Obstetric pelvic floor and anal sphincter injuries

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Key content

• Vaginal delivery, especially the first, contributes to the development of pelvic organ prolapse, and urinary and anal incontinence.
• Although the effects of childbirth on the pelvic floor are commonly reported within 12 months of delivery, the peak presentation of symptoms occur some 2–3 decades later.
• Vaginal childbirth, advancing maternal age and increasing body mass index are the most consistent risk factors.
• Diagnosis of obstetric anal sphincter trauma can be improved by training and thereby minimising the risk of anal incontinence.
• Further research, using pelvic floor imaging, may improve our understanding of the nature of pelvic floor and anal sphincter injuries.

Learning objectives

• To review the effect of childbirth on pelvic organ function.
• To identify and appropriately manage anal sphincter injuries following childbirth.

Ethical issues

• There are inadequate long-term data from well-conducted longitudinal studies to make definitive recommendations in many aspects of pelvic organ dysfunction.

Key words: anal incontinence / childbirth / pelvic floor dysfunction / pelvic organ prolapse / urinary incontinence

Introduction

The earliest evidence of severe perineal injury sustained during childbirth is from the mummy of Henhenit, an Egyptian woman approximately 22 years of age from the harem of King Mentuhotep II of Egypt in 2050BC.1 Surgical repair of severe perineal injury was first mentioned in the Arabic book Al Kanoun, but the first recorded case of perineal suture was by Guillemau around 1610.1 In 1943, Gainey examined 1000 consecutive women at the postnatal visit and identified levator damage in 31%, a cystocele in 26% and a rectocele in 12%. In 1955, he presented data on a second series of 1000 consecutive women delivered by prophylactic forceps aided by a mediolateral episiotomy and reported a considerable reduction in levator damage, cystocele and rectocele to 11.5%, 9.9% and 1.5%, respectively.3 In recent years, with advances in imaging techniques, the understanding and management of birth-related trauma to the pelvic floor has improved dramatically.

Anatomy

The perineum corresponds to the pelvic outlet and is bound anteriorly by the pubic arch, posteriorly by the coccyx and laterally by the ischiopubic rami, ischial tuberosities and sacrotuberous ligaments. The perineum can be divided into two triangular parts by drawing an arbitrary line transversely between the ischial tuberosities. The anterior triangle (urogenital triangle) contains the superficial muscles, which include the superficial transverse perineal, bulbospongiosus and ischiocavernosus muscles. The posterior triangle (anal triangle) contains the anal sphincter complex. The pelvic floor mainly consists of the musculotendinous sheet called the levator ani muscle. There is considerable inconsistency in the literature regarding the anatomy of the levator ani muscle.4 However, it is broadly accepted that the levator ani is subdivided into the iliococcygeus, pubococcygeus and ischiococcygeus. The pubococcygeous is further subdivided into the pubourethralis, pubovaginalis and puborectalis. The puborectalis is the most caudal...
component of the levator ani complex and is situated cephalad to the deep component of the external anal sphincter (EAS), from which it is almost inseparable. The puborectalis therefore participates in the sphincter mechanism. Between the two arms of the puborectalis lies the levator hiatus, through which the rectum, vagina and urethra pass. The innervation of the pelvic floor is via the sacral nerves S2–S4 and it is believed that some innervation also occurs via the pudendal nerve.5

The anal sphincter complex consists of the EAS and internal anal sphincter (IAS) separated by the conjoint longitudinal coat (Figure 1).6 The IAS is a continuation of the circular smooth muscle of the bowel and ends above the anal margin at the junction of the superficial and subcutaneous part of EAS. The EAS is innervated by the inferior rectal branch of the pudendal nerve and contributes 15–30% of the resting anal pressure and 70% of the squeeze anal pressure.7 The IAS is innervated by the sympathetic (L5) and parasympathetic nerves (S2–S4) and accounts for 50–85% of the resting pressure.8

Classification of perineal trauma

Adopting uniform definitions/classification for perineal and anal sphincter injuries during childbirth reduces under-reporting of true obstetric anal sphincter injury and facilitates future audits and risk management. The current classification of perineal trauma9 includes the classification of anal sphincter tears proposed by Thakar and Sultan (Box 1).10 Incorrect classification and inappropriate repair can have epidemiological, clinical and medico-legal implications.

