Pelvic floor trauma following vaginal delivery
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Purpose of review
Recent years have seen a steady increase in the information available regarding pelvic floor trauma in childbirth. A review of this information is timely in view of the ongoing discussion concerning elective caesarean section.

Recent findings
In addition to older evidence regarding pudendal nerve injury, it has recently been shown that inferior aspects of the levator ani and fascial pelvic organ supports such as the rectovaginal septum can be disrupted in childbirth. Such trauma is associated with pelvic organ prolapse, bowel dysfunction, and urinary incontinence. Elective caesarean section seems to have a limited protective effect that appears to weaken with time. Older age at first delivery may be associated with a higher likelihood of trauma and subsequent symptoms.

Summary
Pelvic floor trauma is a reality, not a myth. It is currently not possible, however, to advise patients as to whether avoidance of potential intrapartum pelvic floor trauma is worth the risk, cost, and effort of elective caesarean section. In some women this may well be the case. The identification of women at high risk for delivery-related pelvic floor trauma should be a priority for future research in this field.

Keywords
childbirth, delivery, levator muscle, pelvic floor, prolapse, ultrasound

Introduction
Obstetrics is currently undergoing its most major change since the introduction of antenatal ultrasound. At times one wonders whether in the long run vaginal childbirth is destined to become a practice limited to fringe groups and resource-poor settings. Not surprisingly, the issue often elicits emotional responses as any change in the status quo would have major implications for the relative role of healthcare providers and require significant redistribution of scarce resources. Inevitably, discussion of pelvic floor trauma in childbirth is seen as partisan, due to the fact that a growing awareness of potential negative effects of vaginal childbirth, such as urinary and faecal incontinence and pelvic organ prolapse, is very likely to contribute to rising caesarean section rates, even if other factors may currently predominate [1,2]. There is no doubt, however, that caesarean section, whether primary or secondary, can lead to significant and occasionally major morbidity and even mortality. For now we have no means of determining whether this risk outweighs the risk of attempted vaginal childbirth in a given patient, and this must be reflected in the advice that we provide to our patients.

For some forms of pelvic floor morbidity, it is not clear whether pregnancy or childbirth is to blame [3,4], and long latencies between the presumptive cause (childbirth) and effect (incontinence and prolapse) further complicate research. Despite all this, we now have relatively firm epidemiologic evidence on the association between vaginal childbirth and urinary incontinence and prolapse [4,5,6], as summarized in Fig. 1 first published in a review article by DeLancey [5**].

Regarding prolapse, pregnancy and childbirth are well documented as major risk factors [3,6]. Caesarean delivery is associated with less need for surgical correction of incontinence or prolapse [3] and seems protective against symptomatic prolapse [7]. Regarding urinary incontinence, several large epidemiologic studies have shown that caesarean section provides partial protection from stress incontinence [4,8,9**]. The one randomized controlled trial that may shed light on the degree of protection to be expected from elective caesarean section is the Term Breech Trial. It showed a relative risk of 0.62 in the elective caesarean section group 3 months after childbirth [8], but of course these data are of limited use due to the special nature of breech delivery. Any protective effect appears to fade over time [9**] as congenital factors and changes related to ageing will increasingly outweigh the
Paradoxically, the situation seems less clear as regards faecal incontinence, despite its being associated with the one type of pelvic floor trauma that is often clearly evident at the time of delivery, i.e. anal sphincter trauma. Epidemiologic evidence generally does not support a protective effect of caesarean section in comparison with normal vaginal delivery [9**,10], although much of the data is diluted by caesarean section performed in labour. The risk of anal incontinence seems significantly higher after forceps delivery [9**,10–12], however, and older age at first delivery is another risk factor [9**].

Our knowledge base regarding the impact of vaginal childbirth on the anatomy and function of the female pelvic floor has increased markedly over the past two decades. While it was established in the 1980s and 1990s that vaginal childbirth (or even just the attempt at vaginal delivery) may significantly change pudendal nerve conduction patterns [13,14], the limited usefulness of tests of nerve function in clinical practice [15] has cast doubts on the relevance of denervation as a major aetiological factor in pelvic floor disorders. Concentric needle electromyography may provide more accurate information but is technically difficult and unlikely to attract many volunteers. It is not surprising therefore that little recent electrophysiologic work has been reported, although what there is tends to confirm previous findings of electromyographic alterations in parous women [16–18]. This review therefore focuses on imaging evidence accumulated over the past 5 years.

