Childbirth and the pelvic floor: “the gynaecological consequences”

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Abstract

This review addresses the effects of childbirth on the pelvic floor, urinary continence mechanisms and the perineum. Genitourinary prolapse affects 15% of women and stress incontinence 20–30%. The major risk factors are age and childbirth, with severity increasing with parity. There are three mechanisms of support for the pelvic organs and bladder neck. These are (i) the muscular component: levator ani and urethral sphincter with their intact nerve supply, (ii) the endopelvic fascial connections with the levator ani, and (iii) the posterior angulation of the vagina. Childbirth causes direct myogenic damage, dennervation and defects in the endopelvic fascia along with widening of the urogenital hiatus. Elective caesarean section without labour has in the past thought to be protective. More recent data suggests this effect to be less pronounced and antenatal stress incontinence appears the most important predictive factor for the development of postnatal stress incontinence. The targeting of pelvic floor exercises under direct supervision from a physiotherapist have shown a reduction in the development of short and long term stress urinary incontinence.

Perineal trauma can effect up to 85% of women after vaginal delivery. The consequences of this include perineal pain and dyspareunia lasting up to 12 months postnatally. Nulliparity and the use of forceps have been identified as the major risk factors along with occipito-posterior position, macrosomia and episiotomy as secondary factors. The role of selective mediolateral episiotomy and methods of perineal repair are discussed.

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1. Introduction

The strength of the muscles of the pelvic floor and other supporting structures of the pelvic organs are affected by various events that occur during a woman’s lifetime. Pregnancy and childbirth have a pronounced influence on maternal anatomy and physiology. After this, menopause and aging have a secondary effect on the pelvic floor. This review concentrates on the long term gynaecological consequences of pregnancy and childbirth with respect to pelvic floor weakness, its incidence, its pathophysiology and ways in which it may be prevented.

The longer-term gynaecological sequelae of pelvic floor weakness are pelvic organ prolapse, stress urinary incontinence, and perineal trauma, its subsequent repair, dyspareunia and perineal pain.

2. Epidemiology

Genitourinary prolapse is a common and distressing condition affecting up to 15% of the female population and is responsible for around 20% of women on waiting lists for major gynaecological surgery [1]. It is defined as the protrusion of a pelvic organ or structure beyond its normal confines within the pelvis. Epidemiological studies have shown a woman has an 11.1% lifetime risk of undergoing a single operation for prolapse or incontinence by the age of 80 [2]. The incidence of women admitted to hospital with prolapse is 2.04 per 1000 person-years of risk [3]. The main risk factors associated with prolapse are parity and increasing age, whilst smoking and obesity are secondary risk factors. Stress incontinence affects around 20–30% of women, the incidence increasing with parity [4]. The mechanisms of support for the bladder neck and anterior urethra are complex and involve the levator ani muscles and their endopelvic fascial connections, and the posterior angulation of the vagina [5]. The most important risk factors for the development of stress incontinence are age, parity, smoking and obesity [6]. The role of episiotomy and perineal repair in the prevention of stress urinary incontinence is discussed in detail elsewhere [7].

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3. Normal anatomy of pelvic support

The uterus and vaginal apex are supported by a muscular component, which requires an intact nerve supply, and a fascial component. Damage to any of these can lead to prolapse of the pelvic organs.

Victor Bonney described three mechanisms of support that exist in the female pelvis: constriction, suspension and flap-valve closure [17].

1. Constriction of the vagina by the levator ani occludes the levator hiatus through which prolapse of pelvic structures can occur.
2. The angulation of the vagina, which allows it to close against the levator plate on increasing the intra-abdominal pressure.
3. Fascia and ligaments suspend the uterus, bladder and bladder neck, and rectum to the pelvic sidewalls.

