of anal incontinence were graded using Parks' classification, and patients were asked to fill in a written questionnaire concerned with the subjective functional results of the operation. The questionnaire results were classified as good if the patients were fully continent, acceptable if they were incontinent for gas and/or liquids and poor if they had fecal incontinence and had to use a pad permanently.

Paper II

The results of primary sphincter repair were assessed in a total of 52 women with clinically diagnosed anal sphincter defects after vaginal delivery during years 1988 to 1999. Thirty-two of them were found from the hospital records and were invited by letter to participate. Thirty were willing to do so. The remaining 22 women were sent to a Department of Surgery at some other hospital for re-examination. Seven of the population had grade IV perineal laceration and the remaining 45 grade III.

The median timing of the clinical interview and examination, EAUS, anal manometry and a PNTML measurement was 15 months (2-144) after primary sphincter repair. Details of the labour were obtained later from the delivery records.

The control group consisted of 51 primiparous women without clinically detectable perineal lacerations who had had their first vaginal delivery between December 1998 and December 1999. There had been 48 cases of normal vertex presentation and 3 of abnormal occipitoposterior presentation (6 per cent). A clinical examination, EAUS and anal manometry were performed after a median interval of 16 weeks (8-34) from delivery. The median (range) age of the control group was 30 years (21-40).

Paper III

A prospective study to discover the incidence of an occult anal sphincter defect and symptoms of anal incontinence before and after a first vaginal delivery was performed between December 1998 and July 2000. A total of 107 women were selected on the grounds of nulliparous status, pregnancy and willingness to participate. Letters of invitation were sent only to women living in the urban area of Helsinki. Ten per cent of the recipients agreed to participate.

A clinical interview and examination, anal manometry and EAUS were performed 4 weeks (mean, range from 0 to 6 weeks) before delivery and 16 weeks (8-36) after delivery. Details of the labour were obtained later from the delivery records.

Out of the initial population of 107, eight women did not come to the examination after delivery and were excluded from the analysis, and thus the final population consisted of 99 primiparous women with a mean age of 31 years (21-41). The mean birth weight of the infants was 3 548g (2 400-4 430). Seventy-five women (76%) had a vaginal delivery, the mean gestational age being 40.4 weeks (36-42). Mediolateral episiotomy was performed in 70 cases, and vacuum extraction was necessary in 20 cases. There were no forceps-assisted deliveries.

Twenty-four of the women (24%) had a caesarean section, and these served as a control group. Five of them had an elective section before the onset of labour and the others had a caesarean section during labour. The extent of cervical dilatation at the time of the caesarean section ranged from 3 cm to full dilatation (mean 6.8 cm).

Paper IV

To evaluate the possibility of using a prostatic coil endovaginally for detecting anal sphincter defects, we assessed the value of preoperative EAUS and EVMRI examinations for detecting such defects in individuals with fecal incontinence, validated against observations at operation. The decision to operate was made on the basis of the medical history, a physical examination and EAUS findings. A total of nineteen patients with a median age of 32 years (range 26-56) who had undergone surgical repair for anal incontinence between April 1999 and December 2000 were included. The background to anal incontinence was childbirth in 18 cases and anorectal surgery in one.

A clinical interview and examination, EAUS and EVMRI had been performed before the operation, the average interval between the EAUS and EVMR examinations being 2 months (range 1 day to 6 months) and that between EVMRI and the operation 8 months (range 1 day to 13 months).

2. Ethical approval

The research was approved by the Ethical Committees of the Departments of Surgery, Obstetrics and Gynaecology and Radiology at Helsinki University Central Hospital. All the patients gave their informed consent and participated voluntarily.

METHODS

1. Clinical data

The clinical data were obtained from the patients' records. Those in Paper I consisted of the age of the patient, body mass index (BMI), obstetric history, duration of incontinence, preoperative incontinence score, preoperative signs of perineal descent, operative findings and duration of follow-up. In Paper II they included details of the labour (number of previous deliveries, gestational age, duration of labour and delivery, mode of delivery and birth weight), in Paper III other details of the labour such as the use of vacuum extraction and clinically diagnosed perineum and anal sphincter ruptures were also recorded, and in Paper IV they consisted of age, obstetric history, duration of incontinence and preoperative incontinence score.

2. Symptoms of anal incontinence

Symptoms of anal incontinence were graded using both Parks' and Wexner's classifications. Wexner's scores (Jorge and Wexner 1993a) vary between 0, normal continence, and 20, total anal incontinence (Table 2).

Type of incontinence	Never	Rarely	Sometimes	Usually	Always
Solid	0	1	2	3	4
Liquid	0	1	2	3	4
Gas	0	1	2	3	4
Wears pad	0	1	2	3	4
Lifestyle alteration	0	1	2	3	4

Table 2. Continence grading scale according to Jorge and Wexner.

0=perfect; 20= complete incontinence.

Rarely < 1/month; 1/month < Sometimes < 1/week; 1/week < Usually < 1/day; Always > 1/day.

3. Anal manometry

Anal manometry was performed using the Medtronic Polygram program® (Medtronic, Copenhagen Denmark) in the left lateral position, using the 'pull-through' technique with a water-perfused 4 mm catheter according to the standard procedure described previously (Pedersen and Christiansen 1989). The mean resting and squeeze pressures were measured. The results were analysed by computer.

4. EAUS

The EAUS examination was performed in the left lateral position using a B&K Medical® (Mileparken 34, 2730 Herlev, Denmark) ultrasound scanner with a 10 MHz rotating endoprobe, covered with a hard plastic cone filled with degassed water. This was withdrawn downwards in the anal canal and three pictures were obtained (Bartram and Sultan 1995).

An EAS defect was seen as a break in the normal texture of the muscle ring. EAUS findings were classified into three categories: normal (Figure 2), partial rupture and total rupture (Figure 3). If there was a clear discontinuity in the EAS but the sphincter ends were close to each other, the rupture was classified as partial. If there was a marked discontinuity (at least one quarter) in the EAS muscle, the rupture was classified as total. Any break in the internal anal sphincter (IAS) muscle observed in the EAUS examination was considered abnormal. The same person (TP) performed all the EAUS examinations.

Figure 2. Normal anal sphincters in an EAUS examination.

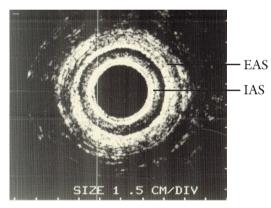


Figure 3. EAS and the IAS ruptures in the EAUS examination (indicated by arrowheads).



5. PNTML

PNTML was measured on both sides using a St Marks' electrode® (Medtronic-Dantec) and EMG equipment (Medtronic-Dantec, Tonsbakken 16-18, DK 2740 Skuvlunde, Denmark). The normal value for the latency was less than 2.3 ms and that for the amplitude of the motor response higher than 0.1 mV (Swash M 1985). The same person (TS) performed all the PNTML measurements.

6. EVMRI

EVMRI was performed with a 1.5. Tesla machine (Siemens Vision, Erlangen, Germany) using the parameters shown in Table 3. A prostatic coil (MedRad) was inserted endovaginally, directed towards the sphincters (backwards). No bowel preparation was used. To simulate the situation in EAUS in terms of sphincter distension, a 10-mm rectal catheter was inserted into the anus.

All the patients received an i.v. injection of gadolinium (Magnevist) 0.1 mmol/kg. EVMRI was performed altogether on 52 patients.

The condition of the EAS was classified as 'total rupture', 'partial rupture' or 'normal', and the condition of the IAS as 'rupture' or 'normal'.

A sphincter defect was defined as a discontinuity in the muscle ring. Scar formation differed from normal muscle in all the sequences used.

MR SEQUENCES									
	TR (ms)	TE (ms)	TI (ms)	Flip Angel (0)	Slice (mm)	Matrix	FoV (mm)	Acq	
T 2 fs	6000	112		180	3	240 x 256	150	2	А
T 2 dess 3d	26.8	8		40	2	202 x 256	180	1	В
T 2 imr 2385	2385	50	200		4	148 x 256	180	2	С
T 2 TSE	5913	112		180	3	240 x 256	120	2	D
Т 1	600	17		90	3	256 x 256	125	2	Е
T 1 fs	640	17		90	3	256 x 256	150	2	F

Table 3. Endovaginal magnetic resonance imaging (EVMRI) parameters (TR time of repetition, TE echo time, TI time of inversion, FoV field of view, Acq acquisitions) (reprinted from Paper IV with permission).

A and C show edema and fluid collections, if present, and muscles (not clearly).

B The sphincter was localized; a large imaging volume with a thin slice.

D was used to separate muscle tissue from scar tissue (intermediate ability to separate).

E Scar tissue separated well from muscle tissue (second best).

F With and without contrast enhancement. Scar tissue and muscle tissue were distinguished best.

7. Primary sphincter repair

Primary sphincter repair was performed by a gynaecologist soon after delivery in the operating room. The end-to-end technique was used without further mobilization of the sphincters. A covering colostomy was performed in one case with grade IV perineal rupture (II).

8. Anterior overlapping repair

Parks' overlapping sphincter repair was used in every incontinent case if an anterior anal sphincter defect was detected in EAUS. With the patient in the lithotomy position, an anterior skin incision was made between the vagina and the anus. The healthy sphincter muscle on both sides of the defect was identified, the scar tissue was either spared or removed according to clinical situation, and an overlapping repair was performed. Both sphincters were sutured together or separately, depending on the identification. The wound was always closed. The patients received antibiotics during induction and for 5 to 10 days afterwards. The only complication was wound infection in 17 cases (44%), but these were mild and were treated at home (I).