Recent investigations using techniques such as magnetic resonance imaging (MRI) and three-dimensional or four-dimensional ultrasound have focused on pelvic floor support, the integrity of fascial support structures, function and morphology of the levator ani muscle, and the external and internal anal sphincter. While the relative roles of muscle and fascia for pelvic organ support continue to be debated, both seem to be subject to delivery-related trauma.

**Pelvic organ support**

Clinical studies of pelvic organ support were until recently limited by a lack of sufficiently sensitive tools for prolapse assessment. This has changed with the introduction of the pelvic organ prolapse quantification system (POP-Q) [19]. Vaginal parity seems to be a risk factor for pelvic organ prolapse as defined by the POP-Q assessment [20] and is associated with higher degrees of pelvic organ mobility as shown by POP-Q in young women [21], but studies comparing antepartum with postpartum findings are scarce due to the fact that even such a simple assessment is invasive and less well tolerated in pregnancy and puerperium.

Several authors have demonstrated increased pelvic organ mobility in parous women on using real-time ultrasound imaging, whether in cohorts of symptomatic older patients presenting to urogynaecology clinics [22] or in the puerperium. There are few data on the effect of pregnancy itself [23], but several authors have observed the effect of labour and delivery, examining women both before and after childbirth [24–27]. From this evidence, it seems clear that prelabour caesarean section and caesarean section in first stage result in little change in bladder neck support. Vaginal delivery, conversely, in particular operative vaginal delivery, is associated with a highly significant increase in bladder neck mobility. An example of markedly increased bladder and urethral mobility after a vacuum extraction for failure to progress in second stage is shown in Fig. 2 [27].

There seems to be sufficient proof for the hypothesis that pelvic organ support can be impaired by vaginal childbirth. It is unclear whether this effect is due to stretching or avulsion of structures and whether the observed changes are primary (i.e. directly due to childbirth) or the medium-term or long-term consequence of levator impairment. Several mechanisms may well coexist in one individual. Risk factors are operative vaginal delivery, prolonged second stage, and possibly high birth weight. The extent of such trauma, however, clearly varies from one person to the next.

Furthermore, it has recently been shown that any delivery-related changes occur against the background of marked variations in pelvic organ support in young
nulliparous women [28]. As the most significant changes are observed in those with the least organ mobility antenatally [29], the effect of childbirth may be a partial equalization of those interindividual differences. We are not currently able to distinguish between preexisting or postpartum (postdelivery) prolapse, but recent developments hold out promise that this may soon change.

Direct evidence of fascial trauma
Several attempts have been made to define fascial trauma after vaginal delivery. In the anterior compartment it has long been assumed that childbirth may result in disruption of the ‘endopelvic fascia’, in particular of paraurethral and paravaginal structures. To date, such attempts have generally been unsuccessful, although transrectal three-dimensional or four-dimensional ultrasound using high-frequency transducers may be able to identify the subvesical part of the endopelvic fascia. Contrary to what has been surmised in the past, such defects may be multiple and, analogous to striae gravidarum, too complex to invite surgical reconstruction.

As opposed to the situation regarding the anterior compartment, posterior compartment prolapse commonly provides indirect evidence of fascial trauma, due to the distinct appearances of a ‘true’ rectocoele, which is thought to represent a defect of the rectovaginal septum or Denonvillier’s fascia. Analogous to increased bladder descent after childbirth, there is a highly significant increase in caudal displacement of the rectal ampulla after childbirth [27]. In addition, however, it has recently been shown that vaginal childbirth also results in an increased prevalence of

Figure 2 Bladder neck mobility on Valsalva manoeuvre at 37 weeks (a) and 3 months postpartum (b)

Markedly increased bladder and urethral mobility after a vacuum extraction for failure to progress in second stage. Reproduced with permission from [27].

Figure 3 Comparison of posterior compartment imaging (midsagittal plane) in the third trimester (a) and 3 months after normal vaginal delivery (b), on maximal Valsalva manoeuvre

The anorectal junction appears normal on the left. On the right, there is a rectocoele of a depth of about 2 cm, filled with stool. Reproduced with permission from [30].
true rectocoele, i.e. presumptive defects of the rectovaginal septum, and that existing defects seem to grow larger [30]. Such defects are strongly associated with symptoms of pelvic organ prolapse and obstructed defecation [31]. Figure 3 [30] shows a case of de-novo true rectocoele 3 months after vaginal delivery.

