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Pelvic floor disorders following vaginal or cesarean delivery

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Abstract

Purpose of review—Pelvic floor disorders affect women of all ages and are associated with significant economic burden and poor quality of life. Current literature suggests an association between childbirth and these disorders. In this review, we summarize recent advancements in our understanding of this association.

Recent findings—Vaginal childbirth appears to be strongly associated with stress urinary incontinence and pelvic organ prolapse. There is less evidence to suggest an association between vaginal delivery and overactive bladder symptoms. History of more than one perineal laceration increases the likelihood of developing prolapse. Similar association has not been established for episiotomy. Disruption or denervation of structural components of pelvic floor support system, particularly levator ani muscle complex, is associated with later development of pelvic floor disorders. Imbalance in homeostasis of connective tissue remodeling of the vaginal wall from overstretching during childbirth is another possible mechanism.

Summary—Pelvic floor disorders represent a significant health problem affecting women of all ages. Identification of potential modifiable risk factors and advancement in understanding of the underlying pathophysiology is crucial for primary and secondary prevention of these disorders and for improvement in treatment strategies.

Keywords

prolapse; urinary incontinence; vaginal childbirth

INTRODUCTION

‘Pelvic floor disorders’ include stress urinary incontinence (SUI), overactive bladder, pelvic organ prolapse and fecal incontinence. Pelvic floor disorders affect 24% of US females [1]. The prevalence of these conditions increase with age: 39% of women aged 60–79 years and 50% of women aged 80 years or older suffer from at least one disorder [1]. The number of American women with at least one pelvic floor disorder will increase from 28.1 million in 2010 to 43.8 million in 2050 [2]. Thus, pelvic floor disorders are common and have significant public health impact.

In addition to the association with aging, pelvic floor disorders have an established association with childbirth. Specifically, these disorders are more prevalent among women who have delivered at least one child [1]. In this review, we summarize recent discoveries

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There are no conflicts of interest.

regarding the aspects of childbirth most strongly associated with the development of pelvic floor disorders later in life.

STRESS URINARY INCONTINENCE AND VAGINAL CHILDBIRTH

The International Continence Society defines SUI as involuntary loss of urine on effort or physical exertion or sneezing or coughing [3]. SUI is a common condition which affects women of all ages [4]. During the last 10 years, there has been a 27% increase in the rate of surgical management of SUI [5]. In addition to causing significant financial burden to healthcare system and the patient [6], this condition has significant negative impact on the quality of life of women [7].

Parity is a known risk factor for stress incontinence. Primiparous women have three times higher prevalence of stress incontinence than their age-matched nulliparous controls [8], and risk of stress incontinence surgery increases with increased parity [9▪]. Recent evidence suggests that mode of delivery has a significant impact. In a retrospective cohort study of parous women, the odds of bothersome symptoms of SUI doubled among women who had experienced at least one vaginal birth compared with women who delivered exclusively via cesarean [10▪]. In addition, history of operative vaginal birth has been associated with SUI [11▪▪]. The odds of surgical intervention for SUI is three times higher for women who delivered vaginally and 20 times higher for those who have experienced forceps assisted delivery compared with women who gave birth exclusively via cesarean [9▪].

OVERACTIVE BLADDER AND VAGINAL CHILDBIRTH

Urinary urgency is a sudden, compelling desire to pass urine which is difficult to defer. Overactive bladder is defined as urinary urgency (usually accompanied by frequency and nocturia), with or without urge incontinence, in the absence of urinary tract infection or other obvious disorder [3]. Overactive bladder is reported by 27–45% of women above age 40 years [12]. Recent estimates suggest that the annual economic cost of overactive bladder in the USA is more than US$ 60 million [13].