The first postoperative clinical control examination took place six weeks after surgery, when Park' classification was used. The patients started doing sphincter exercises immediately at home. Those who had difficulties in starting sphincter exercises were offered biofeedback therapy (I).

The condition of the EAS at the time of the operation was classified as 'total rupture', 'partial rupture' or 'normal', and that of the IAS as 'normal' or 'rupture' (IV).

9. Statistical methods

The results in Papers I and II are expressed as median values with ranges. Comparisons of continuous data were performed using the Mann-Whitney U-test, comparisons of related samples using the Wilcoxon signed ranks test, and correlations were examined using Spearman's rank correlation. SPSS 9.0 for Windows (Chigaco, Illinois, USA) was used for the statistical analyses (I, II).

The results in Paper III are presented as means and ranges. For the statistical analyses, we used Fisher's exact test, Student's paired t-test, the X2 test and Wilcoxon's rank-sum test, employing the statistical software program SPLUS 2000. A difference was regarded as statistically significant with a P-value <0.05.

The results in Paper IV were assessed by calculating k values. Simple Kappa was used when there were two categories and weighted Kappa when there were three categories (EAS). Agreement is excellent when the k value is 0.81-1.00, good at 0.61-0.80, moderate at 0.41-0.60, fair at 0.21-0.40, and poor at 0.20 or less. A positive predictive value (PPV) and negative predictive value (NPV) were calculated for EAUS and MRI to demonstrate the condition of the anal sphincters (IV).

RESULTS

1. Results of delayed anterior overlapping repair

The median follow-up time for the 39 patients was 22 (range 2-99) months. A postoperative wound infection was found in 43.6% of cases, but there was no statistically significant correlation with the postoperative grading of incontinence. The results are shown in Table 4.

Parks	Patients' question- naire results	EAUS	PNTML
gr I-II 59%	good 31% acceptable 38%	overlap 72%	pudendopathy 36%
gr III-IV 41%	poor 31%		

Table 4. Results of overlapping anal sphincter repair (Paper I).

1.1. Parks' incontinence score

The symptoms of anal incontinence according to Parks' classification were significantly less marked 6 weeks after the operation than in the late follow-up control (P<0.001), although continence was seen in the late follow-up to have improved significantly after sphincter repair, corresponding to grade I to II in 23 patients (59 %). The postoperative Parks' classification correlated well with the subjective functional result (R=0.8, P<0.001).

1.2. Postoperative questionnaire

The follow-up patients' questionnaire results were good for 12 patients (31%), acceptable for 15 (38%) and poor for 12 (31%). The presence of preoperative cystocele and a long duration of incontinence had positive correlations with a poor functional result (R=0.5, P<0.01; R=0.4, P<0.05, respectively). There was no statistically significant correlation of BMI, the number of deliveries or the presence of wound infection with subjective functional results.

1.3. EAUS

Postoperative EAUS showed an overlap in 28 cases (72%), but a defect was still found in 11 (28%), correlating with a poor clinical result according to Parks' classification (R= 0.8, P<0.01) and the questionnaire results (R=0.7, P<0.01). The presence of an overlap in the postoperative EAUS examination correlated well with a favourable

clinical outcome in terms of Parks' classification (R=0.8, P<0.01) and the questionnaire (R=0.7, P<0.01), absence being more common in the Parks III/IV group than in the Parks I/II group (P<0.001). There was no correlation between rupture of the IAS detected in EAUS and the clinical outcome in terms of Parks' classification results. The postoperative EAUS results were not affected by the number of deliveries, duration of incontinence symptoms or occurence postoperative wound infections.

1.4. PNTML

Fourteen (36%) patients had pudendal neuropathy postoperatively, three of them bilaterally. There was no statistically significant correlation between the postoperative PNTML results and either the patients' questionnaire or postoperative Parks' classification results.

1.5. Analysis of failure

The duration of incontinence symptoms correlated with poor functional results (R0.4, P<0.05), but previous hysterectomy did not affect the Parks' classification results. The differences between the patients with good and poor functional results are shown in Table 5.

Variable	Parks I/II	Parks III/IV	P-value
Number of patients	23	16	
Length of follow-up (months)	10(2-78)	11(6-99)	ns
Parks' classification before operation	4(2-4)	4(3-4)	< 0.05
Patients' questionnaire result (1,2,3)	1(1-2)	2(1-3)	< 0.001
Age of the patients	45(27-79)	63(26-73)	< 0.05
EAS rupture after operation (n	0	11	< 0.001
Preoperative cystocele (n)	1	4	< 0.05
Preoperative rectocele (n)	8	11	< 0.05
Cysto and /or rectocele (n)	8	11	< 0.05

Table 5. Differences between the patients with good (Parks I/II) and poor (Parks III/IV) postoperative functional results (reprinted from paper I with permission).

The results are presented as median and range or number of patients (n).

Patients' questionnaire result (1=good, 2=acceptable, 3=poor), EAS = external anal sphincter.

P-value indicates the difference between the groups Parks I/II and Parks III/IV (Mann-Whitney U).

2. Results of primary sphincter repair

Forty-one women in group I (n=52) were primiparous, ten women were having their second delivery and one woman had had four deliveries previously. The earlier deliveries had been vaginal in seven cases and by caesarean section in four. The median duration of gestation was 40 weeks and 3 days (372/7 - 411/7), and the presentation at delivery was normal in 42 cases and abnormal in 10 (19 per cent). Mediolateral episiotomy had been performed in 43 cases. The delivery was assisted by vacuum extraction in 18 cases (35 per cent), but there was only one forceps-assisted delivery. The median birth weight of the infants was 3 628g (2 900-5 055).

In the control group (group II, n=51), normal vertex presentation was achieved in 48 cases and abnormal occipitoposterior presentation in 3 (6 per cent). The median duration of gestation was 40 weeks and 2 days (374/7-424/7). Mediolateral episiotomy had been performed in 48 cases and vacuum extraction in 14 (27%), but there were no forceps-assisted deliveries. The median weight of the infants was 3 450g (2 400-4 430). The results are shown in Table 6.

	fecal incontinence	EAUS persistent defect	mean resting pressure cmH2O	mean squeeze pressure cmH2O
rupture group n=52	20%	75%	80	40
control group n=51	4%	20%	99	67
P-value	< 0.001	< 0.01	0.001	<0.001

Table 6. Results of primary sphincter repair (Paper II).

2.1. Clinical results

Symptoms of anal incontinence existed in 31 (61%) cases, of whom 10 (20%) had fecal incontinence. Group I had more severe symptoms according to Parks' and Wexner's classifications than group II (P<0.001). One woman with colostomy was excluded from the analysis of incontinence symptoms. A total of 23 women in group I (44%) and 3 in group II (6%) had fecal urgency incontinence (P<0.001).

2.2. EAUS results

A persistent EAS defect was detected in 39 women in group I (75%), of whom 15 (29%) had a total rupture, while in group II there were 10 women with an EAS defect (20%) and only one with a total rupture. EAS rupture was thus a significantly more common finding in group I than in group II (P<0.01). An IAS defect was

found in 20 women in group I (38%) and in 4 (8%) in group II (P<0.001). Both anal sphincters were ruptured in 8 women in group I (15%) and 4 in group II (7.8%). All the defects occurred in the anterior portion of the anal sphincter.

A persistent EAS or IAS defect correlated positively with a poor clinical outcome according to Parks' (r=0.59, P<0.01) and Wexner's classifications (r=0.61, P<0.01).

2.3. Anal manometric results

Mean resting and squeeze were significantly lower in the rupture group than in the controls, but there was a negative correlation between the manometric results and the clinical outcome according to Parks' (resting pressure: r=-0.34, P<0.01; squeeze pressure: r=-0.26, P<0.05) and Wexner's classifications (resting pressure: r=-0.42, P<0.01; squeeze pressure: r=-0.29, P<0.01).

2.4. PNTML results

Six women in group I had only right-sided neuropathy, three had left-sided neuropathy and none had bilateral neuropathy. There was no statistically significant correlation between the PNTML measurements and the clinical results in Parks' or Wexner's classification. Neither was there any significant correlation between the PNTML results and the EAUS findings.

2.5. Risk factors for anal sphincter rupture

Abnormal presentation of the fetus was the only risk factor for anal sphincter rupture during delivery. There was no statistically significant difference between the groups concerning mother's diabetes mellitus, duration of the second stage of delivery, use of mediolateral episiotomy, use of vacuum extraction, gestational age or birth weight.

3. Sphincter rupture and anal incontinence after first vaginal delivery

3.1. Symptoms of anal incontinence

Symptoms of mild anal incontinence, mainly gas incontinence, increased more after vaginal delivery than after caesarean section (P<0.032), (Table 7). Fecal urgency was found in 9 cases.

Wexner points	Vaginal delivery (n=75) before/after	Section group (n=24) before/after
0	63/41	19/19
1-5	12/33	5/5
6-10	0/1	0/0
10-20	0/0	0/0

Table 7. Numbers of women with symptoms of anal incontinence according to Wexner' classification.

3.2. EAUS

Occult anal sphincter defects were detected by using EAUS after vaginal delivery in 17 of the 75 women (23%), 15 of whom (20%) had an EAS defect, 2 (2.7%) an IAS defect and 4 (5.3%) defects in both sphincters. There were no clinically observable sphincter ruptures in this population. Lacerations of the perineum and vagina were found in 21 (28%) women, 17 being of grade I and four of grade II.

3.3. Risk factors

New anal sphincter defects were detected after vacuum extraction in 9 of the 20 women (45 %), i.e. significantly more commonly than in those who had had a normal vaginal delivery (P=0.015) No new sphincter defects were found in the caesarean section group.