**Levator trauma**

What clinicians and physiotherapists call 'the pelvic floor' is, for practical purposes, the pubococcygeus–puborectalis complex. This muscle complex forms a V-shaped or U-shaped sling around the anorectal junction, inserting on the pelvic sidewall from the pubic rami to the ischial spine. The levator hiatus, i.e. the space bordered by this sling, contains the urethra anteriorly, the vagina centrally, and the anorectum posteriorly (see Fig. 4 [32**] for a comparison of MRI and ultrasound imaging of the hiatus in the axial plane).

The area of the levator hiatus in young nulliparous women varies from 6 to 36 cm² on Valsalva manoeuvre [33*]. The area of the average fetal head in the plane of minimal diameters measures 70–90 cm² (equivalent to a head circumference of 300–350 mm), requiring marked distension and deformation of the levator complex. Lien et al. [34] in Ann Arbor, Michigan, have been able to show with the help of MRI-based computer modelling that the most inferior and medial parts of the levator complex may have to increase in length by a factor of 3 or more during crowning of the fetal head. Given this degree of acute distension, it is remarkable that many women seem to go through childbirth without sustaining major soft tissue trauma.

Evidence on the effects of childbirth on levator structure and function is available both from clinical research on pelvic floor function, usually obtained by physiotherapists, and from imaging. Several papers over the past decade have described patterns of injury observed on MRI, although no comparative study of antepartum and postpartum levator anatomy has been published to date. From studies in parous women [35,36], it has been speculated that the change in the typical H-shaped appearance of the vagina may be due to traumatic loss of paravaginal supports unilaterally or bilaterally. Hoyte et al. [37] have shown a significant decrease in levator muscle volume and increase in levator hiatus width in women with stress incontinence and prolapse. Other authors [38] have attributed abnormalities in density and structure of the levator ani to childbirth-related trauma.

With the advent of sonographic three-dimensional imaging techniques, pelvic floor ultrasound is now also capable of demonstrating both the normal pubococcygeus–puborectalis complex and abnormalities seen in parous women [32**,39,40**]. Recent technologic developments such as speckle reduction algorithms and tomographic or multislice imaging have markedly increased resolutions and now allow quantification of levator trauma in all dimensions [41] (Figs. 5–8). As a result, four-dimensional pelvic floor ultrasound now achieves spatial resolutions similar to or better than MRI in all three dimensions, while temporal resolutions are markedly superior to MRI, allowing the real-time observation of manoeuvres.

Presence and extent of levator defects seem to be associated with symptoms and signs of pelvic organ prolapse, especially of the anterior and central compartment [32**,40**,41]. While levator avulsion was observed in 11% of women with normal uterine support, it was found in 25% of all women with uterine descent and in about

**Figure 4 A comparison of magnetic resonance (a) and three-dimensional pelvic floor ultrasound (b) imaging of the pubovisceral muscle in a 23-year-old nulliparous volunteer**

The pelvic floor consists of the pubococcygeus–puborectalis complex, which forms a V-shaped or U-shaped sling around the anorectal junction, inserting on the pelvic sidewall from the pubic rami to the ischial spine. The levator hiatus, i.e. the space bordered by this sling, contains the urethra anteriorly, the vagina centrally, and the anorectum posteriorly. Reproduced with permission from [32**].
half of those with second-degree or third-degree uterine prolapse [40**]. There appears to be a link between levator trauma and worsened or de-novo stress incontinence 3 months postpartum [32**], although this association seems to disappear later in life [40**], an observation that accords with the fact that epidemiologic evidence demonstrates much stronger associations between childbirth and prolapse than between parity and stress incontinence (Fig. 1). Avulsion of the puborectalis may be the cause of stress incontinence, or it may just be a marker for (currently undetectable) trauma to fascial supports of the urethra and anterior vaginal wall.

Major trauma to the levator ani, as described in Fig. 5 [40**] and Figs 6–8, is palpable and was first described in 1943 [42]. Palpation of the levator ani by physiotherapists and gynaecologists or urogynaecologists does not currently include a morphologic assessment, however, and the digital diagnosis of such defects may require significant teaching. In a recent study comparing three-dimensional ultrasound and levator palpation by a conventionally trained physiotherapist, agreement between the two methods was very poor [43], but another paper comparing digital assessment by a specifically trained physician and MRI showed better agreement [44*].