<table>
<thead>
<tr>
<th>Box 1. Classification of perineal trauma9,10</th>
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<tr>
<td><strong>First-degree:</strong> Laceration to the vaginal epithelium or perineal skin only</td>
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<tr>
<td><strong>Second-degree:</strong> Involvement of the perineal muscles but not the anal sphincter</td>
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<tr>
<td><strong>Third-degree:</strong> Disruption of the anal sphincter muscles which should be further subdivided into:</td>
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<tr>
<td>3a &lt;50% thickness of external sphincter torn</td>
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<tr>
<td>3b &gt;50% thickness of external sphincter torn</td>
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<tr>
<td>3c: Internal sphincter also torn</td>
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<tr>
<td><strong>Fourth-degree:</strong> Third-degree tear with disruption of the anal epithelium</td>
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Prevalence

More than 85% of women in the UK sustain some form of perineal trauma during vaginal delivery.11 However, the prevalence is dependent on variations in obstetric practice, including rates and type of episiotomy, which vary not only between one country and the next but also at a national level between delivery units and individual practitioners. In the UK, the episiotomy rate is 8–16%12 compared with 52–82% in some European countries13 and 39% in the USA.14

The prevalence of third- and fourth-degree tears, collectively referred to as obstetric anal sphincter injuries (OASIS), is dependent on the type of episiotomy practised. In centres where mediolateral episiotomies are practised, the rate of OASIS is 1.7% (2.9% in primiparae) compared with 12% (19% in primiparae) in centres practising midline episiotomy.6 A recent (2010–2011) survey of maternity units in the UK has revealed an OASIS rate of 3% (range of 0–8%)
(21% in primiparae). This wide range suggests that at the highest extreme there could be an element of overdiagnosis. However, what is more alarming is that units in the lower extreme are probably failing to diagnose a large number of OASIS.

Pelvic floor dysfunction following childbirth

Although the effects of childbirth on the pelvic floor are commonly reported within 12 months of delivery, the peak presentation of symptoms occur some 2–3 decades later. In addition to direct trauma to the anal sphincter, perineal and levator ani muscles, stretching and possible tearing of the endopelvic fascia may lead to pelvic floor dysfunction. The much higher prevalence of incontinence and prolapse in later life may reflect the cumulative effects of covert obstetric trauma, ageing, progression of neuropathy, weakness of fascial supports and the hormonal changes at menopause.

Symptoms of obstetric-related pelvic floor dysfunction are as follows:

- Anal incontinence
- Urinary incontinence
- Pelvic organ prolapse
- Sexual dysfunction.

Anal incontinence

The WHO International Consultation on Incontinence defines anal incontinence as the involuntary loss of flatus, liquid or solid stool that is a social or hygienic problem. Mechanism

Direct mechanical injury. The processes of continence and defaecation are complex and hence the understanding of pathophysiological mechanisms related to childbirth continues to evolve. Direct mechanical injury to the EAS and/or IAS resulting from obstetric trauma is a major cause of anal incontinence.

Neurological injury. Pudendal neuropathy may be the result of nerve compression from the fetal head, fetal macrosomia, prolonged pushing during the second stage of labour and forceps delivery. The pudendal nerve is particularly susceptible to compression and damage at the point where it curves round the ischial spine and enters the pudendal canal enclosed in its thick fibrous sheath (Alcock’s canal). Stretched or compressed nerves often undergo demyelination but usually recover with time or re-innervation of muscle occurs.

Combined mechanical and neurological trauma. Isolated neurological injury, as described above, is believed to be rare. Neuropathy more commonly accompanies mechanical damage.

Other mechanisms. Additional factors also play a role in maintaining anal continence, namely, normal consistency of stool, normal bowel transit and rectal sensitivity, a functional rectal reservoir, and intact somatic and autonomic innervation.

Incidence

In a postal questionnaire study of 906 women, 10 months after vaginal delivery, 4% developed new faecal incontinence. Flatus incontinence is more common and has been reported in 29% of primiparous women 9 months after delivery. A meta-analysis of five studies (717 vaginal deliveries) imaging the anal sphincter revealed a 26.9% incidence of anal sphincter defects in primiparous women and an 8.5% incidence of new sphincter defects in multiparous women. Although approximately 35% of women with a sphincter defect on endoanal ultrasound were symptomatic, 3.4% of women experienced postpartum faecal incontinence without a sphincter defect.

OASIS are clinically detectable in about 3% of vaginal deliveries in centres practising mediolateral episiotomy but rates of 7% have been described. When endoanal ultrasound was first performed in a prospective childbirth study, it was assumed that the OASIS seen on endoanal ultrasound were occult. However, it has been subsequently shown that genuine occult injuries are a rare entity as virtually all OASIS can be detected clinically by an appropriately trained clinician, highlighting the need for intensive and more focused training in the identification of OASIS. Although Faltin et al. have shown that detection can be improved by performing endoanal ultrasound before suturing, there were occasions when OASIS diagnosed by endoanal ultrasound could not be identified and repaired clinically.

