

# Effect of pregnancy and childbirth on urinary incontinence and pelvic organ prolapse

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## INTRODUCTION

Pelvic floor disorders (PFDs) include pelvic organ prolapse (POP), urinary incontinence, and fecal incontinence. The burden of disease related to PFDs is substantial. These disorders affect one-third of adult women in the United States, with considerable impact on their quality of life [1-4]. As many as 20 percent of women undergo surgical treatment for PFDs and approximately 17 percent of these women require reoperation [5-7]. The scope of this clinical issue is even greater than the surgical treatment rate suggests, because many women are managed conservatively with pessaries, pads, or no therapy.

An area of intense investigation is the effect of pregnancy and childbirth on a woman's risk of developing PFDs and whether this risk can be reduced by modifications to obstetric care. Some women have requested cesarean delivery for this reason.

This review will focus on urinary incontinence and POP. The data are more comprehensive for incontinence, because prolapse is difficult to study [8]. Many women with prolapse are asymptomatic and measurement of the true prolapse rate requires pelvic examination. Measurement of symptomatic prolapse is more clinically relevant, since asymptomatic prolapse does not require treatment. Even this presents a research challenge because prolapse symptoms may take longer to develop than symptoms of incontinence and the symptoms (eg, vaginal bulge or pressure, urinary retention, difficult defecation) are less specific. Thus, even symptomatic prolapse is not well measured by symptom questionnaires [9]. Further, use of surgery as a surrogate measure for the prevalence of prolapse does not

detect the number of women with symptomatic prolapse who have not had surgical treatment [10-12].

The association of urinary incontinence and POP with pregnancy and childbirth and selected management issues are reviewed here. The role of pregnancy and childbirth in the development of fecal incontinence is discussed separately. (See "[Fecal and anal incontinence associated with pregnancy and childbirth: Counseling, evaluation, and management](#)".)

General principles of the diagnosis and treatment of urinary incontinence and POP are also discussed separately.

- (See "[Female urinary incontinence: Evaluation](#)".)
- (See "[Female urinary incontinence: Treatment](#)".)
- (See "[Pelvic organ prolapse in females: Epidemiology, risk factors, clinical manifestations, and management](#)".)
- (See "[Pelvic organ prolapse in women: Diagnostic evaluation](#)".)

In this topic, when discussing study results, we will use the terms "woman/en" or "patient(s)" as they are used in the studies presented. However, we encourage the reader to consider the specific counseling and treatment needs of transgender and gender diverse individuals.

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## MECHANISMS OF PELVIC FLOOR INJURY

**Clinical anatomy of the pelvic floor** — The pelvic floor is primarily made up of the levator ani and coccygeus muscles (ie, paired puborectalis, pubococcygeus, and iliococcygeus). The urethral and anal sphincter muscles are also part of the pelvic floor.

The endopelvic connective tissues lie superior to the pelvic floor muscles and connect to the pelvic side walls and sacrum. The perineal membrane (ie, bulbocavernosus, transverse perineal, and ischiocavernosus muscles) lies external and inferior to the pelvic floor.

The innervation of this region is from the S2, S3, and S4 segments of the spinal cord, which fuse to form the pudendal nerve. The pudendal nerve innervates the external anal sphincter, whereas the levators, coccygeus muscles, and urogenital diaphragm appear to be innervated by a direct connection of S2, S3, and S4 nerve fibers [13].

Female pelvic floor anatomy is discussed in detail separately. (See "[Surgical female urogenital anatomy](#)".)

**Effect of pregnancy and childbirth** — The biologic mechanisms of injury to the pelvic floor during pregnancy and childbirth have not been fully determined. Data suggest that

pregnancy and delivery contribute to pelvic floor injury due to compression, stretching, or tearing of nerve, muscle, and connective tissue ( [figure 1](#)). Intact neuromuscular function and pelvic organ support are both critical to normal function of pelvic viscera.

**Neural injury** — During labor and vaginal delivery, descent of the fetal head may cause stretching and compression of the pelvic floor and the associated nerves. This process can lead to demyelination and subsequent denervation [14]. This mechanism of injury is supported by neurophysiologic tests, including concentric needle electromyography (EMG) and pudendal nerve motor latency, which have demonstrated denervation of the pubovisceral muscles and anal sphincter after 40 to 80 percent of vaginal births [14,15]. Risk factors for denervation and pudendal nerve damage include operative delivery, prolonged second stage, and high birth weight [16].