3.4. Manometric results

An abnormal EAUS finding after vaginal delivery entailed a significant decrease in mean squeeze pressures relative to the manometric results before delivery (P=0.0025). Anal resting and squeeze pressures after vaginal delivery by vacuum extraction (mean 95/72cmH2O, range 30-152/19-152) were lower than after normal vaginal delivery (mean 98/80cmH2O, range 51-146/11-252), but the difference was not statistically significant.

4. Diagnosis by EAUS or EVMRI

The findings were evaluated independently and compared with those at operation. EAUS and EVMRI showed similar levels of agreement with the surgical findings, 12 out of 19 (63%) vs. 11 out of 19 (58%), respectively. IAS defects were detected equally by EAUS and EVMRI in relation to the surgical diagnosis. There was considerable variation between radiologists in diagnosing defects by EVMRI.

4.1. EAS

A total rupture of the EAS was confirmed at surgery, in ten patients and a partial rupture in eight patients. The EAS of one patient was normal. The EAS defect in 12 out of the 19 patients (63%) was assessed correctly by EAUS. Agreement with surgical results was fair (kappa=0.36). The decision to perform surgery had been based on the presence of an anal sphincter defect as seen in the EAUS examination and clinical symptoms of anal incontinence. In this regard, there were no false-negative EAUS examinations and only one false-positive. The MR images were false-negative in one case and false-positive in one.

An EAS defect was correctly assessed by EVMRI on a consensus reading in 11 out of the 19 cases (58%), and agreement with the surgical results was fair (kappa=0.26). Agreement between the observers on the condition of the EAS in EVMRI is shown in Table 8.

Table 8. Agreement between two observers on the condition of the EAS and IAS asseen in endovaginal MRI (reprinted from Paper IV with permission).

Readers	EAS	IAS
LK and AK	0.45	0.32
LK and ET	0.32	0.18
AK and ET	0.69	0.47

The numbers are k-values EAS= external anal sphincter IAS= internal anal sphincter

4.2. IAS

A rupture of the IAS was found at surgery in 12 patients, while seven had a normal IAS. The condition of the IAS was properly diagnosed by EAUS in 14 out of the 19 cases (74%), the result that is indicative of moderate accuracy (kappa=0.51). The condition of the IAS was correctly assessed by EVMRI on a consensus reading in 14 out of the 19 cases (74%), again indicating moderate agreement (kappa=0.45) with the surgical results. The radiologists' opinions varied considerably.

DISCUSSION

1. Delayed sphincter repair after obstetric rupture: analysis of failure

The results presented here are short-term ones and show that only a minority of the incontinent patients became completely continent after the operation, although two thirds showed an improved degree of continence. In the remaining one third the symptoms persisted, so that other options must be considered for such cases. These findings are in accordance with those reported earlier (Parks and McPartlin 1971, Christiansen and Pedersen 1987a, Pezim et al. 1987, Laurberg et al. 1988, Fleshman et al. 1991, Wexner et al. 1991a, Engel et al. 1994, Sitzler and Thomson. 1996, Kammerrer-Doak et al. 1998, Savoye-Collet et al. 1999).

Unfortunately, long-term results have not been satisfactory. Malouf et al. (2000c) reported in their long-term results for patients who had had a repair a minimum of five years previously that none of these patients was fully continent to both stool and flatus, and that the degree of continence deteriorated with time in the majority of cases, showing an outcome failure rate of 50 per cent. Even the patients who had had a good overlap repair as visualized by EAUS did not maintain good function in the long term. The reason for this may lie in the nature of the obstetric damage, which most likely affects both the muscles and the nerves. Karoui et al. (2000) showed that the results of sphincterplasty deteriorate with time, and that the presence of an IAS rupture is a poor prognostic factor.

Our patients with a poor outcome were older than those with a more favourable outcome, and the duration of incontinence was longer. Moreover, preoperative cystocele and rectocele, indicating perineal descent and possible nerve damage, were found more often in these patients. The effect of age has varied in previous studies, however (Simmang et al. 1994, Nikiteas et al. 1996, Gilliland et al. 1998).

Oliveira et al. (1996) noted a significant change in mean squeeze pressures and anal high pressure zone length after successful repair in patients over 60 years of age, implying that improved sphincter function seems to be a major factor for a good outcome irrespective of age. All the patients in our series had the defect on the anterior side of the sphincter, irrespective of sphincter function, an the differences in the results may arise from the fact that others may have regarded a non-functioning sphincter as a contraindication to repair. Age itself does not seem to be important, but a well-contracting muscle is.

We used Parks' scoring system to assess the degree of incontinence, which may provide better results than other, more recent grading scales (Pescatori et al. 1992, Jorge and Wexner 1993a, Vaizey et al. 1999b). The Wexner scale has better clinical applicability than Parks' scale, and is now used uniformly, but we were not able to use it in this retrospective study.

Savoye-Collet et al. (1999) have demonstrated an association between postoperative EAUS findings and clinical outcome, whereas Kammerer-Doak et al. (1998) found no correlation between symptomatic relief and endovaginal US findings. We, in turn, noted that a defect in EAUS was significantly related to an unimproved clinical outcome. The reasons for a repair failure are either incomplete plasty at operation or the subsequent rupture of a successful plasty. It is likely that older patients may initially have an incomplete plasty due to the lack of sufficient muscle, on account of sphincter atrophy or scar tissue. This was the obvious reason in three of our failed patients, in whom we made a repeated reconstruction without achieving any progress. It is also possible that some of the failures in our series were due to rupture of the repair. The ultimate reason for persistent EAS rupture in late follow-up is impossible to know in the absence of an early postoperative EAUS examination. Others have reported good results after secondary repair in such cases (Pinedo et al. 1999), and all the symptomatic patients with persistent EAS rupture in our series underwent a re-operation. When considering a new repair, MRI of the sphincters might be a valuable option, in addition to EAUS, for ruling out the possibility of sphincter atrophy (Rociu et al. 1999a).

Laurberg et al. (1988) and Gilliland et al. (1998) have previously shown that there is a correlation between preoperative PNTML and postoperative clinical results, but we were unable to establish any statistical relationship between these. In cases of pudendal neuropathy, or so called idiopathic incontinence, sphincter repair is of no significance at all. The potential of the newly developed technique of sacral nerve stimulation is under evaluation at present, and is reported to be a promising prospect in such cases, and maybe also after failed sphincter repair (Vaizey et al. 1999a, Kenefick et al. 2002).

Our conclusion is that anterior overlapping sphincter repair is worthwhile in symptomatic patients with an EAS defect visible in EAUS if there is enough functioning muscle left.

2. Primary sphincter repair: are the results good enough?

The results of primary sphincter repair are evidently not good enough, as symptoms of anal incontinence persisted in 61 per cent of our patients, 20 per cent of whom had fecal incontinence. The clinical features of anal incontinence were usually minimized and hidden at the beginning. According to earlier studies, approximately one third of women continue to suffer from anal incontinence despite primary sphincter repair (Haadem et al. 1988, Nielsen et al. 1992, Bek and Laurberg 1992, Sorensen et al. 1993, Crawford et al. 1993, Sultan et al. 1994a, Tetzschner et al.1996, Fornell et al. 1996, Walsh et al. 1996, Kammerer-Doak et al. 1999).

We found a persistent EAS defect in 75 per cent of our cases, including 29 per cent with a total rupture. Both anal sphincters were ruptured in 15 per cent of cases.

Similar results have been obtained elsewhere (Zetterström et al. 2003, Davis et al. 2003). Poen et al. (1998) reported that as many as 88 per cent of the women in their long-term study had a sphincter defect visible in the EAUS examination and up to 40 per cent had symptoms of anal incontinence.

One of the reasons for poor results after primary repair is thought to be the fact that the operations are often performed by junior medical staff with an insufficient understanding of anal anatomy. The various structures of the sphincters and perineum may be difficult to identify because of swollen tissues, bleeding and contamination with liquid or solid feces.

The only risk factor that we could find was occipitoposterior presentation of the fetus, as also shown by Sultan et al. (1994a) and Fitzpatrick et al. (2001). Our primary sphincter repairs were performed by a gynaecologist in the operating room soon after the delivery, using the end-to-end technique without further mobilization of the sphincters.

The first article to report on primary sphincter repair by the overlap technique instead of the end-to-end technique was that of Sultan et al. (1999), after which Fitzpatrick et al. (2000) published a prospective randomized clinical trial comparing primary overlap (N=55) with approximation repair (N=57) in the case of third-degree obstetric tears. Fecal urgency persisted in 20 per cent of the overlap cases and 30 per cent of the approximation cases. There was no statistically significant difference between the groups in functional, manometric or EAUS findings. The overall symptomatic outcome was good, although up to 66 per cent of the women still had an EAS defect visible in EAUS examination.

More recently, Kairaluoma et al. (2004) have reported promising medium-term results of overlapping primary sphincter repair. They made a prospective study of 31 women with anal sphincter tears on whom an anterior overlap sphincter repair had been performed immediately by an experienced surgeon in an operating room. After a median follow-up time of 24 months, occasional incontinence to flatus and stools occurred in 17 and 7 per cent of the patients, respectively, and overlap of the EAS was found in EAUS in as many as 94 per cent.

Cooke et al. (1999) demonstrated that there may be a role for a colorectal team in the management of acute severe vaginal tears involving the rectal or anal mucosa. Our conclusion is that primary sphincter repair should be done in an operating room with good lighting by an experienced, senior obstetrician or surgeon, together with a junior one, both being used to dealing with primary tears after delivery, with a subsequent follow-up examination at a colorectal clinic three to six months after delivery (Walsh et al. 1996).

