Pelvic floor trauma in childbirth – Myth or reality?

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Abstract
The issue of traumatic damage to the pelvic floor in childbirth is attracting more and more attention amongst obstetric caregivers and laypersons alike. This is partly due to the fact that elective Caesarean section, as a potentially preventative intervention, is increasingly available and perceived as safe. As there are a multitude of emotive issues involved, including health economics and the relative roles of healthcare providers, the discussion surrounding pelvic floor trauma in childbirth has not always been completely rational. However, after 25 years of urogynaecological research in this field it should be possible to determine whether pelvic floor trauma in childbirth is myth or reality.

On reviewing the available evidence, it appears that there are sufficient grounds to assume that vaginal delivery (or even the attempt at vaginal delivery) can cause damage to the pudendal nerve, the caudal aspects of the levator ani muscle, fascial pelvic organ supports and the external and internal anal sphincter. Risk factors for such damage have been defined and variously include operative vaginal delivery, a long second stage, and macrosomia. It is much less clear, however, whether such trauma is clinically relevant, and how important it is in the aetiology of pelvic floor morbidity later in life.

Key words: birth trauma, incontinence, levator muscle, magnetic resonance imaging, pelvic floor, prolapse, ultrasound

Introduction

The continuing trend towards elective Caesarean section1,2 is at least partly due to a growing awareness of potential negative effects of (attempted or successful) vaginal childbirth. Patients and their doctors increasingly opt for elective abdominal delivery in an attempt to avoid future morbidity such as urinary and faecal incontinence or prolapse, all of which have been associated with vaginal parity in epidemiological studies.3-5 Meanwhile, both older age at first birth and the obesity epidemic increase the likelihood of problems in labour such as emergency operative delivery.6,7

There is, however, no consensus as to whether it is pregnancy or childbirth that is to blame for the higher rates of pelvic floor morbidity observed in parous women.8,9 The protective effect of caesarean delivery appears to be only partial;9 and long latencies between the presumptive cause (childbirth) and effect (incontinence and prolapse) have hindered clinical research in this field. As more research widens our knowledge of pelvic floor disorders, the discussion of elective caesarean section as a preventative measure continues.

The two opposing camps have been well-defined by a recent editorial in the International Urogynecology Journal and an opinion piece in the British Journal of Obstetrics and Gynaecology.10,11 Willy Davila, a subspecialist urogynaecologist with strong opinions on the perils of vaginal delivery, claims that there is overwhelming evidence for adverse effects of vaginal childbirth, and that we ought to obtain informed consent prior to an attempt at normal delivery. Drs Bewley and Cockburn, both practising obstetricians with a non-interventionist philosophy, state emphatically that pelvic floor trauma in childbirth is a myth, and that the trend towards elective caesarean section is misguided at best, or an attempt by unscrupulous colleagues to enhance their role and income at worst.

Where do we stand in 2004? Is delivery-related pelvic floor damage myth or reality? One should be able to answer this question from data accumulated in the last 25 years. The evidence might be considered separately for:

- Adverse effects on nerve structure and function
- Adverse effects on pelvic floor muscle structure and function
- Adverse effects on structure and function of the external anal sphincter
- Adverse effects on pelvic organ support

Epidemiological data will be considered to help elucidate whether such adverse effects are in fact clinically relevant.

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Adverse effects on nerve structure and function

It has been postulated that labour and vaginal delivery, and in particular the second stage of labour, might have a negative effect on nervous structures supplying pelvic organs and the pelvic floor. While this might also be true for the autonomic innervation of bladder and anorectum, the best-investigated structure in this regard is the pudendal nerve and its branches. Due to its location and ease of access to innervated structures such as the pubococcygeus – puborectalis complex and the anal sphincter, external electrodes can be used in a minimally invasive fashion for electrophysiological research. If necessary, both the levator ani and the external anal sphincter are easily identified and investigated with needle electrodes.

Two groups in particular have studied the effects of childbirth on the pudendal nerve. Allen et al. used an invasive technique, concentric needle electromyography (CN-EMG), to investigate these issues. The technique involves placement of a needle electrode in the muscle in question in order to register electrical activity during voluntary contraction. The main parameter indicative of neuropathy in CN-EMG studies is an increase in motor unit potential (MUP) duration which is associated with reinnervation, i.e. nerve damage and subsequent repair. Such changes were observed in the levator ani of 80% of women after vaginal delivery, with the mean MUP duration increased from 3.3 ms antepartum to 5.2 ms at 2-months postpartum. A long second stage of labour and high birthweight seemed associated with more severe changes. Caesarean section was protective, although protection was only complete in women delivered electively. The authors concluded that vaginal delivery (or even the attempt at vaginal delivery) causes partial denervation of the pelvic floor in most women. As they also measured pelvic floor contraction strength manometrically, Allen et al. were able to correlate neuropathic changes with function. Not surprisingly, they observed a reduction in mean maximal perineometric contraction pressure from 15.6 cm H₂O to 10.1 cm H₂O at the postpartum visit, confirming that the neuropathological changes observed on CN-EMG were associated with impaired levator function.