Figure 5 A comparison of magnetic resonance (a) and ultrasound (b) imaging in patients with a typical right-sided avulsion injury (arrows) to the pubovisceral muscle

Reproduced with permission from [40**].

Figure 6 Small unilateral trauma to the left anterior aspect of the pubovisceral muscle (asterisk), as seen in the axial plane on translabial three-dimensional/four-dimensional ultrasound

(a) Image obtained at 37 weeks; (b) 4 months postpartum.
There seems to be an association between age at first delivery and levator trauma [32**,45,46**]. This observation agrees with recent epidemiologic data from Norway. Rortveit and Hunskaar [47] showed in a reanalysis of the EPINCONT study (Epidemiology of Incontinence in the County of Nord-Trøndelag) that stress trauma following vaginal delivery [533,533].

Figure 7 Major bilateral trauma to the pubovisceral muscle (asterisks), as imaged on translabial three-dimensional/four-dimensional ultrasound. Both insertions of the pubovisceral muscle have been avulsed from the pelvic sidewall. (a) Image obtained at 36 weeks; (b) 3 months postpartum.

Figure 8 Bilateral avulsion injury to the pubovisceral muscle 4 years after rotational forceps delivery (asterisks), associated with third-degree cystocele and second-degree uterine prolapse, as seen on tomographic ultrasound imaging, translabial three-dimensional/four-dimensional ultrasound. The figure shows a coronal reference plane (top left) and eight successive axial planes from 5 mm caudal to the hiatus to 15 mm cranial to the hiatus, showing the bulk of the pubovisceral muscle avulsed from the pelvic sidewall on both sides.
incontinence was more likely in women who had had their first baby at an older age, a finding that has since been confirmed by others [48**]. These observations may have significant public health implications, as in Western societies the age of primiparae has risen by about 10 years over the past two generations.

Childbirth does not just affect morphology or macro-anatomic appearances, it seems to have an effect on biomechanics as well. Childbirth clearly changes both dimensions and distensibility of the hiatus [32**,49], as shown in Fig. 9, but it is currently unclear whether this is due to functional or morphologic change.

**Anal sphincter trauma**

Anal sphincter injury is a form of pelvic floor trauma familiar to all practising obstetricians, and (since it is generally obvious to the naked eye) there seems little scope for debating a causal link with childbirth. While primary repair is undertaken at delivery as a matter of course, the quality of both detection and repair of such trauma seems to vary significantly. Anal sphincter

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**Figure 9** Hiatal dimensions before (a) and 4 months after vaginal delivery (b) as seen on maximal Valsalva manoeuvre in the axial plane

These images were obtained by translabial four-dimensional ultrasound, with the plane of minimal dimensions identified in the midsagittal plane and rotated through 90° to obtain a representation of the hiatus in the axial plane. It is evident that hiatal dimensions – both diameters and area – are increased in the postpartum image on the right.

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**Figure 10** Appearances on translabial ultrasound, coronal plane, in three women (a–c) 6–8 weeks after primary repair of third-degree anal sphincter tears

Scars or defects are indicated by asterisks.

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injuries may well occur more frequently than previously reported, in up to one-quarter of vaginal deliveries, with lower estimates due to ineffective intrapartum detection rather than covered 'occult' defects [50]. The main risk factor is considered to be instrumental vaginal delivery [51]. Anal incontinence is common after third-degree and fourth-degree tears, even if they are recognized and repaired at the time of injury, and can have a major effect on a woman’s quality of life. As in other pelvic floor disorders, however, the condition may be influenced by several other aetiologic factors such as chronic diarrhoea, anorectal surgery, cognitive impairment, and neuropathy (either as a result of traumatic childbirth or due to other causes). Not surprisingly, the strength of the association between obstetric factors and faecal incontinence varies with the age of the population under scrutiny [9]. It seems that many if not most women remain asymptomatic after anal sphincter injury [52]. Conversely, evidence of old sphincter trauma is found in more than 70% of women with late-onset faecal incontinence [53].

The anal sphincter is generally imaged by endoanal ultrasound, using high-resolution probes with a field of vision of 360°. This method is firmly established as one of the cornerstones of a colorectal diagnostic workup for anal incontinence and is covered extensively in the colorectal and radiologic literature [54–56]. Due to the limited availability of such probes, obstetricians and gynaecologists have taken to using high-frequency curved array probes placed exoanally, i.e. transperineally [57,58,59] in the coronal rather than the midsagittal plane.