Despite primary repair, anal incontinence occurs in 39% of women. This may be indicative of inappropriate diagnosis or inadequate repair technique. Sultan et al. conducted an interview of 75 doctors and 75 midwives and reported that 91% and 60%, respectively, indicated inadequate training in perineal anatomy and 84% and 61%, respectively, reported inadequate training in identifying third-degree tears.

In a 10-year longitudinal study following OASIS, deterioration in anal pressures occurred over time and were greater among women who had experienced complete rather than partial tears. Flatus and liquid faecal incontinence were more common among women who had sustained OASIS and symptoms were most severe after IAS injury. Despite obvious injury to the anal sphincters, symptoms of anal incontinence may not occur for some time after delivery. Bek and Laurberg found that in women who experienced transient anal incontinence after a complete tear, 39% had a
relapse of symptoms after the next vaginal delivery. The major long-term problem seen in these women was incontinence of flatus. Full-thickness anal sphincter disruption (Figure 2) was the most significant risk factor in the development of faecal incontinence.

Risk factors
In a Swedish study of 1336 women, the prevalence of flatus incontinence was found to be higher among women who had sustained OASIS.31 The first vaginal delivery confers the greatest risk of sustaining OASIS, with new injuries occurring up to 10 times more commonly in primiparous women.32 The prevalence of faecal incontinence is predominantly associated with forceps delivery, unassisted delivery at home, large fetal head circumference, obesity and increasing maternal age.33 Midline episiotomy,34 first vaginal delivery, shoulder dystocia and a persistent occipito-posterior position have been identified as the main risk factors for the development of OASIS.20,27 In a study of 531 consecutive women who had sustained OASIS,35 the risk factors and outcome of different grades of OASIS were assessed. The use of epidural analgesia was found to be the only independent factor predicting a major tear and hence a higher risk of faecal incontinence and lower anal canal pressures.

Can caesarean section reduce the risk of anal incontinence?
No significant difference in anal incontinence was seen in a randomised controlled trial on short and long-term follow-up.36,37 In a cohort study, first delivery by caesarean section appeared to reduce significantly the prevalence of faecal incontinence but not symptoms of severe flatus incontinence.38 In a 12-year longitudinal study, MacArthur et al.39 reported that there was no difference in faecal incontinence among women who had delivered either exclusively by caesarean section or who had some spontaneous vaginal births and some deliveries by caesarean section.39 In the absence of OASIS, delivery by caesarean section does not reduce the risk of anal incontinence. Caesarean section protects against new sphincter tears, which would explain why some studies have found a reduced prevalence of faecal incontinence after caesarean section.40

Role of imaging
Endoanal ultrasound yields highly detailed images of the sphincter (Figure 2). Imaging has become essential in the assessment and management of faecal incontinence particularly related to obstetric trauma.

Urinary incontinence
Mechanism
The exact mechanism of stress urinary incontinence in pregnancy and childbirth is not clear and is probably multifactorial. The development of symptoms of urinary incontinence during pregnancy, with a rapid postpartum recovery followed by a steady decline of continence over time suggests a dual mechanism of nerve and tissue involvement. Pudendal nerve conduction and electromyography studies have shown denervation injury after childbirth.41,42

In pregnancy, as a result of a reduction in the total collagen content and consequent change in the tensile properties of connective tissue, there is an increase in mobility of the bladder neck (Figure 3).43–45 Compared with continent women, those who have postpartum stress urinary incontinence have been found to have greater bladder neck mobility during straining before delivery.45

Prospective urodynamic studies performed during pregnancy and 6 to 9 weeks postpartum revealed a notable decrease in urethral closure pressure and urethral length44,46 after vaginal delivery. Women with persistent urinary incontinence after childbirth were found to have a shorter urethral length and a lower closure pressure compared with continent women.47 Subsequent studies have demonstrated that the midurethral closure pressure
(above 20 cmH₂O) was a contributing factor in maintaining urinary continence.48,49 Using magnetic resonance imaging (MRI), a two-fold increase in levator ani injuries has been shown in women with stress incontinence compared with primiparous continent women 50 but this association was not found using three-dimensional ultrasound.51