Snooks et al. focused on the effect of childbirth on the external anal sphincter, using pudendal nerve terminal motor latencies (PNTML) as their main outcome parameter. This technique involves trans-anal stimulation of the pudendal nerve with a finger-mounted electrode, while a second, lower electrode at the base of the finger picks up the resulting muscle action potentials in the external anal sphincter. The same electrode setup can be used to stimulate intravaginally and register muscle potentials in the pubococcygeus/puborectalis muscle.

In studies spanning more than 5 years, Snooks et al. were able to show that pudendal nerve terminal motor latencies as measured at the external anal sphincter were increased after childbirth, indicating pudendal nerve damage. Forceps delivery, length of second stage and birthweight were the main predictors of such changes. Compared to controls, PNTML was increased from 1.9 ms in control subjects to 2.2 ms in normal vaginal delivery and 2.4 ms in forceps delivery, and Caesarean delivery seemed protective of such changes. In a follow-up study undertaken 5 years later, the authors concluded that pudendal neuropathy persisted, and might even become more marked over time. They also found an association between abnormal fibre density measurements/increased PNTML 5 years after childbirth and stress urinary incontinence and anal incontinence, although numbers were low.

A number of other authors have used PNTML to investigate pelvic floor innervation. In a clinical context, PNTML are of questionable clinical usefulness but there is corroborating evidence that PNTML readings are prolonged after vaginal childbirth implying a degree of neuropathy in some women. There seems to be some recovery over the first 3 months postpartum, and virtually no change over time from then on.

Indirect evidence for denervation has been obtained from levator ani biopsies although this data is controversial, and the urethral rhabdosphincter, likely more important for continence, seems too small a structure to reliably investigate with current neurophysiological techniques such as concentric needle EMG. The effect of childbirth on the rhabdosphincter is controversial, with some authors postulating partial denervation due to childbirth and others disputing this concept. In symptomatic women at least, maximum urethral closure pressure is associated with parity, which would lend support to the concept of denervation injury extending to the urethral musculature.

Despite the fact that a number of important questions remain to be answered, there seems little doubt that vaginal childbirth can have significant negative effects on the pudendal nerve and its branches in some women. The extent of the resulting deterioration in nervous function seems to be associated with length of second stage rather than mode of delivery. This implies that even an attempt at vaginal delivery that ends in an emergency Caesarean section after full dilatation might have adverse effects on pelvic floor innervation. The clinical relevance of these findings however, remains to be defined.

Adverse effects on pelvic floor muscle structure and function

What we call ‘the pelvic floor’ is, to a large extent, the pubococcygeus-puborectalis complex. This muscle complex forms a V-shaped sling running from the pelvic sidewall on one side, posteriorly around the anorectal junction, and back towards the contralateral pelvic sidewall. The levator hiatus, i.e. the space between the arms of the V, contains the urethra anteriorly, the vagina centrally and the anorectum posteriorly. The area of the levator hiatus in young nulliparous women varies from 6 to 36 cm² on Valsalva manoeuvre.

The area of the average fetal head in the plane of minimal diameters measures 70–100 cm² (equating to a head circumference of 300–350 mm), requiring marked distension and deformation of thelevator complex. There has been some interesting work on the mechanics of levator deformation in labour recently.
John De Lancey’s group in Ann Arbor, Michigan, has been able to show, with the help of MRI-based computer modelling, that the most inferior and medial parts of the levator complex have to increase in length by a factor of 3.5 during crowning of the fetal head.26 Given this degree of acute distension, it is remarkable that many women seem to go through childbirth without sustaining muscular 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. There have been a number of papers over the last decade describing patterns of injury observed on magnetic resonance imaging (see Figs 1–3). Static images allow a morphological assessment of the pelvic floor muscles. Dynamic imaging during strain and contraction gives insight into function and changes that might be related to pregnancy and childbirth. Because of the complexity of the pelvic floor anatomy, the review in multiple planes is most helpful. Potential disadvantages of MRI are the cost, possible difficulty in access to a suitable scanner and compliance issues affecting research activities.