On exoanal or translabial ultrasound, the external sphincter appears as an isoechoic to hyperechoic ring structure, while the internal anal sphincter is hypoechoic. Anal sphincter defects appear as a discontinuity of these ring structures (Figs 10 and 11). In the coronal plane, defects are conveniently described using a clock face notation. In the longitudinal plane, sphincter defects can be described by measuring defect length relative to total sphincter length.

After repair of third-degree and fourth-degree tears, ultrasound commonly demonstrates residual defects, and the extent of such incompletely or inadequately repaired defects seems associated with decreased sphincter pressures and an increased risk of anal incontinence [60]. Figure 10 shows the degree to which appearances in the coronal plane may vary after primary repair of third-degree anal sphincter trauma, and Fig. 11 illustrates the potential cranial extent of such trauma.

**Conclusion**

There is little doubt that some women suffer significant trauma to pelvic floor structures as a consequence of (successful or unsuccessful) attempts at vaginal childbirth. Trauma may affect the pudendal nerve or its branches, the anal sphincter, the puborectalis–pubococcygeus complex, or pelvic fascial structures. The more protracted a delivery is and the longer the duration of second stage, the higher seems the likelihood of anatomic or functional alteration, although some forms of trauma may also occur as a result of precipitate labour. Vaginal operative delivery seems to be a risk factor for all forms of impairment mentioned above, whether independently or due to its association with prolonged second stage. Changes in demographics and obstetric practice may influence the likelihood of pelvic floor trauma and future incontinence or prolapse. Ever-increasing caesarean section rates may ultimately prevent the need for pelvic floor surgery in some women, but for the next few decades we are likely to see an increase in the prevalence of pelvic floor morbidity, especially pelvic organ prolapse, due to delayed childbearing.

Delivery-related pelvic floor trauma is a reality, not a myth. It is an entirely different question, however, as to whether such trauma is common or severe enough to require a change in clinical practice. Currently, we cannot

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**Figure 11** Appearances on translabial ultrasound of a major fourth-degree tear

(a) Coronal plane and (b) midsagittal plane. EAS, external anal sphincter, IAS, internal anal sphincter. Image courtesy of Dr A. Steensma, Rotterdam.
be sure whether avoidance of potential intrapartum pelvic floor trauma is worth the risk, cost, and effort of performing an elective caesarean section. In order to make preventive intervention feasible, we may first have to learn to identify women most at risk of delivery-related pelvic floor trauma, and this should be a priority topic for future research. Ultimately, however, only randomized controlled trials of planned vaginal birth versus planned caesarean section, whether in high-risk patients or in an unselected population, will eventually provide meaningful information to women and their healthcare providers.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

● of special interest
**● of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 577–578).


17 Dietz HP. The classification of major pathological abnormalities of the pubovisceral muscle. Neurourol Urodyn; 2006; 25 In press.


First ultrasound study to estimate the prevalence of levator avulsion injury in symptomatic women and the first to show an association between such trauma and pelvic organ prolapse.
This paper demonstrates that levator trauma identified on imaging can be palpated vaginally, although palpation requires significant training and may underestimate the extent of trauma.
45 Dietz HP. Does delayed childbearing increase the risk of levator injury in labour? Neurourol Urodyn. 2006; 25 In press.
This paper investigates obstetric risk factors for levator trauma and confirms that older age at first delivery seems to be a risk factor.
Another outstanding paper that has arisen from the study mentioned in MacArthur et al. (Br J Obstet Gynaecol 2005; 112:1075–1082).
A very interesting study suggesting that ‘occult’ anal sphincter injuries are generally visible on diligent examination and that previous low prevalence estimates for anal sphincter injuries are due to our failure to detect sphincter trauma.
Interesting paper describing the use of three-dimensional pelvic floor ultrasound for the assessment of anal sphincter tears.
60 Starch M, Bohe M, Valentin L. The extent of endosonographic anal sphincter defects after primary repair of obstetric sphincter tears increases over time and is related to anal incontinence. Ultrasound Obstet Gynecol 2006; 27:188–197.
Important longitudinal study showing that most women show evidence of residual sphincter defects after repair and that the extent of such defects is associated with contractile function and symptoms of anal incontinence.