3.1. Muscular supports

The muscular support of the pelvic viscera is mainly provided by the levator ani muscles: iliococcygeus and pubococcygeus. The iliococcygeus muscles form a sheet-like structure extending from the arcus tendineus anteriorly, travelling behind the rectum to meet a midline raphe fused posteriorly to the coccyx. The two muscles act as a plate lying over the urogenital hiatus, upon which the pelvic viscera lie. When the levator ani muscles relax the urogenital hiatus widens and the suspensory ligaments, made of connective tissue are left holding the uterus and vagina in place. This has been confirmed using three-dimensional ultrasound of the levator ani hiatus. Measurements show that the hiatus is larger in women with prolapse than in those without [18]. Control of levator tone at rest and at stress is maintained by an intact nerve supply. The levator ani muscles are innervated by the anterior sacral nerve roots of S2–4. The pubococcygeus muscle forms a sling-like structure around the urogenital hiatus and so is involved in sphincteric mechanisms of the urethra. The pubococcygeus muscle, along with the external anal sphincter is innervated by branches of the pudendal nerve (S2–4).

3.2. Connective tissue supports

Integrity of the endopelvic fascia is also important in the support of the uterus, bladder and bowel. Anteriorly the pelvic fascia forms the pubourethral ligaments. These lie either side of the midline to form an aponuerotic plate supporting the cranial aspect of the proximal urethra. The pubourethral ligaments extend anteriorly becoming continuous with the suspensory ligament of the clitoris. The endopelvic fascia then extends as a fibrous band running from the symphysis pubis anteriorly, widening posteriorly to attach to the ischial spine. This structure called the arcus tendineus fascia pelvis (ATFP) is also known as the “white line”, and can easily be seen in the space of Retzius at colposuspension. The fascia extends continuously from the symphysis pubis anteriorly, the arcus tendineus and levator ani muscles laterally to the ischial spines posteriorly, enveloping the pelvic organs. In areas the fascia forms condensations called ligaments, e.g. the uterosacral/cardinal complex. The endopelvic fascia is made up mainly of connective tissue and smooth muscle in various proportions according to site and structure [19]. The cardinal ligaments contain perivascular connective tissue whereas the uterosacral ligaments are predominantly smooth muscle and connective tissue [20,21] (see Fig. 1). Mengert demonstrated in experiments on cadavers that the uterosacral ligaments, pararetrum and paracolpium are most important in the support of the uterus and vagina and this has been further shown to involve three levels of support to the pelvic viscera [22,23]:
Level 1 extends upper 1/3 of the vagina and the cervix suspending the uterus and vaginal apex to the lateral pelvic side walls.

Level 2 attaches the mid-portion of the vagina directly to the pelvic walls forming the pubocervical and rectovaginal fascia.

The Hammock hypothesis for urethral support was derived after anatomical dissection and correlation with magnetic resonance imaging [24]. The pubocervical fascia between the anterior vaginal wall and the bladder retains continuity with the arcus tendineus and the levator ani to form a “hammock” below the bladder neck and urethra. This continuity is important in maintaining continence of urine as well as supporting the bladder neck and urethra.

Level 3 direct attachment of lower portion of vagina to surrounding structures without paracolpium.

Loss of support at each level gives rise to different types of prolapse. Damage to the upper suspensory fibres of the paracolpium can lead to prolapse of the uterus and vaginal apex. Loss of level 2 support anteriorly would give rise to urethrocele, cystourethrocele or cystocele, often accompanied by stress incontinence. Loss of continuity of the rectovaginal fascia and the pelvic side-wall would lead to rectocele.

Support of the pelvic viscera is achieved by the interplay of the three mechanisms described. The analogy of a boat in dry dock has been used. The water supports the weight of the boat whilst the mooring ropes hold it in position. The water is analogous to the levator ani muscles and the mooring ropes to the endopelvic fascia and suspensory ligaments. If the water level were to fall significantly then excessive forces would be placed on the mooring ropes. After an assault such as childbirth, neuromuscular damage may cause a reduction in levator resting tone and a widening of the urogenital hiatus. The uterus and vagina are therefore only supported by the endopelvic fascia of the parametrium and paracolpium. If the fascia is inherently weak or already damaged then the ligaments will stretch and lead to prolapse.