In the sagittal plane, the point of reference is the pubococcygeal line (PCL). Pelvic organ descent is defined by the distance of the organ to the PCL.27 On axial views the signal intensity and integrity of the puborectalis muscle and its changes can be shown (Figs 1–3). Any disruption of the muscle extending from the symphysis pubis/pelvic sidewall around the rectum to the contralateral symphysis pubis/pelvic sidewall can be identified as tearing or loss of substance (Figs 2 & 3). Vaginal shape and width of the levator hiatus has been used in the assessment of pelvic floor relaxation. Increased levator width and ballooning with strain is indicating increased pelvic floor relaxation. The change of the typical H shape appearance of the vagina might be due to loss of the paravaginal supports uni- or bilaterally.26,29 Hoyte et al. have shown a significant decrease in levator muscle volume and increase in levator hiatus width between normal and stress incontinent women and women with prolapse.30

With the advent of sonographic 3D imaging techniques, pelvic floor ultrasound is now also capable of showing both the normal pubococcygeus – puborectalis complex and abnormalities seen in parous women.25,31 In Figure 4, the standard acquisition mode of 3D pelvic floor ultrasound is shown. Three orthogonal planes (sagittal plane top left, coronal plane top right, axial plane, bottom left) are represented as well as a rendered volume showing the pubococcygeus/puborectalis...
complex appearing as a V-shaped structure surrounding the urethra and vaginal anteriorly and the rectum posteriorly (bottom right) in this asymptomatic 36-years-old p3. Figure 5 shows measurements of the levator hiatus at rest and on Valsalva in a 21-year-old asymptomatic nulliparous woman. These measurements are taken in the axial plane at the level of minimal hiatal dimensions. Both Figures 4 and 5 illustrate intact anatomy, as opposed to Figure 6 in which a unilateral right – sided detachment of the levator ani from the pelvic sidewall is shown in a rendered volume. While no significant asymmetries were found in a series of 49 nulligravid women,25 approximately 5–10% of parous symptomatic women show evidence of such partial separation of the puborectalis/pubococygeus complex from the arcus tendineus of the levator ani.31 In a smaller number of women, unilateral atrophy was found which might well be neuropathic in origin. Figure 7 shows a rendered volume of the puborectalis at 38 weeks and 3-months postpartum from an ongoing unpublished study (HPD), with a right-sided avulsion injury clearly visible 3 months after a normal vaginal delivery. This particular patient complained of significant de novo stress incontinence postpartum.

Two of the main advantages of 3D pelvic floor ultrasound are the ease with which a dynamic assessment can be obtained in real-time, and the fact that true volume data is obtained, allowing the assessment of any arbitrarily defined plane at postprocessing. The dimensions of the levator hiatus vary markedly in nulliparous women, and width and area of the hiatus seem associated with parity and prolapse.31 Table 1 gives an ANOVA graph of vaginal parity versus the dimensions of the levator hiatus in a group of 189 women seen with symptoms of pelvic floor dysfunction (unpublished data). The area of the levator hiatus at rest is seen to be clearly associated with vaginal parity.

There is some clinical data on pelvic floor function before and after childbirth, and a number of investigators have reported reductions in levator strength as measured by perineometry, palpation,12–35 or bladder neck displacement on levator contraction.36 Such changes seem to correlate with neurophysiological evidence as discussed above.12 It is intriguing that all investigational methods have shown a similar effect, that is, a 25–35% reduction in levator function postpartum. In some instances, a long second stage was shown to be a risk factor.12,36

**Adverse effects on structure and function of the external anal sphincter**

It has been known, for a long time, that anal sphincter trauma is a not uncommon consequence of vaginal childbirth, with an incidence of between 1 and 5%. The most important risk factors seem to be primiparity, large babies and vaginal operative delivery.37 The effect of childbirth on sphincter innervation has already been discussed.14 On ultrasound, structural alterations of the external anal sphincter are seen more commonly, with occult trauma being observed in up to 38% of vaginal deliveries.38,39 While the consequences of such trauma are occasionally severe and clearly apparent in the
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short or medium term (such as rectovaginal fistula),\textsuperscript{37} in most instances primary surgical repair seems effective, and women remain asymptomatic. It seems that, while trauma to the anal sphincter undoubtedly occurs, the extent of functional impairment is much less well defined. Faecal incontinence is a complaint with a multifactorial aetiology, and elective caesarean section does not provide more than partial protection.\textsuperscript{37,40,41} Figure 8 shows exoanal ultrasound after early postpartum repair of complete third degree tears of the external anal sphincter.\textsuperscript{42} Despite standardised repair techniques (overlapping technique), findings vary from no visible evidence of trauma (left) to major defects that are digitally palpable and often associated with symptoms (right).

Adverse effects on 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) system introduced by the International Continence Society.\textsuperscript{43} Parity seems to be a risk factor for pelvic organ prolapse as defined by the POP-Q assessment,\textsuperscript{44} although data are scarce due to the fact that even such a simple assessment is invasive and less well-tolerated in pregnancy and puerperium.