If a primary repair has failed, a secondary repair can be done. This is often complicated by extensive scar tissue and poor functioning of the EAS, depending on the degree of existing pudendal neuropathy. Moreover, separate repair of the IAS is not usually possible, and incontinence for flatus remains. Short-term results of secondary repairs have been acceptable, but the true long-term outcome is disappointing, in that less than 50 per cent of the patients become continent and in most cases the functional results deteriorate significantly with time (Malouf et al. 2000c, Halverson and Hull 2002). The role of a caesarean section with regard to preventing anal incontinence in subsequent deliveries after primary sphincter repair is not clear. There is some evidence that the routine use of caesarean section does not prevent anal incontinence (Lal et al. 2003, Harkin et. al 2003). As a rule, it can recommended for women with both a previous history of anal sphincter tears and persisting symptoms of anal incontinence, because of the high risk of deteriorating incontinence consequent on further vaginal deliveries.

3. Sphincter rupture and anal incontinence after the first vaginal delivery

The symptoms of anal incontinence observed after the first vaginal delivery in this prospective study were mild, consisting mainly of flatus incontinence and affected 27 per cent of the patients. In earlier studies the incidence of anal incontinence following vaginal delivery has been up to 44 per cent (Sultan et al. 1993a, Crawford et al. 1993, Zetterstöm et al. 2003). A half of our patients in whom an anal sphincter defect was found at the EAUS examination had symptoms of anal incontinence and 17 per cent had fecal incontinence.

The first article about occult anal sphincter defects found in EAUS examination, published by Sultan and colleagues (1993a), was based on examinations of 150 primiparous women 6 weeks before delivery and 6 to 8 weeks postnatally, and showed that where there were no sphincter defects before delivery, but 35 per cent had defects in the EAUS examination after delivery, of which 13 per cent became symptomatic. Vacuum extraction was not found to be a risk factor for an anal sphincter defect. Since then, occult anal sphincter defects have been reported in several other studies: Donnelly et al. (1998), Rieger et al. (1998), Zetterström et al. (1999), Varma et al. (1999), Faltin et al. (2000), Belmonte-Montes et al. (2001), Williams et al. (2001), and Nazir et al. (2002).

Earlier results support our finding that occult anal sphincter defects visible in an EAUS examination correlate with symptoms of anal incontinence (Crawford et al. 1993, Belmonte-Montes et al. 2001). Faltin et al. (2000) have also shown that a diagnosis of anal sphincter defects obtained by EAUS predicts anal incontinence in women with no clinically detected sphincter tears.

We found vacuum extraction to be an important risk factor for an occult anal sphincter defect and symptoms of anal incontinence, in that, occult defects were diagnosed in 23 per cent of the primiparous women after normal vaginal delivery but in as many as 45 per cent after vacuum extraction. Some earlier studies support this idea (Macarthur et al. 1997, Groutz et al. 1999, de Leeuw et al. 2001b). According to de Leeuw et al. (2001b), the relative risk of anal sphincter rupture was 1.79 per cent if vacuum extraction was used, while Groutz et al. (1999) found in their prospective study that the incidence of postpartum anal incontinence was significantly higher after vacuum extraction than after normal spontaneous delivery (25 per cent versus 3.8 per cent).

We found no new sphincter defects in the EAUS examination if a caesarean section had been performed, as shown earlier by Sultan et al. (1993a). On the other hand, Fynes et al. (1998) maintained that a caesarean section performed in late labour does not protect the anal sphincter mechanism, while Zetterström et al. (2003) demonstrated that a caesarean section protects the anal sphincters from occult injury but not from symptoms of anal incontinence. Our results support this idea. Lal et al. (2003) and Harkin et al. (2003) have concluded recently that a caesarean section does not prevent anal incontinence.

Williams et al. (2001), studying anal sphincter damage after vaginal delivery by three-dimensional endosonography, found that 11 per cent of their subjects had an EAS defect associated with a significant decrease in squeeze pressure and an increase in incontinence symptoms compared with cases without trauma. We noted that mean squeeze pressures were reduced by sphincter defects found in EAUS, but there are also some studies in which no such correlation could be established (Nielsen et al. 1992, Nazir et al. 2002).

We did not observe any clinically detected sphincter ruptures, even though the incidence of perineal lacerations was 28 per cent and the proportion of vacuum extraction deliveries was 20 per cent. A long-term study by Pollack et al. (2004) has shown that anal incontinence among primiparous women increases with time and is affected by further childbirth, the risk factors for symptoms of anal incontinence being a clinically diagnosed sphincter tear and subsequent delivery.

In conclusion, occult anal sphincter defects and mild symptoms of anal incontinence are common in primiparous women, and vacuum extraction leads to a higher incidence of occult sphincter defects. If an occult sphincter tear without clinical signs of incontinence is found in the EAUS examination, no surgical intervention is needed, but women with a symptomatic EAS defect should be offered sphincter repair. As a rule, more attention should be paid to possible symptoms of anal incontinence after the delivery as well as to symptoms of urine incontinence.

4. Anal Incontinence: Diagnosis by EAUS or EVMRI

The normal anatomy of the sphincter area as imaged by the endorectal coil is welldocumented (Hussain et al. 1995, deSouza et al. 1996, Rociu et al. 1999a, Williams et al. 2002), but the resolution of sphincter imaging with the body coil, which is more comfortable for the patient, is not adequate (Aronson et al. 1990, Schafer et al. 1994). Sphincter imaging with a phased array body coil shows good focal resolution, and combined with an endoanal coil, even better resolution in normal volunteers (Beets-Tan et al. 2001).

In view of the high price of the coils, we evaluated the possibility of using an endorectal prostatic surface coil as an endovaginal coil directed posteriorly towards the sphincters. The problem with the use of the surface coil is the high signal intensity in the closest area, i.e. the vaginal wall and the external sphincter in its immediate neighbourhood, which reduced the potential value of the imaging. In spite of careful windowing, the vaginal wall and the tissue about five mm underneath showed an artificially high signal intensity, detracting from image quality and resolution in that area. Most ruptures of the EAS at delivery occur in this weakest area. The EAS has been shown to be evaluated better by EAMR than by EAUS, and EVMR and EAUS were equal in the present study (Rociu et al. 1999b, Beets-Tan et al. 2001). The IAS is well characterized by an endovaginal coil and well enhanced by i.v. contrast material, although this does not increase the potential for detecting a rupture, whereas it does unnecessarily increase the expense and duration of the examination.

The explanation of why the radiologists' opinions varied considerably regarding the MRI results is that we classified the condition of the EAS into three categories and the condition of the IAS into two. The three radiologists reading the images (at the beginning of their learning curves) showed the most marked agreement if both anal sphincters were ruptured, the IAS was intact or the EAS rupture was total. Atrophy of the sphincters was not evaluated here, although there are EAMR reports on sphincter atrophy (deSouza et al. 1996, Briel et al. 1999, Beets-Tan et al. 2001).

There is abundant experience of the use of EAMRI for diagnosing anal sphincter defects. Stoker and colleagues (1996) found EAMRI to be preferable over an EAUS examination and reliable for the detection of anal sphincter defects, and a group under Rociu et al. (1999b) reported in a retrospective study that EAMRI was better than EAUS for their detection and characterization. Stoker et al. (2002), in a recent review of advances in the imaging of posterior pelvic floor disorders, notably constipation, prolapse and anal incontinence, suggested that dynamic MRI may be a valuable alternative, as the pelvic floor muscles are visualized and both EAUS and endoanal MRI can be used to detect anal sphincter defects.

EAUS is a well-tolerated, easily available and cheap examination as compared with EVMRI, and unlike EVMRI, it can be undertaken soon after delivery and the condition of the anal sphincters can be confirmed. Its weakness is poor contrast, which makes analyses of the EAS difficult. Atrophy of the EAS cannot be visualized by EAUS, and the technique is also operator-dependent (Bartram and Sultan 1995, Frudinger et al. 1999, Damon et al. 2002). The ultrasound technique has also been developed, so that nowadays a three-dimensional EAUS examination is available that facilitates sagittal and coronal reconstruction of the anal canal, resulting in a better diagnosis of anal sphincter defects (Williams et al. 2001).

The results of Malouf et al. (2000b), who studied prospectively the accuracy of EAMRI and EAUS in patients with fecal incontinence, suggest that EAUS and EAMRI are equivalent for diagnosing EAS defects, but MRI is inferior for diagnosing IAS defects. In our study EAUS proved superior to EVMRI for diagnosing EAS defects, but they were equivalent for diagnosing IAS defects.

Since the decision to operate was based on EAUS findings and symptoms of anal incontinence, this may indicate some bias in favour of EAUS.

5. Limitations of this work

The patient data used in Papers I and II were gathered retrospectively, the control visits being arranged prospectively, which weakens the value of the information. Unfortunately, we did not have anal manometry available at the time of the work for Paper I.

Our operation results were assessed in the short term, after a median interval of 22 months (range 2-99) in Paper I and 15 months (range 2-144) in Paper II, and it would be interesting to know the long- term outcomes (after five or more years). The control group for Paper II consisted only of primiparous women, so that we did not perform PNTML measurements, as this would have been too laborious and expensive.

We used Parks' and Wexners' classifications to assess the difficulty of the symptoms of anal incontinence. The women were asked about their subjective symptoms of anal incontinence and the replies were written down. More objective results could have been obtained using standard quality of life measurements.