**Incidence**

Compared with nulliparous women, new urinary incontinence is more common in parous women. A lower incidence after planned caesarean section has been shown in a randomised controlled trial (absolute relative risk [RR] difference –2.8%, 95% confidence interval [CI] –5.1% to –0.5%).36 A prospective cohort study of 949 women found that urinary incontinence was experienced by 22.3% before pregnancy, 65.1% during the third trimester and 31.1% after delivery.52

Viktrup et al.53 interviewed 278 primiparae and found that the prevalence of stress urinary incontinence 5 years after a first delivery was 30%. Nineteen percent of those without symptoms after their first delivery had urinary incontinence in contrast with 92% in those who had symptoms at 3 months postpartum. At 12 years follow-up, 91% of women who developed symptoms during the first pregnancy or shortly after delivery but without remission 3 months postpartum had stress urinary incontinence compared with 57% in those who had remission of symptoms at 3 months postpartum.54

**Risk factors**

The evidence regarding the contribution of obstetric factors to the development of stress urinary incontinence is conflicting and it is debatable whether pregnancy or delivery is the major contributor to postpartum incontinence. Some investigators found a relationship with the duration of the second stage of labour (>1 hour)55 and birthweight,22 while others have found no significant correlation between stress urinary incontinence and fetal head circumference, second stage of labour,56 or birthweight.53 There is, however, a group of women at an inherent increased risk of developing urinary incontinence because of abnormalities in collagen.57

Rortveit et al. 58 reported that the prevalence of any incontinence was 10% in the nulliparous group, 16% in the caesarean section group and 21% in the vaginal delivery group. Thus, pregnancy itself rather than delivery may also be an important causal factor in the development of urinary incontinence. Postpartum urinary incontinence was independently associated with incontinence prior to and during pregnancy, even among those having a caesarean section. These studies highlight that antenatal and pre-pregnancy stress urinary incontinence increases the risk of future stress urinary incontinence.

**Can caesarean section reduce the risk of urinary incontinence?**

The short-term effect of caesarean section on the prevalence of postpartum urinary incontinence has been seen in four cohort studies. Two studies 52,58 have found a lower prevalence of urinary incontinence after caesarean section. Two cohort studies22,26 have reported the long-term effect of delivery mode: one reported reduced prevalence of urinary incontinence after first delivery by caesarean section and the other reduced prevalence of persistent postpartum urinary incontinence after delivery exclusively by caesarean section. Women who have never given birth appear to be at lower risk of urinary incontinence than those who have delivered only by caesarean section. In a 12-year follow-up study, MacArthur et al.39 found that after adjustment for parity, body mass index and age at first birth, women who delivered exclusively by caesarean section were less likely to have urinary incontinence than those who only had spontaneous vaginal births, but not if they had a combination of caesarean section and spontaneous vaginal births.
Role of imaging
Ultrasound imaging is increasingly being used to study bladder and urethral anatomy (Figure 3). Three-dimensional imaging after vaginal delivery shows an enlarged levator hiatus (Figure 4), a reduction in urethral sphincter volume and increased bladder neck mobility both in the antenatal and postpartum period. Other ultrasound studies have shown that the bladder neck is lower (Figure 3) after vaginal delivery as compared with nulligravid controls and women delivered by elective caesarean section.59

Levator avulsion injury (Figure 4) was previously reported to have a significant association with new stress urinary incontinence.60 However, recent research by the same group has demonstrated that puborectalis trauma is not associated with an increased risk of stress urinary incontinence or urodynamically proven stress urinary incontinence in a urogynaecological population.51

Pelvic organ prolapse
Mechanism
Levator ani muscle injury (partial or complete) found in vaginally parous women increases the chance of a woman developing pelvic organ prolapse (POP) by 4 to 11 times.58 Large imaging studies50,61 and biomechanical studies62 have shown that levator avulsion injuries or denervation occurs during descent of the fetal head and maternal expulsive forces during the active second stage of labour and crowning. Consequently, the levator hiatus enlarges and leads to anterior and central compartment prolapse.63

Incidence
Prolapse is noted to be more common in women after a vaginal delivery. Fifty percent of parous women have some degree of POP, of whom 15% are symptomatic whereas symptomatic prolapse is found in 2% of nulliparous women.64

Risk factors
O’Boyle et al.65 found that increase in POP quantification stage was seen in nulliparous pregnant women and also in those who had a vaginal delivery. Hence, both pregnancy and vaginal delivery are important risk factors for the development of POP. Hormonal and mechanical effects associated with the gravid uterus contribute to changes in pelvic organ support during pregnancy. Electromyography (EMG) studies have shown that significant pelvic denervation and reinnervation are associated with stress incontinence and POP.51,63 Collagen changes in the pelvic floor that are related to childbirth and endogenous hormone changes may also predispose to POP.