4. Pathophysiology of prolapse and stress urinary incontinence: evidence of pelvic floor damage

The following factors are implicated in the aetiology of prolapse and stress urinary incontinence:

- Mechanical trauma to the pelvic floor musculature.
- Damage to the nerve supply to the pelvic floor muscles.
- Insult to the endopelvic fascia.

4.1. Myogenic damage

There is histological and imaging evidence that mechanical trauma causes rupture of the pelvic musculature at delivery. Studies using magnetic resonance imaging of the pelvic floor after vaginal delivery have demonstrated areas of muscle deficiency within the striated muscle of the pelvic floor as a direct consequence of childbirth [25]. Imaging of the levator ani has shown an association between severity of prolapse and loss of muscle size on measurement of the
pubococcygeus muscle, which correlated with muscle strength [26]. Measurements of the genital hiatus also show a correlation between increasing severity of prolapse and reduction in muscle bulk [27]. Histological examination of biopsies of levator ani from mice have shown characteristics of myogenic injury occur after parturition [28]. This is been supported by histological examination of human cadaveric levator ani where fibre density also suggested direct myogenic damage [29].

4.2. Neuromuscular injury

Damage to the nerve supply of the pelvic floor caused by childbirth may cause progressive denervation of the musculature. Subsequent re-innervation of the pelvic floor leads to an altered function, morphology and neurophysiology. Histological, histochemical and electromyography studies have measured the degree of re-innervation and have assumed this is the result of denervation caused by delivery [30,31]. The exact nature of denervation injury during parturition is unknown but one mechanism may be pudendal nerve compression. The pudendal nerves innervate the levator ani muscles from the second, third and fourth sacral nerve roots (S2–4). During its course from S2–4 to the levators, the pudendal nerve passes around the ischial spine which is a potential site for compression by the fetal head during childbirth. Prolonged pudendal nerve terminal motor latency (PNTML) has been reported after childbirth, but may also be as a result of other pathologies, such as demyelination. Snooks et al. showed that women having a vaginal delivery had significantly prolonged PNTML and greater perineal displacement on straining compared with controls [32]. PNTML are further significantly prolonged in primiparas who had a forceps delivery rather than a normal vaginal delivery. Caesarean section had no effect on neurophysiology compared with controls [32]. Although pelvic floor neuropathy has been demonstrated in many women immediately after delivery, most will recover neuromuscular function and only few will suffer long term problems. Sultan et al. measured PNTML at 34 weeks gestation and at 6 weeks and 6 months post-partum [33]. Their results supported the findings of Snooks et al., but also showed that PNTML can be prolonged in women who have a caesarean section after the onset of labour indicating that pudendal nerve damage may not be due to pregnancy but labour, irrespective of vaginal delivery. In view of the conflicting data on PNTML, the reliability of PNTML measurement is already being challenged.

It is not known why neuromuscular function is regained in some women after childbirth while others continue to have permanent damage. Decreased neuromuscular function can be seen in women with greater perineal body descent on straining. It has been postulated that repeated stretching of the pudendal nerve by straining causes recurrent injury to the nerve, preventing it from recovery [32–34]. Factors associated with greater pelvic floor nerve damage are parity, forceps delivery, prolonged second stage of labour, third degree perineal tears and macrosomia [35].