Compared with other pelvic floor disorders, the association between vaginal childbirth and overactive bladder is not well established. For example, it is not completely clear that overactive bladder is associated with parity: urge incontinence is reported by similar proportions of women delivered vaginally and nulliparous women [14]. Also, it is not clear that overactive bladder is associated with vaginal versus cesarean delivery. Specifically, the odds for overactive bladder in women 5–10 years after childbirth do not appear to be significantly increased after vaginal versus cesarean birth [10▪]. However, operative vaginal birth, particularly forceps delivery, may be associated with overactive bladder [10▪,11▪▪].

PELVIC ORGAN PROLAPSE AND VAGINAL CHILDBIRTH

Pelvic organ prolapse is defined as descent of the uterus and vaginal walls into the vaginal canal. Most women have at least some degree of prolapse. Objective prolapse severity is weakly correlated with symptom burden [15–17]. However, the general trend is that prolapse beyond the hymen is associated with an increase in symptoms [17–19], and therefore the hymen may represent a clinically significant threshold.

Women with symptomatic prolapse may experience a high degree of bother and substantial negative impact on physical function and quality of life [20]. In addition, the public health impact of prolapse is substantial with respect to the incidence of surgery: the lifetime incidence of surgically managed pelvic organ prolapse is as high as 19%, higher than previously estimated [21].

Research on the epidemiology of prolapse has been limited: most population-based studies of pelvic organ prolapse do not use objective measures for diagnosis of prolapse (e.g. a physical examination with the Pelvic Organ Prolapse Quantification System). Instead, most studies have used surrogate markers, such as prolapse symptoms [22,23] or surgical treatment [9▪,24]. These surrogate measures may not be reliable and can lead to biased estimates of prevalence [10▪].

Recent studies using a graded or quantitative physical examination suggest that prolapse is more common among parous versus nulliparous women [25]. In addition, vaginal childbirth, particularly operative vaginal delivery, increases the risk of pelvic organ descent to or beyond the hymen [10▪,11▪▪]. Six months postpartum, stage 2 pelvic organ prolapse was noted in 18% of Spanish primiparous women delivered vaginally compared with 7% of women who had cesarean birth [26]. Similar findings were noted in a multicenter US study [27]. Several studies support that the actual mode of delivery is more critical than the process of labor. For example, no difference was seen in prevalence of prolapse in women who delivered exclusively via unlabored cesarean compared with women who delivered abdominally after active labor and complete cervical dilatation [10▪].

The role of episiotomy and spontaneous perineal laceration are also debated with respect to pelvic organ support. Episiotomy was first recommended in the 1930s as a strategy to prevent perineal lacerations and postpartum pelvic organ support was found to be better among women who had undergone an episiotomy [28,29]. However, a 2005 JAMA systematic review observed that the impact of episiotomy on the development of prolapse remains unknown [30]. A potential association between spontaneous laceration and prolapse is suggested by a recent study of vaginally parous women 5–10 years after first delivery. Those who had experienced more than one spontaneous perineal laceration (across all her deliveries) were more likely to have prolapse to or beyond the hymen [11▪▪]. No increase in prolapse was seen in association with episiotomy. There is also recent evidence to suggest that a mediolateral episiotomy protects against development of central support defects of the anterior vaginal wall which is the most common site of prolapse [31▪▪]. These studies raise the question whether episiotomy may be preferable to spontaneous laceration with respect to prolapse risk and clearly this is an important area for further research.

ANAL INCONTINENCE AND VAGINAL CHILDBIRTH

Direct trauma and laceration of the anal sphincter complicates 2–16% of vaginal deliveries [32,33]. Injury to the anal sphincter, even with recognition and repair, contributes to the development of anal incontinence. Numerous studies have demonstrated a significant short-term risk of anal incontinence after obstetrical anal sphincter laceration [34,35]. More recently, this increased risk has been shown to persist for at least 10 years after delivery [36].

One question is the incidence of ‘occult’ anal sphincter lacerations. Initially, the incidence of ‘occult’ lacerations was felt to be quite high [37]. However, subsequent studies demonstrate that most ‘occult’ lacerations can be identified clinically with appropriate training and a diligent examination [38]. Thus, true ‘occult’ lacerations are probably uncommon. Recent research suggests that vaginal delivery, in the absence of a sphincter injury, does not appear to increase a woman’s odds of anal incontinence [34,36].