Role of imaging
Translabial/transperineal ultrasound allows definition of the presence of POP by demonstrating descent of the vaginal wall sonographically (Figures 3 and 5). Vaginal childbirth results in 28% enlargement of the levator hiatus (Figure 4) after a levator avulsion compared with 6% hiatus enlargement without avulsion injury.66 Enlarged hiatus is found in women who have clinically significant POP.67 Dietz et al.68 used translabial ultrasound to demonstrate that pregnancy and vaginal delivery result in an increased prevalence and size of a true rectocele (Figure 5).

An MRI study50 has shown that 20% of primiparous women had a visible defect in the pubovisceral or iliococcygeal portion of the levator ani muscle. These defects were not seen in nulliparous women. The most common morphologic abnormality of the levator ani is described as an avulsion injury of the pubovisceral muscle from the pubic ramus (Figure 4). This has been described in one-third of women and appears to be related to childbirth.60

Sexual dysfunction
Although sexual health problems are common after childbirth, they are frequently under-reported. Embarrassment and
preoccupation with the newborn are some of the reasons why many women do not seek help. Furthermore, there is a lack of professional awareness and expertise in recognition of sexual dysfunction. In the postpartum period, the most common sexual disorder is sexual pain followed by disorders of sexual desire, arousal and orgasm. These disorders may occur in a different sequence and may be interdependent.

Mechanism
The evolution from a pre-pregnant state to a postpartum period is a life-changing event with complex physiological and psychological sequelae. Perineal pain and dyspareunia are commonly a result of spontaneous perineal trauma, episiotomy and instrumental delivery. In a retrospective cohort of 626 primiparous women over a 6-month period after delivery, women with second-degree and third or fourth-degree tears were 80% and 270%, respectively, more likely to report dyspareunia at 3 months postpartum. This highlights that mechanisms leading to sexual dysfunction are multifactorial.

Incidence
Sexual health problems, as recalled by women, increase significantly after childbirth. Healthcare workers need to be made aware of this silent affliction as sexual morbidity can have a detrimental effect on a woman’s quality of life, impacting on her social, physical and emotional wellbeing. Perineal pain occurs in up to 42% of women immediately after delivery and significantly reduces to 22% and 10% at 8 and 12 weeks, respectively. In a cross-sectional study of 796 primiparous women over a 6-month period after delivery, Barrett et al. found that 62% experienced dyspareunia in the first 3 months postpartum, decreasing to 31% at 6 months. Thirty two percent had resumed intercourse within 6 weeks of giving birth and the majority of respondents (89%) had resumed intercourse within 6 months.

Risk factors
Compared with normal delivery, perineal pain occurs more frequently and persists for a longer period after an instrumental or breech vaginal delivery. In a prospective study, Barrett et al. found that dyspareunia in the first 3 months after delivery was significantly associated with vaginal delivery and previous experience of dyspareunia. However, at 6 months there was no association between dyspareunia and mode of delivery but previous experience of dyspareunia remained a significant risk factor. This suggests that women with pre-existing dyspareunia prior to pregnancy may have specific needs or issues that could be identified and managed antenatally. Further analysis of the same cohort of women found that depression is another risk factor in delaying the resumption of sexual intercourse by 6 months. Glazener found that women who reported perineal pain, depression and tiredness experienced problems related to intercourse more often than those who did not report these concerns.

Management
Role of the perineal clinic
The perineal clinic is an ideal setting to offer patients support, debriefing and focus on health concerns relating to the delivery including urinary, bowel, sexual, prolapse and wound problems. A large number of women attending this clinic will have sustained a third- or fourth-degree tear. Antenatal women with any of these problems, including female genital mutilation, are also being seen in the perineal clinic. As these problems are usually of a sensitive nature, women should ideally be seen in a dedicated perineal clinic instead of a busy general clinic approximately 6-8 weeks after delivery. Furthermore, this environment would facilitate...
childcare and breastfeeding. A dedicated one-stop perineal clinic provides women with an opportunity to be given an explanation of the circumstances under which the perineal injury occurred and appropriate counselling regarding mode of subsequent delivery. However, the best model of care is dependent on local expertise and resources.