Thus, it is not surprising that most available data on the effect of pregnancy and delivery on pelvic organ support has been obtained by imaging, and translabial ultrasound has been most useful in this regard. A number of authors have shown increased pelvic organ mobility in parous women,

Table 1 Area of the levator hiatus at rest versus parity in a series of 189 symptomatic women (series unrelated to those given in Tables 2 & 3); ANOVA < 0.001. Means (*) and 95% CI (brackets)\textsuperscript{32}

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Figure 7 Rendered volume of the puborectalis (axial plane) at 38 weeks (left) and 3-months postpartum (right), with a rightsided avulsion injury clearly visible 3 months after a normal vaginal delivery. Such defects are generally palpable on vaginal examination. This particular patient complained of significant de novo stress incontinence postpartum.

Figure 8 Translabial exoanal ultrasound 3 months after primary repair of third degree tears of the external anal sphincter. The spectrum reaches from no evidence of trauma (left) to minor irregularities in the sphincter indicative of a scar (middle) and significant defects in the external sphincter (right) which are digitally palpable and, as in this case, sometimes associated with symptoms of anal incontinence.
whether in cohorts of symptomatic older patients presenting to urogynaecology clinics, but several authors have observed the effect of labour and delivery, examining women both before and after childbirth. Table 3 shows an ANOVA graph of changes in bladder neck mobility versus delivery mode, with forceps delivery being associated with the most marked changes. Prelabour caesarean section and caesarean section in the first stage of labour resulted in very little change in bladder neck support. An example of markedly increased bladder and urethral mobility, after a vacuum extraction for failure to progress in the second stage of labour, is shown in Figure 9.

There seems to be sufficient proof for the hypothesis that pelvic floor fascial support structures can be impaired by vaginal childbirth. It is unclear whether this effect is due to stretching or avulsion of structures, and both mechanisms might well coexist in one person. Some of the negative effect on pelvic organ support might also be due to impairment of muscular structures (see above). Risk factors are operative vaginal delivery (see Table 3), prolonged second stage, and possibly high birthweight. 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. As the most significant changes are observed in those with the stiffest support structures antenatally, the effect of childbirth might be a partial equalisation of those interindidual differences. Somebody presenting with a second degree cystocele at age 55 might have had this cystocele for all her life, or she might have acquired it due to a traumatic delivery. Currently, we are unable to distinguish clinically between pre-existing or postpartum (postdelivery) prolapse. It might soon become feasible to document newly developed fascial defects postpartum, especially with the help of 3D volume ultrasound, but such work has not progressed beyond the pilot stage.

**Clinical significance**

While there is a growing body of evidence supporting the contention that childbirth often has a deleterious effect on pelvic floor structures, the long latency of symptoms related to pelvic floor morbidity impede studies into the clinical relevance of delivery-related changes. Epidemiological studies are often much more equivocal than the pathophysiological findings described above, as the aetiology of prolapse, urinary and faecal incontinence is clearly multifactorial, and as the importance of a given aetiologial factor is likely to shift over a woman’s lifetime.

The situation regarding prolapse seems least controversial as vaginal childbirth is consistently found to be amongst the strongest risk factors for this condition. With regards to
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Several large epidemiological studies have shown that caesarean section provides only partial protection from stress incontinence and even this partial protection seems to fade with time. Congenital factors and changes related to ageing might increasingly outweigh the effects of traumatic childbirth as women grow older. This also holds true for faecal incontinence. It is quite irrelevant whether an 86-year-old nursing home inpatient with senile dementia has had her levator, endopelvic fascia or anal sphincter disrupted in labour. Her risk of urinary or faecal incontinence will be rather high regardless, and currently there is next to nothing we can do about that. One might argue, however, that loss of protection over time does not argue against the relevance of such a protective effect, in particular as this effect will apply during what might well be the most active decades of a woman’s life.

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 might affect the pudendal nerve or its branches, the anal sphincter, the puborectalis- pubococygeus complex, and/or pelvic fascial structures. The more protracted a delivery is, and the longer the duration of second stage, the higher the likelihood of anatomical or functional alteration. 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.

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 and/or severe enough to require a change in clinical practice. Currently, we cannot 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 preventative intervention feasible, we may first have to learn to identify those women most at risk of delivery-related pelvic floor trauma. This may not be as difficult as previously assumed. While there is no doubt that this topic will provide cause for controversial discussion in years to come, only randomised controlled trials will eventually provide meaningful information to women and their healthcare providers.

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Figure 9 Midsagittal translabial image of bladder neck and urethra, at rest and on Valsalva. The upper pair of images was obtained in a primigravida at 35 weeks, the lower pair of images in the same patient 3 months after a vacuum extraction for maternal exhaustion after 136 min in second stage. Bladder neck descent has increased from 6 mm to 38.1 mm.


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