4.3. Damage to the endopelvic fascia

There are numerous studies that show prolapse occurs in association with defined defects in the supporting endopelvic fascia, which can be demonstrated on vaginal examination. At operation, defects can be dissected and repaired, akin to a hernia repair, as a treatment for prolapse [36,37]. During parturition tears in the connective tissue can occur which are not clinically apparent after delivery. With further vaginal deliveries and ageing the defects may become larger, more apparent and the bladder and bowel begin to protrude into the vagina resulting in a cystocele or rectocele. It is believed that some women are more susceptible to connective tissue damage during vaginal delivery due to an inherent weakness in their connective tissue. It has been suggested that if it were possible to isolate these individuals prior to pregnancy or labour then elective caesarean section may prevent/reduce their chance of developing prolapse. Collagen is the most important component of the connective tissue with regard to providing the tissue strength and prolapse is more common in women with disorders of collagen metabolism [38]. Altered collagen fibre orientation and thickness has been showed histologically in women with prolapse [39]. Urodynamic stress incontinence is increased in premenopausal nulliparous women with reduced collagen content in their anterior vaginal wall [11]. Jackson et al. showed a reduction in the total collagen content of vaginal fascia in premenopausal women with prolapse compared with controls [40]. In addition they showed a reduction in the ratio of stronger mature pyridinoline cross-link to the weaker cross-link hydroxylysino-ketonorleucine (HLKNL) and an increase in matrix metalloproteinase activity in this tissue, suggesting increased collagen degradation. However, the vaginal fascia does not support the uterus or bladder. Recent work has shown that similar (although less pronounced) changes in collagen metabolism occur in the uterosacral ligaments of women with uterine prolapse [41,42].

Pregnancy itself has an effect on the connective tissue. Landon et al. examined the biochemical properties of rectus sheath fascia in pregnant women compared with non-pregnant controls [43]. They found that during pregnancy the fascia becomes more elastic and weaker than outside pregnancy and women with stress incontinence showed exaggerated changes in fascial strength during pregnancy compared with those without stress incontinence.

5. Prevention

5.1. The role of elective caesarean section

A survey found 31% of female obstetricians would prefer elective caesarean section to vaginal delivery, with 80% of
those doctors indicating fear of perineal damage as their main reason [44]. Although caesarean section under regional anaesthesia is now safer than ever, maternal risks include haemorrhage, infection, ileus, pulmonary embolism and Mendelson’s syndrome. The risk of hysterectomy due to haemorrhage after caesarean section is 10 times that after vaginal delivery, whilst the risk of maternal mortality is 16-fold by caesarean section. Furthermore, the majority of damage to the pelvic floor occurs during the first vaginal delivery. It may therefore be better clinically to only offer caesarean section to women at a high risk of perineal floor damage if vaginal delivery occurred, in the absence of any other clinical indication. Some centres advocate caesarean section in women with a history of anal incontinence or who have had successful surgery for prolapse or urodynamic stress incontinence [45], as these conditions have been shown to deteriorate with subsequent vaginal deliveries. Chaliha et al. looked at antenatal urinary symptoms and physical markers of collagen weakness such as joint hypermobility, striae, perineal descent, prolapse, perineal body length and antenatal incontinence, in an attempt to predict post-partum urinary incontinence [13]. Unfortunately, they found no relationship with antenatal assessment and the development of urinary incontinence. King et al. found that the antenatal detection of increased bladder neck mobility on ultrasound predisposed to postnatal incontinence, however the specificity of this test does not allow this technique to be clinically useful [46].

Wilson et al. studied a population of around 7800 women for 6 years after delivery. They found that 27% of women who were incontinent at 3 months postnatal were continent at 6 years follow-up. Conversely 32% of women who were continent at 3 months postnatal had developed urinary incontinence after 6 years. They found the biggest predictor of stress incontinence at 6 years postpartum was the presence of stress incontinence prior to pregnancy. Caesarean section only appeared to be partially protective 6 years after delivery. For women who had two or three caesarean sections the group found the prevalence of urinary incontinence after 6 years was 32% compared with 46% in women who had a vaginal delivery. Mode of vaginal delivery (spontaneous, ventouse or forceps) did not influence outcome. The group estimated they would have to perform seven caesarean sections to prevent one woman from being incontinent which would not be economically or ethically viable [47].