Who should look after these women?
Ideally, a team of healthcare providers with the knowledge and expertise to care for these women should be available to mothers. Experienced professionals (such as urogynaecologists and trained nurses or midwives) who are trained in anal manometry and endoanal ultrasound scanning, form an integral part of this clinic. Integration of multidisciplinary professionals such as physiotherapists, continence nurse specialists, colorectal nurse specialists, colorectal surgeons and psychosexual counsellors promote a holistic approach to pelvic floor and perineal problems.

Delivery after anal sphincter injury
Women who sustain OASIS need counselling regarding their management in a subsequent pregnancy. The two major issues following OASIS that concern women most when contemplating delivery in a subsequent pregnancy are the risk of recurrence of OASIS and the risk of developing anal incontinence.

Risk of recurrence of OASIS
While there is evidence that a policy of restrictive episiotomy is better than routine episiotomy, there are no randomised studies regarding the use of prophylactic episiotomy in women who previously sustained OASIS. Most of the published studies are from centres practising selective midline episiotomy where the recurrence rate of OASIS is 11%. In centres where the normal practice is to perform selective mediolateral episiotomy, OASIS recurred in 4.4% to 6.8%.

The authors therefore do not recommend routine episiotomy following previous OASIS.

Risk of developing anal incontinence after subsequent vaginal delivery
The data available are limited and retrospective. Poen et al. identified 43 women (original cohort of 117 women with OASIS) who had a subsequent vaginal delivery. The rate of anal incontinence was 56% compared with 34% in those who did not subsequently deliver (RR 1.6; 95% CI 1.1–2.5) but there was no comparable caesarean section group. Sangalli et al. followed up 177 women 13 years after OASIS and found that only 2.5% of women complained of faecal incontinence after a third-degree tear compared with 26.5% following a fourth-degree tear. This could reflect the effect of not specifically identifying and repairing the IAS. These results are also supported by Roos et al. who did a follow-up study of 531 women 9 weeks after delivery and found that women with grade 3c/4 tears had significantly worse defaecatory symptoms and bowel-related quality of life compared with women with grade 3a/3b tears.

Recommended mode of delivery for subsequent pregnancy
There are no randomised studies to determine the most appropriate mode of delivery following OASIS. In order to counsel women, it is useful to have a symptom questionnaire as well as anal ultrasound and manometry results. If vaginal delivery is contemplated then these tests should be performed during the current pregnancy.

Figure 6 shows a flow diagram illustrating management after a third/fourth-degree tear in subsequent pregnancies conducted in Croydon University Hospital, Surrey, UK over the past 10 years. Follow-up of these women through a subsequent pregnancy and delivery revealed that 70% of women achieved a vaginal delivery without any compromise of anorectal function.

Women who sustained OASIS with subsequent severe incontinence should be offered secondary sphincter repair provided there is an EAS defect and there is evidence of contractility in the residual muscle. Some women with faecal incontinence may choose to complete their family before embarking on anal sphincter surgery. Following counselling, these women are allowed a vaginal delivery as the risk of further damage is minimal and should not impact on the outcome of surgery. This benefit should be weighed against the risks associated with caesarean section for all subsequent pregnancies.

Figure 6. Management of obstetric anal sphincter injuries for subsequent pregnancies

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Conclusion

Pelvic floor injuries during a vaginal delivery can be considered a significant factor in the development of urinary incontinence, faecal incontinence, pelvic organ prolapse and sexual dysfunction. About eight out of ten women in the UK sustain some form of perineal trauma during childbirth. Overall, although there has been a noticeable increase in the prevalence of OASIS, this could be largely attributed to improvements in detection of such injuries. A good understanding of perineal and anal sphincter anatomy and adherence to sound principles of management are associated with a better outcome. Increased body mass index, large fetal head, forceps delivery and first vaginal delivery have been implicated in the development of pelvic floor dysfunction. However, a recent study from 2011 has shown that unless women have all their deliveries by the abdominal route, caesarean section does not protect from subsequent urinary incontinence. Even among those who deliver exclusively by caesarean section, 40% still report urinary incontinence and caesarean section confers no benefit in terms of subsequent faecal incontinence. It would therefore appear that the pregnancy itself rather than mode of delivery alone may be a contributory factor in the development of pelvic floor dysfunction.

Only a large randomised controlled trial of elective caesarean section versus vaginal delivery could determine the role of elective caesarean section in protecting against pelvic floor dysfunction without a resultant increase in morbidity and mortality. In the absence of such information, we need to focus on making vaginal delivery safer by more focused training and implementing evidence-based obstetric practice.

Conflict of interest

None declared.

References

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