The number of cases used in the prospective study of occult anal sphincter defects and symptoms of incontinence in primiparous women could have been higher, as only 10 per cent of the women invited actually participated, and the follow-up time was short, a median of 4 months (range 2-9) after delivery. A long-term follow-up would be warranted in future.

No control group was used in Paper IV, which detracts the eventual value of this information, and all the EAUS examinations were carried out by the same person (TP), which must be taken into consideration as a possible source of interpretation bias. However, the fact that the same person performed the EAUS examinations before and after delivery throughout the investigation is more likely to increase the accuracy rather than reduce it. EAUS is not an exact method, but it is relatively easy to perform and the results of earlier blinded trials have been in agreement.

6. Future aspects and clinical applications

More attention should be paid to symptoms of anal incontinence after childbirth. All women should be asked about possible symptoms of anal incontinence after delivery as well as about symptoms of urine incontinence.

The results of the anterior overlap operation could be improved by use of a better technique, insistence on an experienced operator, better patient selection and better timing of the operation. Sphincter repair should perhaps be offered only to patients with a clearly functioning EAS.

Sphincter repair is of no significance at all in cases of pudendal neuropathy. The role of the newly developed technique of sacral nerve stimulation is under evaluation and it is reported to be promising in such cases, and maybe also after failed sphincter repair. Further research addressing SNS in patients with anal incontinence is needed.

There is some evidence that better results may be achieved in primary sphincter repair if the anterior overlap technique is used instead of the end-to-end technique, but further randomised controlled trials of the two techniques are needed. The operation should be performed immediately (at least the next morning) in an operating room by an experienced physician together with a junior colleague. A follow-up is then needed, and a clinical examination, including an assessment of symptoms of fecal urgency, EAUS and anal manometry, should be undertaken 3 -6 months after the repair. If the rupture is still found and the patient continues to have symptoms of anal incontinence, surgical repair of the anal sphincter should be repeated by a proctologist alone.

There is some evidence to suggest that in order to prevent anal sphincter tears, episiotomy should be avoided (Dannecker et al. 2005). If episiotomy is needed, the mediolateral approach is recommended. Manual help for the infant's head and support for the perineum during delivery also seems to prevent anal sphincter defects (Pirhonen et al. 1998). In order to avoid anal sphincter tears in the case of a high infant birth weight relative to the mother, occiputposterior presentation, or a high position of the infant's head, so that vacuum extraction seems to be necessary, caesarean section should be seriously considered as an alternative.

If an occult anal sphincter defect is found in EAUS without symptoms of anal incontinence, no surgical measures are needed. On the other hand, Pollack et al. (2004) show in their long-term study that anal incontinence increases with time in primiparous women and is affected by further childbirth, the risk factors being a clinically diagnosed sphincter tear and subsequent delivery.

The role of subsequent deliveries by caesarean section after primary sphincter repair with regard to preventing anal incontinence is not clear. There is some evidence that the routine use of caesarean section does not prevent anal incontinence (Lal et al. 2003, Harkin et. al 2003), but it can as a rule be recommended for women with a previous history of anal sphincter tears and persisting symptoms of fecal incontinence during pregnancy because of the high risk of deteriorating incontinence following further vaginal deliveries. After secondary or delayed sphincter repair, further deliveries should be by caesarean section. Further studies are needed on the prevention, recognition and management of anal sphincter tears.

CONCLUSIONS

- 1. Anterior overlapping sphincter repair is worthwhile in symptomatic patients with an EAS defect found in EAUS after obstetric trauma, if there is enough functioning muscle left. Advanced age, preoperative cystocele and rectocele, long-lasting severe incontinence symptoms and EAS rupture visible in postoperative EAUS are risk factors for a poor outcome. A repeat EAUS examination is valuable for planning further procedures after a failed sphincter repair.
- 2. The results of primary sphincter repair were unsatisfactory. Symptoms of anal incontinence persisted in 61 per cent of the cases examined by EAUS and a residual defect was found in 75 per cent. Abnormal fetal presentation was the only risk factor for anal sphincter rupture during vaginal delivery. Patients need to be followed up after primary sphincter repair, and a clinical examination, including assessment of symptoms of fecal urgency, EAUS and anal manometry, should be undertaken in every patient 6 months after repair. If the rupture is still found and the patient continues to have symptoms of anal incontinence, surgical repair of the anal sphincter should be repeated by a proctologist alone.
- 3. Occult sphincter tears are common in primiparous women, especially following vacuum extraction. Sphincter defects found in EAUS reduce mean squeeze pressures. More attention should be paid to anal incontinence after childbirth, and women with a symptomatic EAS defect should be offered sphincter repair.
- 4. The EAUS examination and EVMRI are of equal value for diagnosing EAS and IAS defects, although EAMRI has previously been shown to be better.

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REFERENCES

Abramowitz L, Sobhani I, Ganansia R et al. Are sphincter defects the cause of anal incontinence after vaginal delivery? Results of a prospective study. Dis Colon Rectum 2000; 43:590-6.

Aitola P, Hiltunen KM, Matikainen M. Functional results of anterior levatorplasty and external sphincter plication for faecal incontinence. Ann Chir Gynaecol 2000; 89:29-32.

American Medical Systems. Fecal incontinence scoring system. Minnetonka: American Medical Systems.

Aronson MP, Lee RA, Berquist TH. Anatomy of anal sphincters and related structures in continent women studied with magnetic resonance imaging. Obstet Gynecol 1990; 76:846-51.

Baeten CG, Geerdes BP, Adang EM et al. Anal dynamic graciloplasty in the treatment of intractable fecal incontinence. N Engl J Med 1995; 332:1600-5.

Baeten CG, Bailey HR, Bakka A et al. Safety and efficacy of dynamic graciloplasty for fecal incontinence: report of a prospective, multicenter trial. Dynamic Graciloplasty Therapy Study Group. Dis Colon Rectum 2000; 43:743-51.

Bartram CI, Sultan AH. Anal endosonography in fecal incontinence. Gut 1995; 37:4-6.

Beets-Tan RG, Morren GL, Beets GL et al. Measurement of anal sphincter muscles: endoanal US, endoanal MR imaging, or phased-array MR imaging? A study with healthy volunteers. Radiology 2001; 220:81-9.

Bek KM, Laurberg S. Risks of anal incontinence from subsequent vaginal delivery after a complete obstetric anal sphincter tear. Br J Obstet Gynaecol 1992; 99:724-6.

Belmonte-Montes C, Hagerman G, Vega-Yepez PA, Hernandez-de-Anda E, Fonseca-Morales V. Anal sphincter injury after vaginal delivery in primiparous females. Dis Colon Rectum 2001; 44:1244-8.

Briel JW, Stoker J, Rociu E et al. External anal sphincter atrophy on endoanal magnetic resonance imaging adversely affects continence after sphincteroplasty. Br J Surg 1999; 86:1322-7.

Browning GG, Parks AG. Postanal repair for neuropathic fecal incontinence: correlation of clinical result and anal canal pressures. Br J Surg 1983; 70:101-4.

Browning GG, Motson RW. Anal sphincter injury. Management and results of Parks sphincter repair. Ann Surg 1984; 199:351-7.

Buhr HJ, Kroesen AJ. [The importance of diagnostics in fecal incontinence. Endosonography]. Chirurg 2003; 74:4-14.

Chapman AE, Geerdes B, Hewett P et al. Systematic review of dynamic graciloplasty in the treatment of fecal incontinence. Br J Surg 2002; 89:138-53.

Cheetham MJ, Kenefick NJ, Kamm MA. Non-surgical management of fecal incontinence. Hosp Med 2001; 62:538-41.

Christiansen J, Pedersen IK. Traumatic anal incontinence. Results of surgical repair. Dis Colon Rectum 1987a; 30:189-91.

Christiansen J, Lorentzen M. Implantation of artificial sphincter for anal incontinence. Lancet 1987b; 2:244-5.

Christiansen J, Sparso B. Treatment of anal incontinence by an implantable prosthetic anal sphincter. Ann Surg 1992; 215:383-6.

Christiansen J, Roed-Petersen K. Clinical assessment of the anal continence plug. Dis Colon Rectum 1993; 36:740-2.

Coats PM, Chan KK, Wilkins M, Beard RJ. A comparison between midline and mediolateral episiotomies. Br J Obstet Gynaecol 1980; 87:408-12.

Cook TA, Mortensen NJ. Management of fecal incontinence following obstetric injury. Br J Surg 1998; 85:293-9.

Cooke T, Keane D, Morsensen N. Is there a role for the colorectal team in the management of acute severe third-degree vaginal tears? Colorectal Dis 1999; 1:263-266.

Cornes H, Bartolo DC, Stirrat GM. Changes in anal canal sensation after childbirth. Br J Surg 1991; 78:74-7.

Crawford LA, Quint EH, Pearl ML, Delancey JO. Incontinence following rupture of the anal sphincter during delivery. Obstet Gynecol 1993; 82:527-31.

Cuesta MA, Meijer S, Derksen EJ, Boutkan H, Meuwissen SG. Anal sphincter imaging in fecal incontinence using endosonography. Dis Colon Rectum 1992; 35:59-63.

Damon H, Henry L, Barth X, Mion F. Fecal incontinence in females with a past history of vaginal delivery: significance of anal sphincter defects detected by ultrasound. Dis Colon Rectum 2002; 45:1445-50.

Dannecker C, Hillemanns P, Strauss A, Hasbargen U, Hepp H, Anthuber C. Episiotomy and perineal tears presumed to be imminent: the influence on the urethral pressure profile, analmanometric and other pelvic floor findings--follow-up study of a randomized controlled trials. Acta Obstet Gynecol Scand 2005; 84:65-71. Davis K, Kumar D, Stanton SL et al. Symptoms and anal sphincter morphology following primary repair of third-degree tears. Br J Surg 2003; 90:1573-9.