BIOLOGICAL MECHANISMS MEDIATING THE ASSOCIATION BETWEEN VAGINAL CHILDBIRTH AND PELVIC FLOOR DISORDERS

In summary, vaginal childbirth appears to be strongly associated with SUI and pelvic organ prolapse. There is less evidence to suggest an association between vaginal delivery and other pelvic floor disorders. In order to understand the clinical implication of the observed associations, the sections that follow review some of the purported mechanisms that explain how vaginal childbirth may lead to SUI and prolapse.

Disruption of structural elements of support

Pelvic organs are maintained in their normal anatomic position due to the support system comprised of the levator ani muscles, the endopelvic fascia and its lateral condensation (arcus tendinous fascia pelvis), the uterosacral ligaments and the perineal body. In the presence of intact support system, the vagina is pulled superiorly and posteriorly toward the sacrum, thus, facilitating compression of vaginal against the levator ani muscle during increased intra-abdominal pressure. Damage to this anatomical relationship, as a result of injury to levator ani muscles or other components of the pelvic support system, may lead to downward descent of the pelvic organs and stretching of the connective tissues, predisposing development of prolapse.

Using MRI and three-dimensional ultrasound, it is now possible to identify obstetrical injuries to the levator ani muscle complex. The levator muscle, particularly the pubococcygeal muscle, has been shown to undergo significant strain during the second stage of labor [39]. This can lead to overstretching and damage to muscles [40]. Recent studies suggest that vaginal birth, particularly operative vaginal delivery, is associated with levator injury. A number of small studies suggest that 20–36% of women experience a significant levator injury at the time of vaginal birth [41,42]. In comparison, levator injury is rarely, if ever, observed after cesarean birth [43–45]. In addition, compared with spontaneous vaginal birth, forceps assisted vaginal delivery has been associated with three times increased odds of levator trauma [43–45]. In summary, vaginal delivery, especially instrumented vaginal delivery, increases the probability of major levator injury.

The long-term consequences of levator injury are uncertain. Several recent studies have shown that pelvic muscle strength is poorer after vaginal versus cesarean birth [46,47]. Also, a recent study of parous women more than 5 years from delivery shows poorer muscle strength after vaginal delivery, with additional reduction after forceps delivery [48]. Reduced levator function has been shown in computer simulation models to contribute to the development of prolapse [49,50]. Indeed, women with prolapse are more likely to have imaging evidence of levator injury. Delancey *et al.* [51] found major levator defects in 55% of women seeking prolapse treatment and 16% of community controls [adjusted odd ratio (OR) 7.3, 95% confidence interval (CI) 3.9, 13.6]. Dietz and Simpson [52] imaged 781 women presenting to a tertiary clinic, noting levator injuries in 150 of 415 (36%) with prolapse and in 31 of 366 (8%) presenting for treatment of other pelvic floor disorders (OR 6.1, 95% CI 4.0 9.6). Both of these studies found increased odds of levator injury with prolapse. However, in both studies, cases (women with prolapse) were seeking treatment at tertiary centers and these women may not be typical of prolapse in general. In addition, it is plausible that levator injury is a marker for delivery trauma and may not be the actual mechanism underlying the association between pelvic floor disorders and childbirth. Thus, longitudinal studies are needed to clarify the role of levator injury and levator function in the later development of pelvic floor disorders.

Denervation injury to pelvic floor

More than 20 years ago, British researchers suggested that pudendal nerve stretching and compression during labor could result in denervation injury of the pelvic floor muscles [53]. This was hypothesized as another mechanism implicating childbirth in development of pelvic floor disorders. The pudendal nerve is vulnerable to stretching and compression injury from fetal head engagement during labor. Such injuries have been reported with 38–42% of vaginal deliveries [54]. Injury to the afferent pathways carrying sensory input to the cerebral cortex from the anal canal has been seen in animal models of second stage of labor [55▪].