5.2 Pelvic floor exercises

Perhaps a more appropriate form of prevention is antenatal pelvic floor exercises, as they are less invasive and have less morbidity and mortality compared with caesarean section. Properly performed pelvic floor exercises have been shown to be effective in the treatment of established urodynamic stress incontinence as well as reduce the incidence of short and longterm incontinence when performed postnataally, especially in the well motivated patient. [48,49]. However, they have not been shown to be effective in the treatment of established prolapse. Studies have shown that simple verbal advice and leaflets result in no improvement in the prevention of postnatal stress urinary incontinence [50]. Randomised controlled trials have shown that antenatal pelvic floor exercises lead to a reduction the development of short and long term postnatal stress urinary incontinence [51,52].

6. The perineum

Vaginal delivery can cause significant long term damage to the perineum including perineal discomfort, dyspareunia and anal sphincter damage and weakness. These symptoms are often long-standing and may require repeated gynaecological surgery. Therefore, the clinician at delivery should aim to minimise the amount of trauma encountered by the patient in an attempt to prevent later consequences.

6.1. Prevalence

An estimated 85% of women will sustain some degree of perineal trauma during vaginal delivery with 60–70% of these women requiring suturing [53]. Perineal discomfort and pain in the days after a vaginal delivery is common. Abraham et al. showed that perineal pain may persist up to 6 months after vaginal delivery (range 0–6 months), 20% experiencing discomfort for more than 2 months [54]. It took up to 3 months (range 1–12 months) for women to have sexual intercourse without discomfort. Twenty percent of women took longer than 6 months before sexual intercourse became comfortable. Factors associated with prolonged perineal discomfort and pain, are also associated with increased perineal trauma at the time of delivery such as the use of forceps and vaginal (not perineal) tearing. Disruption of the internal and external anal sphincters can lead to short and long term anal and faecal incontinence in up to 10% of women after vaginal delivery, especially if not recognised and corrected at primary repair [55–57].

6.2. Pathophysiology and prevention of perineal damage

Studies have also shown a relationship between perineal pain and the extent of perineal disruption [58,53]. The aim of the attending obstetrician/midwife should therefore be to minimise the degree of perineal trauma during vaginal delivery. guarding the perineum and applying pressure to the fetal head during normal vaginal delivery causes less post-partum pain than if the midwife keeps her hands poised, not touching the head or perineum [59]. Two recent large retrospective studies have identified major risk factors for perineal trauma and anal sphincter disruption during delivery. One reviewed the case notes of 2078 vaginal deliveries over a 2-year period examining maternal age,
race, weight, gestational age, parity, smoking, duration of first and second stage of labour, oxytocin usage, method of delivery (spontaneous, vacuum, forceps), infant birth weight, epidural usage and midline episiotomy [60]. Nulliparity and forceps delivery were recognised as risk factors for perineal and anal sphincter injury [61]. A further study looked at a database of 91,206 births over a 10-year period. Again nulliparity and forceps delivery were major risk factors for perineal laceration (RR 6.97 and 5.69, respectively). Occiput posterior position, second stage >120 min, delivery by an obstetrician, episiotomy and birth weight greater than 3000 g were secondary risk factors. Assisted vaginal delivery using vacuum extraction causes significantly less perineal trauma than forceps as well less disruption to anal sphincter function [62,63]. Two large randomised controlled trials have shown that antenatal perineal massage may have some benefit in reducing the incidence of second and third degree tears, episiotomy or instrumental delivery, particularly in nulliparous women aged 30 years and above [64,65].