Deen KI, Kumar D, Williams JG, Olliff J, Keighley MR. The prevalence of anal sphincter defects in fecal incontinence: a prospective endosonic study. Gut 1993; 34:685-8.

de Leeuw JW, Vierhout ME, Struijk PC, Hop WC, Wallenburg HC. Anal sphincter damage after vaginal delivery: functional outcome and risk factors for fecal incontinence. Acta Obstet Gynecol Scand 2001a; 80:830-4.

de Leeuw JW, Struijk PC, Vierhout ME, Wallenburg HC. Risk factors for third degree perineal ruptures during delivery. BJOG 2001b; 108:383-7.

de Parades, V, Etienney I, Thabut D et al. Anal sphincter injury after forceps delivery: myth or reality? A prospective ultrasound study of 93 females. Dis Colon Rectum 2004; 47:24-34.

deSouza NM, Kmiot WA, Puni R et al. High resolution magnetic resonance imaging of the anal sphincter using an internal coil. Gut 1995a; 37:284-7.

deSouza NM, Puni R, Gilderdale DJ, Bydder GM. Magnetic resonance imaging of the anal sphincter using an internal coil. Magn Reson Q 1995b; 11:45-56.

deSouza NM, Puni R, Zbar A et al. MR imaging of the anal sphincter in multiparous women using an endoanal coil: correlation with in vitro anatomy and appearances in fecal incontinence. AJR Am J Roentgenol 1996; 167:1465-71.

Donnelly V, O'Connell PR, O'Herlihy C. The influence of oestrogen replacement on fecal incontinence in postmenopausal women. Br J Obstet Gynaecol 1997; 104:311-5.

Donnelly V, Fynes M, Campbell D et al. Obstetric events leading to anal sphincter damage. Obstet Gynecol 1998; 92:955-61.

Engel AF, Kamm MA, Sultan AH, Bartram CI, Nicholls RJ. Anterior anal sphincter repair in patients with obstetric trauma. Br J Surg 1994; 81:1231-4.

Faltin DL, Boulvain M, Irion O et al. Diagnosis of anal sphincter tears by postpartum endosonography to predict fecal incontinence. Obstet Gynecol 2000; 95:643-7.

Fang DT, Nivatvongs S, Vermeulen FD et al. Overlapping sphincteroplasty for acquired anal incontinence. Dis Colon Rectum 1984; 27:720-2.

Falk PM, Blatchford GJ, Cali RL, Christensen MA, Thorson AG. Transanal ultrasound and manometry in the evaluation of fecal incontinence. Dis Colon Rectum 1994; 37:468-72.

Felt-Bersma RJ, Cuesta MA, Koorevaar M et al. Anal endosonography: relationship with anal manometry and neurophysiologic tests. Dis Colon Rectum 1992; 35:944-9.

Felt-Bersma RJ, Cuesta MA, Koorevaar M. Anal sphincter repair improves anorectal function and endosonographic image. A prospective clinical study. Dis Colon Rectum 1996; 39:878-85.

Fernando RJ, Sultan AH, Radley S, Jones PW, Johanson RB. Management of obstetric anal sphincter injury: a systematic review & national practice survey. BMC Health Serv Res 2002; 2:9.

Fitzpatrick M, Behan M, O'Connell PR, O'Herlihy C. A randomized clinical trial comparing primary overlap with approximation repair of third-degree obstetric tears. Am J Obstet Gynecol 2000; 183:1220-4.

Fitzpatrick M, McQuillan K, O'Herlihy C. Influence of persistent occiput posterior position on delivery outcome. Obstet Gynecol 2001; 98:1027-31.

Fleshman JW, Peters WR, Shemesh EI, Fry RD, Kodner IJ. Anal sphincter reconstruction: anterior overlapping muscle repair. Dis Colon Rectum 1991; 34:739-43.

Fornell EK, Berg G, Hallbook O, Matthiesen LS, Sjodahl R. Clinical consequences of anal sphincter rupture during vaginal delivery. J Am Coll Surg 1996; 183:553-8.

Franz HB, Hirsch HA. [Pelvic floor reconstruction after traumatic childbirth damage]. Zentralbl Chir 1996; 121:688-91.

Frenckner B, Euler CV. Influence of pudendal block on the function of the anal sphincters. Gut 1975; 16:482-9.

Frenckner B, Ihre T. Influence of autonomic nerves on the internal and sphincter in man. Gut 1976; 17:306-12.

Frudinger A, Halligan S, Bartram CI, Spencer JA, Kamm MA. Changes in anal anatomy following vaginal delivery revealed by anal endosonography. Br J Obstet Gynaecol 1999; 106:233-7.

Fynes M, Donnelly VS, O'Connell PR, O'Herlihy C. Cesarean delivery and anal sphincter injury. Obstet Gynecol 1998; 92:496-500.

Fynes M, Donnelly V, Behan M, O'Connell PR, O'Herlihy C. Effect of second vaginal delivery on anorectal physiology and fecal continence: a prospective study. Lancet 1999a; 354:983-6.

Fynes MM, Marshall K, Cassidy M 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 1999b; 42:753-8.

Fynes MM, Behan M, O'Herlihy C, O'Connell PR. Anal vector volume analysis complements endoanal ultrasonographic assessment of postpartum anal sphincter injury. Br J Surg 2000; 87:1209-14. Gattuso JM, Kamm MA. Adverse effects of drugs used in the management of constipation and diarrhoea. Drug Saf 1994; 10:47-65.

Gee AS, Durdey P. Urge incontinence of faeces is a marker of severe external anal sphincter dysfunction. Br J Surg 1995; 82:1179-82.

Gilliland R, Altomare DF, Moreira H, Jr. et al. Pudendal neuropathy is predictive of failure following anterior overlapping sphincteroplasty. Dis Colon Rectum 1998; 41:1516-22.

Gold DM, Bartram CI, Halligan S et al. Three-dimensional endoanal sonography in assessing anal canal injury. Br J Surg 1999; 86:365-70.

Groutz A, Fait G, Lessing JB et al. Incidence and obstetric risk factors of postpartum anal incontinence. Scand J Gastroenterol 1999; 34:315-8.

Haadem K, Ohrlander S, Lingman G. Long-term ailments due to anal sphincter rupture caused by delivery--a hidden problem. Eur J Obstet Gynecol Reprod Biol 1988; 27:27-32.

Haadem K, Gudmundsson S. Can women with intrapartum rupture of anal sphincter still suffer after-effects two decades later? Acta Obstet Gynecol Scand 1997; 76:601-3.

Halverson AL, Hull TL. Long-term outcome of overlapping anal sphincter repair. Dis Colon Rectum 2002; 45:345-8.

Hardcastle JD, Parks AG. A study of anal incontinence and some principles of surgical treatment. Proc R Soc Med 1970; 63:116-8.

Harkin R, Fitzpatrick M, O'Connell PR, O'Herlihy C. Anal sphincter disruption at vaginal delivery: is recurrence predictable? Eur J Obstet Gynecol Reprod Biol 2003; 109:149-52.

Henriksen TB, Bek KM, Hedegaard M, Secher NJ. Episiotomy and perineal lesions in spontaneous vaginal deliveries. Br J Obstet Gynaecol 1992; 99:950-4.

Hussain SM, Stoker J, Lameris JS. Anal sphincter complex: endoanal MR imaging of normal anatomy. Radiology 1995; 197:671-7.

Jameson JS, Chia YW, Kamm MA et al. Effect of age, sex and parity on anorectal function. Br J Surg 1994; 81:1689-92.

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

Johanson JF, Lafferty J. Epidemiology of fecal incontinence: the silent affliction. Am J Gastroenterol 1996; 91:33-6.

Jorge JM, Wexner SD. Etiology and management of fecal incontinence. Dis Colon Rectum 1993a; 36:77-97.

Jorge JM, Wexner SD. Anorectal manometry: techniques and clinical applications. South Med J 1993b; 86:924-31.

Jorge JM, Habr-Gama A. The value of sphincter asymmetry index in anal incontinence. Int J Colorectal Dis 2000; 15:303-10.

Kairaluoma M, Raivio P, Kupila J, Aarnio M, Kellokumpu I. The role of biofeedback therapy in functional proctologic disorders. Scand J Surg 2004; 93:184-90.

Kairaluoma MV, Raivio P, Aarnio MT, Kellokumpu IH. Immediate repair of obstetric anal sphincter rupture: medium-term outcome of the overlap technique. Dis Colon Rectum 2004; 47:1358-63.

Kamm MA. Obstetric damage and fecal incontinence. Lancet 1994; 344:730-3.

Kammerer-Doak DN, Dominguez C, Harner K, Dorin MH. Surgical repair of fecal incontinence. Correlation of sonographic anal sphincter integrity with subjective cure. J Reprod Med 1998; 43:576-80.

Kammerer-Doak DN, Wesol AB, Rogers RG, Dominguez CE, Dorin MH. A prospective cohort study of women after primary repair of obstetric anal sphincter laceration. Am J Obstet Gynecol 1999; 181:1317-22.

Karoui S, Savoye-Collet C, Koning E, Leroi AM, Denis P. Prevalence of anal sphincter defects revealed by sonography in 335 incontinent patients and 115 continent patients. AJR Am J Roentgenol 1999; 173:389-92.

Karoui S, Leroi AM, Koning E et al. Results of sphincteroplasty in 86 patients with anal incontinence. Dis Colon Rectum 2000; 43:813-20.