However, this has been a difficult area of research. Measures of neuromuscular function are challenging and not widely available. Needle electromyography studies are painful and the interpretation is cumbersome (and of unknown reproducibility). Pudendal nerve conduction studies, hailed early as a measure of chronic denervation, have proved unreliable. The end result is that most studies of pelvic floor denervation after childbirth include small samples and the results of these studies conflict. Efforts to develop animal models of pudendal nerve injury have employed vaginal distension or direct pudendal nerve compression/crush. Both vaginal distension and pudendal injury have been shown to result in stress incontinence [56]. However, the resultant stress incontinence is transient unless the pudendal nerve is completely transected. Recovery after vaginal distension is delayed among animals subjected to both pudendal crush and vaginal distension [57].

It has been postulated that denervation injury to the levator ani and the striated sphincter muscles during birth can progressively worsen during straining activities later in life and can lead to secondary changes in levator ani and smooth muscle of the internal urinary sphincter. This could plausibly explain the latency commonly seen between the time of childbirth and development of pelvic floor symptoms decades later.

Soft tissue trauma and remodeling after childbirth

During pregnancy, the pelvic floor undergoes hormone-mediated physiological changes in its biomechanical properties in anticipation of delivery of the fetus. Animal models have shown that the vaginal wall and its supportive tissue complex is more distensible and less stiff during pregnancy. However, its not as strong and maximum tolerable stress is less than the prepregnancy level [58].

With delivery, there is substantial remodeling of the vaginal wall. Collagen is the main constituent of extracellular matrix of the vaginal wall. It has been shown that the fibroblasts in the vaginal wall are sensitive to mechanical stretch. Repetitive stretching of the vaginal wall initiates a degradative response. Stimulation of collagenases results in increased degradation of collagen, as well as other constituents of the vaginal wall. These processes may make the vaginal wall and supportive structures more susceptible to birth trauma [59]. Usually, the increase in collagenase activity is linearly correlated with the magnitude of the mechanical force applied [60]. Thus, difficult or prolonged labor may exceed the stretch limits of the soft tissue, causing imbalance in the repairative and degradative processes.

Elastin is a critical component of vaginal and pelvic floor connective tissue. Elastic fiber homeostasis in the vaginal wall has been investigated as a possible contributor to the development of pelvic floor disorders. Murine models of defective elastin homeostasis have suggested that elastic fiber synthesis is a critical process in recovery from childbirth [61]. Specifically, vaginal distension (such as would occur during descent of the fetal head in labor) results in degradation of elastin. Recovery from childbirth requires synthesis of elastin. Impaired elastin synthesis has been shown, in murine models, to lead to prolapse. It is not clear whether this process plays a role in the development of prolapse among humans.

For example, it is not clear whether women with genetic disorders of elastic fiber homeostasis (e.g. Marfan's syndrome) are at increased risk for prolapse and stress incontinence. In addition, it is not clear whether observed differences in the connective tissue of women with prolapse are due to prolapse or contribute to the development of prolapse [62,63▪▪,64]. This is a critical area for further research.

CONCLUSION

Pelvic floor disorders represent a significant health problem affecting women of all ages. Identification of potential modifiable risk factors and advancement in understanding of the underlying pathophysiology is crucial for primary and secondary prevention of these disorders and for improvement in treatment strategies.

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KEY POINTS

- Vaginal childbirth is associated with increased incidence of SUI and pelvic organ prolapse. The association of other pelvic floor disorders with vaginal birth is less well established; however, operative vaginal delivery is associated with overactive bladder symptoms.
- Structural compromise and/or denervation of the components of pelvic floor support system from compression, stretching or avulsion during labor are possible mechanisms associating vaginal birth with development of pelvic floor disorders.
- Operative vaginal delivery significantly increases the odds of pelvic floor disorders; however, clinical decisions must be individualized based on the risk and benefits of this intervention compared with its alternatives.