6.3. The role of episiotomy

Historically, it was believed that episiotomy reduces perineal injury and anal sphincter tears by controlling the plane and extent of tissue damage. However, there is increasing evidence to suggest that elective episiotomy may not protect the perineum from trauma [66,67]. Median episiotomy, as practised in some countries such as the United States, is associated with a weaker pelvic floor and a greater incidence of tears involving the anal sphincter [60]. In addition spontaneous perineal tears are associated with less perineal pain and normal neuromuscular function on electromyography [68]. Mediolateral episiotomy, as practised in the United Kingdom, should only be used selectively. In a study published in the Lancet, 1555 nulliparous and 1051 primiparous women undergoing vaginal delivery were randomised to selective or routine mediolateral episiotomy [69]. Episiotomy was performed in 30.1% of women in the selective group and 82.6% of women allocated to receiving routine episiotomy. Although anterior perineal trauma was slightly increased in the selective group, posterior perineal surgical repair, perineal pain, healing complications and dehiscence were all less frequent in women receiving selective mediolateral episiotomy. One study has shown that nulliparous women have a higher risk of developing a third degree perineal laceration and that mediolateral episiotomy may prevent anal sphincter tears in these women, especially if other risk factors such as high birthweight and epidural analgesia were present [70]. A prospective survey of practice by obstetricians and midwives suggests obstetricians perform larger episiotomies which are angled further from the sagittal plane than midwifery practice [71]. Such differences in practice need to assessed by prospective observational studies along with association with outcome.

Inadequate repair of an episiotomy or vaginal tear can also lead to longstanding perineal discomfort and dyspareunia, and so attention to anatomy and good surgical technique is important. The type of suture material used, surgical technique and skill of operator are the three major factors that influence outcome after primary repair [72]. However, training of midwives and doctors in perineal anatomy appears to be poor with a minority of doctors and midwives aware of the structures cut whilst performing an episiotomy [56]. Immediate complications also include haemorrhage, infection and wound dehiscence, whilst long term complications include perineal pain and dyspareunia. The Royal College of Obstetricians and Gynaecologists recommend the use of an absorbable suture such as vicryl (or vicryl rapide) or dexon for perineal repair rather than catgut as they are associated with less perineal pain, analgesia used and wound dehiscence and that the repair should be in three layers using a loose non-locking technique for the vaginal tissues and perineal body and a subcuticular technique for the skin [72]. Two randomised controlled trials showed that a two-stage perineal repair leaving the skin unsutured significantly reduces pain and dyspareunia 3 months after delivery compared with the traditional three stage closure, with no increase in dehiscence after two stage repair [73,74].

7. Conclusions

Pregnancy and childbirth have a significant impact on the development of prolapse and urinary symptoms both during pregnancy and long term. Risk factors can be identified but as yet there are no clinical scoring systems in use to detect women at risk of long term postpartum symptoms. Prophylactic caesarean section does not represent a viable alternative due to its increased morbidity and mortality compared with vaginal delivery, as well as the significant cost implications. Properly trained antenatal pelvic floor exercises may help reduce the incidence of postpartum stress incontinence but are less useful in the treatment of established prolapse. Major risk factors associated with the development of perineal trauma are nulliparity and forceps delivery. Other secondary factors include length of second stage, macrosomia, degree of perineal trauma and the adequacy of the primary repair. Every attempt should therefore be made by the clinicians at delivery to reduce duration of second stage and perineal trauma as well as ensuring they are properly skilled in performing the primary repair.

8. Practice points

Prolapse and incontinence

- Support of the pelvic floor and continence mechanisms require a muscular component with an intact nerve supply and fascial connections.
- Direct myogenic injury, denervation, fascial defects and widening of the genital hiatus have all been implicated in the aetiology of prolapse and stress urinary incontinence.
- Elective caesarean section does not appear as protective for the pelvic floor as previously thought.
- The development of antenatal stress incontinence appears to be the single predictive factor for postnatal stress incontinence.
- Targeted antenatal physiotherapy has been shown to reduce stress incontinence long term.

Perineal injury

- Nulliparity and forceps delivery are the major risk factors for perineal trauma and postpartum pain and dyspareunia.
- Occipito-posterior position, episiotomy and birthweight greater than 3 kg are secondary factors.
- Elective episiotomy does not reduce the incidence of third degree tears but selective episiotomy may be protective.
- Absorbable suture such as vicryl (or vicryl rapide) or dexon should be used for perineal repair rather than catgut as they are associated with less perineal pain and wound dehiscence.

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