Keighley MR, Fielding JW. Management of fecal incontinence and results of surgical treatment. Br J Surg 1983; 70:463-8.

Kenefick NJ, Vaizey CJ, Cohen CR, Nicholls RJ, Kamm MA. Double-blind placebocontrolled crossover study of sacral nerve stimulation for idiopathic constipation. Br J Surg 2002; 89:1570-1.

Klein MC, Gauthier RJ, Robbins JM et al. Relationship of episiotomy to perineal trauma and morbidity, sexual dysfunction, and pelvic floor relaxation. Am J Obstet Gynecol 1994; 171:591-8.

Korsgen S, Deen KI, Keighley MR. Long-term results of total pelvic floor repair for postobstetric fecal incontinence. Dis Colon Rectum 1997; 40:835-9.

Lal M, Mann H, Callender R, Radley S. Does cesarean delivery prevent anal incontinence? Obstet Gynecol 2003; 101:305-12.

Laurberg S, Swash M, Henry MM. Delayed external sphincter repair for obstetric tear. Br J Surg 1988; 75:786-8.

Law PJ, Kamm MA, Bartram CI. Anal endosonography in the investigation of fecal incontinence. Br J Surg 1991; 78:312-4.

Lehur PA, Michot F, Denis P et al. Results of artificial sphincter in severe anal incontinence. Report of 14 consecutive implantations. Dis Colon Rectum 1996; 39:1352-5.

Lehur PA, Roig JV, Duinslaeger M. Artificial anal sphincter: prospective clinical and manometric evaluation. Dis Colon Rectum 2000; 43:1100-6.

Leroi AM, Kamm MA, Weber J, Denis P, Hawley PR. Internal anal sphincter repair. Int J Colorectal Dis 1997; 12:243-5.

Macarthur C, Bick DE, Keighley MR. Fecal incontinence after childbirth. Br J Obstet Gynaecol 1997; 104:46-50.

Madoff RD, Williams JG, Caushaj PF. Fecal incontinence. N Engl J Med 1992; 326:1002-7.

Malouf AJ, Vaizey CJ, Nicholls RJ, Kamm MA. Permanent sacral nerve stimulation for fecal incontinence. Ann Surg 2000a; 232:143-8.

Malouf AJ, Williams AB, Halligan S et al. Prospective assessment of accuracy of endoanal MR imaging and endosonography in patients with fecal incontinence. AJR Am J Roentgenol 2000b; 175:741-5

Malouf AJ, Norton CS, Engel AF, Nicholls RJ, Kamm MA. Long-term results of overlapping anterior anal-sphincter repair for obstetric trauma. Lancet 2000c; 355:260-5.

Matzel KE, Stadelmaier U, Hohenfellner M, Gall FP. Electrical stimulation of sacral spinal nerves for treatment of fecal incontinence. Lancet 1995; 346:1124-7.

Matzel KE, Kamm MA, Stosser M et al. Sacral spinal nerve stimulation for fecal incontinence: multicentre study. Lancet 2004; 363:1270-6.

Mavrantonis C, Wexner SD. A clinical approach to fecal incontinence. J Clin Gastroenterol 1998; 27:108-21.

Miller R, Orrom WJ, Cornes H, Duthie G, Bartolo DC. Anterior sphincter plication and levatorplasty in the treatment of fecal incontinence. Br J Surg 1989; 76:1058-60.

Moller BK, Laurberg S. Intervention during labor: risk factors associated with complete tear of the anal sphincter. Acta Obstet Gynecol Scand 1992; 71:520-4.

Mortensen N, Humphreys MS. The anal continence plug: a disposable device for patients with anorectal incontinence. Lancet 1991; 338:295-7.

Nazir M, Carlsen E, Nesheim BI. Do occult anal sphincter injuries, vector volume manometry and delivery variables have any predictive value for bowel symptoms after first time vaginal delivery without third and fourth degree rupture? A prospective study. Acta Obstet Gynecol Scand 2002; 81:720-6.

Nelson R, Norton N, Cautley E, Furner S. Community-based prevalence of anal incontinence. JAMA 1995; 274:559-61.

Nielsen MB, Hauge C, Rasmussen OO, Pedersen JF, Christiansen J. Anal endosonographic findings in the follow-up of primarily sutured sphincteric ruptures. Br J Surg 1992; 79:104-6.

Nielsen MB, Hauge C, Pedersen JF, Christiansen J. Endosonographic evaluation of patients with anal incontinence: findings and influence on surgical management. AJR Am J Roentgenol 1993; 160:771-5.

Nikiteas N, Korsgen S, Kumar D, Keighley MR. Audit of sphincter repair. Factors associated with poor outcome. Dis Colon Rectum 1996; 39:1164-70.

Norton C, Kamm MA. Outcome of biofeedback for fecal incontinence. Br J Surg 1999; 86:1159-63.

Norton C, Kamm MA. Anal plug for faecal incontinence. Colorectal Dis 2001; 3:323-7.

Norton C, Chelvanayagam S, Wilson-Barnett J, Redfern S, Kamm MA. Randomized controlled trial of biofeedback for fecal incontinence. Gastroenterology 2003; 125:1320-9.

Oliveira L, Pfeifer J, Wexner SD. Physiological and clinical outcome of anterior sphincteroplasty. Br J Surg 1996; 83:502-5.

Orrom WJ, Miller R, Cornes H et al. Comparison of anterior sphincteroplasty and postanal repair in the treatment of idiopathic fecal incontinence. Dis Colon Rectum 1991; 34:305-10.

Parks AG, Porter NH, Hardcastle J. The syndrome of the descending perineum. Proc R Soc Med 1966; 59:477-82.

Parks AG, McPartlin JF. Late repair of injuries of the anal sphincter. Proc R Soc Med 1971; 64:1187-9.

Parks AG. Anorectal incontinence. Proc R Soc Med 1975; 68:681-90.

Parks AG, Swash M, Urich H. Sphincter denervation in anorectal incontinence and rectal prolapse. Gut 1977; 18:656-65.

Pedersen IK, Christiansen J. A study of the physiological variation in anal manometry. Br J Surg 1989; 76:69-70. Pemberton JH, Kelly KA. Achieving enteric continence: principles and applications. Mayo Clin Proc 1986; 61:586-99.

Percy JP, Neill ME, Swash M, Parks AG. Electrophysiological study of motor nerve supply of pelvic floor. Lancet 1981; 1:16-7.

Perry S, Shaw C, McGrother C et al. Prevalence of fecal incontinence in adults aged 40 years or more living in the community. Gut 2002; 50:480-4.

Pescatori M, Pavesio R, Anastasio G, Daini S. Transanal electrostimulation for fecal incontinence: clinical, psychologic, and manometric prospective study. Dis Colon Rectum 1991; 34:540-5.

Pescatori M, Anastasio G, Bottini C, Mentasti A. New grading and scoring for anal incontinence. Evaluation of 335 patients. Dis Colon Rectum 1992; 35:482-7.

Pezim ME, Spencer RJ, Stanhope CR et al. Sphincter repair for fecal incontinence after obstetrical or iatrogenic injury. Dis Colon Rectum 1987; 30:521-5.

Pickrell KL, Broadbent TR, Masters FW, Metzer JT. Construction of a rectal sphincter and restoration of anal continence by transplanting the gracilis muscle; a report of four cases in children. Ann Surg 1952; 135:853-62.

Pinedo G, Vaizey CJ, Nicholls RJ et al. Results of repeat anal sphincter repair. Br J Surg 1999; 86:66-9.

Pirhonen JP, Grenman SE, Haadem K et al. Frequency of anal sphincter rupture at delivery in Sweden and Finland--result of difference in manual help to the baby's head. Acta Obstet Gynecol Scand 1998; 77:974-7.

Poen AC, Felt-Bersma RJ, Dekker GA et al. Third degree obstetric perineal tears: risk factors and the preventive role of mediolateral episiotomy. Br J Obstet Gynaecol 1997; 104:563-6.

Poen AC, Felt-Bersma RJ, Strijers RL et al. Third-degree obstetric perineal tear: long-term clinical and functional results after primary repair. Br J Surg 1998; 85:1433-8.

Pollack J, Nordenstam J, Brismar S et al. Anal incontinence after vaginal delivery: a fiveyear prospective cohort study. Obstet Gynecol 2004; 104:1397-402.

Read NW, Abouzekry L. Why do patients with fecal impaction have fecal incontinence. Gut 1986; 27:283-7.

Rieger N, Schloithe A, Saccone G, Wattchow D. A prospective study of anal sphincter injury due to childbirth. Scand J Gastroenterol 1998; 33:950-5.

Riskin-Mashiah S, O'Brian SE, Wilkins IA. Risk factors for severe perineal tear: can we do better? Am J Perinatol 2002; 19:225-34.

Rociu E, Stoker J, Zwamborn AW, Lameris JS. Endoanal MR imaging of the anal sphincter in fecal incontinence. Radiographics 1999a; 19:171-177.

Rociu E, Stoker J, Eijkemans MJ, Schouten WR, Lameris JS. Fecal incontinence: endoanal US versus endoanal MR imaging. Radiology 1999b; 212:453-8.

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

Rogers J, Levy DM, Henry MM, Misiewicz JJ. Pelvic floor neuropathy: a comparative study of diabetes mellitus and idiopathic fecal incontinence. Gut 1988; 29:756-61.

Rosen HR, Urbarz C, Holzer B, Novi G, Schiessel R. Sacral nerve stimulation as a treatment for fecal incontinence. Gastroenterology 2001; 121:536-41.

Ryhammer AM, Bek KM, Laurberg S. Multiple vaginal deliveries increase the risk of permanent incontinence of flatus urine in normal premenopausal women. Dis Colon Rectum 1995; 38:1206-9.

Ryhammer AM, Laurberg S, Hermann AP. Long-term effect of vaginal deliveries on anorectal function in normal perimenopausal women. Dis Colon Rectum 1996; 39:852-9.

Savoye-Collet C, Savoye G, Koning E et al. Anal endosonography after sphincter repair: specific patterns related to clinical outcome. Abdom Imaging 1999; 24:569-73.

Schafer A, Enck P, Furst G et al. Anatomy of the anal sphincters. Comparison of anal endosonography to magnetic resonance imaging. Dis Colon Rectum 1994; 37:777-81.

Schweiger M. Method for determining individual contributions of voluntary and involuntary anal sphincters to resting tone. Dis Colon Rectum 1979; 22:415-6.

Simmang C, Birnbaum EH, Kodner IJ, Fry RD, Fleshman JW. Anal sphincter reconstruction in the elderly: does advancing age affect outcome? Dis Colon Rectum 1994; 37:1065-9.

Sitzler PJ, Thomson JP. Overlap repair of damaged anal sphincter. A single surgeon's series. Dis Colon Rectum 1996; 39:1356-60.

Slade MS, Goldberg SM, Schottler JL, Balcos EG, Christenson CE. Sphincteroplasty for acquired anal incontinence. Dis Colon Rectum 1977; 20:33-5.

Snooks SJ, Setchell M, Swash M, Henry MM. Injury to innervation of pelvic floor sphincter musculature in childbirth. Lancet 1984a; 2:546-50.

Snooks SJ, Barnes PR, Swash M. Damage to the innervation of the voluntary anal and periurethral sphincter musculature in incontinence: an electrophysiological study. J Neurol Neurosurg Psychiatry 1984b; 47:1269-73.

Snooks SJ, Henry MM, Swash M. Fecal incontinence due to external anal sphincter division in childbirth is associated with damage to the innervation of the pelvic floor musculature: a double pathology. Br J Obstet Gynaecol 1985; 92:824-8.

Snooks SJ, Swash M, Mathers SE, Henry MM. Effect of vaginal delivery on the pelvic floor: a 5-year follow-up. Br J Surg 1990; 77:1358-60.

Sorensen M, Tetzschner T, Rasmussen OO, Bjarnesen J, Christiansen J. Sphincter rupture in childbirth. Br J Surg 1993; 80:392-4.

Speakman CT, Burnett SJ, Kamm MA, Bartram CI. Sphincter injury after anal dilatation demonstrated by anal endosonography. Br J Surg 1991; 78:1429-30.

Speakman CT, Hoyle CH, Kamm MA et al. Abnormal internal anal sphincter fibrosis and elasticity in fecal incontinence. Dis Colon Rectum 1995; 38:407-10.

Stoker J, Hussain SM, Lameris JS. Endoanal magnetic resonance imaging versus endosonography. Radiol Med 1996; 92:738-41.

Stoker J, Bartram CI, Halligan S. Imaging of the posterior pelvic floor. Eur Radiol 2002; 12:779-88.

Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. N Engl J Med 1993a; 329:1905-11.

Sultan AH, Nicholls RJ, Kamm MA et al. Anal endosonography and correlation with in vitro and in vivo anatomy. Br J Surg 1993b; 80:508-11.

Sultan AH, Kamm MA, Bartram CI, Hudson CN. Anal sphincter trauma during instrumental delivery. Int J Gynaecol Obstet 1993c; 43:263-70.

Sultan AH, Kamm MA, Hudson CN, Bartram C. Third degree obstetric anal sphincter tears: risk factors and outcome of primary repair. BMJ 1994a; 308:887-91.

Sultan AH, Kamm MA, Hudson CN. Pudendal nerve damage during labor: prospective study before and after childbirth. Br J Obstet Gynaecol 1994b; 101:22-8.

Sultan AH, Kamm MA, Talbot IC, Nicholls RJ, Bartram CI. Anal endosonography for identifying external sphincter defects confirmed histologically. Br J Surg 1994c; 81:463-5.

Sultan AH, Monga AK, Kumar D, Stanton SL. Primary repair of obstetric anal sphincter rupture using the overlap technique. Br J Obstet Gynaecol 1999; 106:318-23.

Sun WM, Read NW, Miner PB. Relation between rectal sensation and anal function in normal subjects and patients with fecal incontinence. Gut 1990a; 31:1056-61

Sun WM, Read NW, Donnelly TC. Anorectal function in incontinent patients with cerebrospinal disease. Gastroenterology 1990b; 99:1372-9.

Sun WM, Read NW, Verlinden M. Effects of loperamide oxide on gastrointestinal transit time and anorectal function in patients with chronic diarrhoea and fecal incontinence. Scand J Gastroenterol 1997; 32:34-8.

Swash M. Anorectal incontinence: electrophysiological tests. Br J Surg 1985; 72 :14-15.

Tan IL, Stoker J, Lameris JS. Magnetic resonance imaging of the female pelvic floor and urethra: body coil versus endovaginal coil. MAGMA 1997; 5:59-63.

Tan IL, Stoker J, Zwamborn AW et al. Female pelvic floor: endovaginal MR imaging of normal anatomy. Radiology 1998; 206:777-83.

Tanagho EA, Schmidt RA. Electrical stimulation in the clinical management of the neurogenic bladder. J Urol 1988; 140:1331-9.

Tetzschner T, Sorensen M, Rasmussen OO, Lose G, Christiansen J. Pudendal nerve damage increases the risk of fecal incontinence in women with anal sphincter rupture after childbirth. Acta Obstet Gynecol Scand 1995; 74:434-40.

Tetzschner T, Sorensen M, Lose G, Christiansen J. Anal and urinary incontinence in women with obstetric anal sphincter rupture. Br J Obstet Gynaecol 1996; 103:1034-40.

Tetzschner T, Sorensen M, Lose G, Christiansen J. Pudendal nerve function during pregnancy and after delivery. Int Urogynecol J Pelvic Floor Dysfunct 1997; 8:66-8.

Tjandra JJ, Milsom JW, Schroeder T, Fazio VW. Endoluminal ultrasound is preferable to electromyography in mapping anal sphincteric defects. Dis Colon Rectum 1993; 36:689-92.

Tobin GW, Brocklehurst JC. Fecal incontinence in residential homes for the elderly: prevalence, aetiology and management. Age Ageing 1986; 15:41-6.

Vaizey CJ, Kamm MA, Bartram CI. Primary degeneration of the internal anal sphincter as a cause of passive fecal incontinence. Lancet 1997; 349:612-5.

Vaizey CJ, Kamm MA, Gold DM et al. Clinical, physiological, and radiological study of a new purpose-designed artificial bowel sphincter. Lancet 1998; 352:105-9.

Vaizey CJ, Kamm MA, Turner IC, Nicholls RJ, Woloszko J. Effects of short term sacral nerve stimulation on anal and rectal function in patients with anal incontinence. Gut 1999a; 44:407-12.

Vaizey CJ, Carapeti E, Cahill JA, Kamm MA. Prospective comparison of fecal incontinence grading systems. Gut 1999b; 44:77-80.

Varma A, Gunn J, Gardiner A, Lindow SW, Duthie GS. Obstetric anal sphincter injury: prospective evaluation of incidence. Dis Colon Rectum 1999; 42:1537-43.

Wald A, Tunuguntla AK. Anorectal sensorimotor dysfunction in fecal incontinence and diabetes mellitus. Modification with biofeedback therapy. N Engl J Med 1984; 310:1282-7.

Walsh CJ, Mooney EF, Upton GJ, Motson RW. Incidence of third-degree perineal tears in labor and outcome after primary repair. Br J Surg 1996; 83:218-21.

Wexner SD, Marchetti F, Jagelman DG. The role of sphincteroplasty for fecal incontinence reevaluated: a prospective physiologic and functional review. Dis Colon Rectum 1991a; 34:22-30.

Wexner SD, Marchetti F, Salanga VD, Corredor C, Jagelman DG. Neurophysiologic assessment of the anal sphincters. Dis Colon Rectum 1991b; 34:606-12.

Williams NS, Patel J, George BD, Hallan RI, Watkins ES. Development of an electrically stimulated neoanal sphincter. Lancet 1991; 338:1166-9.

Williams AB, Bartram CI, Halligan S et al. Anal sphincter damage after vaginal delivery using three-dimensional endosonography. Obstet Gynecol 2001; 97:770-5.

Williams AB, Bartram CI, Halligan S et al. Endosonographic anatomy of the normal anal canal compared with endocoil magnetic resonance imaging. Dis Colon Rectum 2002; 45:176-83.

Wong WD, Jensen LL, Bartolo DC, Rothenberger DA. Artificial anal sphincter. Dis Colon Rectum 1996; 39:1345-51.

Wong WD, Congliosi SM, Spencer MP et al. The safety and efficacy of the artificial bowel sphincter for fecal incontinence: results from a multicenter cohort study. Dis Colon Rectum 2002; 45:1139-53.

Wunderlich M, Swash M. The overlapping innervation of the two sides of the external anal sphincter by the pudendal nerves. J Neurol Sci 1983; 59:97-109.

Zetterstrom J, Mellgren A, Jensen LL et al. Effect of delivery on anal sphincter morphology and function. Dis Colon Rectum 1999; 42:1253-60.

Zetterstrom J, Lopez A, Holmstrom B et al. Obstetric sphincter tears and anal incontinence: an observational follow-up study. Acta Obstet Gynecol Scand 2003; 82:921-8.