In vivo research on pudendal neuropathy after childbirth is challenging because electrophysiologic testing is technically complex, invasive, and difficult to interpret. Thus, most studies of pelvic floor denervation after childbirth are limited to small samples; conflicting results have been obtained. Some insights have been derived from animal models of pudendal nerve injury. In some cases, vaginal balloon distension has been used to simulate pudendal nerve compression by the fetal head [17]. Other models include crush injuries or transection of the pudendal nerve [18,19]. Pelvic pressure by the fetal head during the second stage of labor may lead to combined neurovascular injury [20]. In animal models, direct and indirect pudendal injury has been shown to result in stress incontinence [21]. The resultant stress incontinence persists if the pudendal nerve is completely transected, but resolves after distension injury. Recovery after vaginal distension is delayed among animals subjected to both pudendal crush and vaginal distension [18]. Thus, it is not clear whether these animal models are relevant to childbirth injuries in humans.

Cohort studies suggest that neuromuscular injury resolves during the first year after delivery for the majority of women [15]. This may account for the spontaneous resolution of incontinence over that same period. However, in some cases, electrophysiologic evidence of denervation injury can be seen five to six years after delivery [16,22], and denervation injury may accumulate with increasing parity [14,23,24]. It is not known why some women recover neuromuscular function after childbirth and others demonstrate evidence of permanent damage.

**Injury to the levator ani and coccygeus muscles** — The levator ani muscle complex is critical to pelvic floor function. This muscle complex forms a U-shaped sling around the pelvic viscera. The resting tone of the levator ani muscle keeps the urogenital hiatus closed, resisting the downward displacement of the vagina during increased intraabdominal pressure. Voluntary levator ani contraction can further augment vaginal closure force, leading to compression of rectum, distal vagina, and urethra [25]. Loss of levator function

(eg, due to traumatic disruption, denervation, or atrophy) may lead to widening of the urogenital hiatus and result in pelvic organ descent.

Magnetic resonance imaging (MRI) and ultrasound studies have provided important observations regarding the levator ani muscle after childbirth [26,27]. Evidence suggests that the levator may be avulsed from the pubic bone with vaginal delivery. Specifically, distension of the pubococcygeus by the fetal head may result in detachment of the levator muscle from the pubis [28]. Levator avulsion has been observed among 20 percent of women who have delivered vaginally [29], but only rarely after cesarean [30]. Imaging studies suggest that forceps delivery further increases risk [31]. For example, one study reported that the odds of levator avulsion was almost fivefold higher after forceps-assisted delivery compared with vacuum-assisted birth [32]. Prolonged second stage of labor may also be associated with occult injury to the levator ani [26,29,31,33]. (See "[Assisted vaginal birth](#)".)

Computer simulation models have supported the hypothesis that levator injury leads to prolapse [34]. Finite element models have been used to model deformations of the pelvic floor with vaginal delivery. Using this theoretical approach, a model predicted that the greatest strain would occur at the bony attachments of the levator ani and pubococcygeus muscles, presumably with extension of the fetal head [35]. The degree of deformation predicted would exceed thresholds for injury, especially in the most medial aspects of the levator ani complex. Similar results were obtained with a three-dimensional (3D) computer model of birth [28]. Correlation with anatomic studies of parous women is needed to assess the validity of these simulation models.

The impact of levator ani avulsion on pelvic floor strength, POP, and stress urinary incontinence (SUI) are areas of investigation. Representative studies include:

- **Pelvic floor strength by delivery type** – Pelvic muscle strength may be modified by childbirth. Among 666 patients assessed via perineometry 6 to 11 years from first delivery, those with a history of at least one vaginal delivery demonstrated lower peak muscle strength and duration of contraction compared to those who had delivered only by cesarean [36]. The lowest strengths were in patients who had a forceps delivery.
- **Levator avulsion and pelvic floor strength** – Women with levator avulsion injuries have weaker pelvic floor muscles [26,37] and a wider levator hiatus compared with those without such injury [37]. These characteristics may contribute to the development of prolapse: In a study of vaginally parous women who were an average of 11 years from first delivery, both weaker muscles and a wider hiatus were strongly and independently associated with POP [38].
- **Levator avulsion and POP risk** – Among patients with POP, levator ani avulsion is two to three times more likely than normal pelvic floor anatomy [26,27]. This suggests that

occult levator avulsion may be a mechanism for the development of prolapse in at least a subset of cases.

- **Impact on urinary incontinence** – It is unclear whether levator avulsion is more prevalent in patients with urinary incontinence compared with other parous people [29,39,40]. In a longitudinal study that evaluated over 450 vaginally parous individuals with 3D ultrasound, levator avulsion was associated with prolapse beyond the hymen (odds ratio 2.7) but not with SUI [41]. Thus, levator avulsion may not be a risk factor for SUI.
- **Episiotomy and pelvic floor strength** – Mediolateral episiotomy, which typically involves an intentional incision of the levator ani and coccygeus muscles, has been assumed to impact pelvic muscle function. However, a Swedish study reported that mediolateral episiotomy was not associated with pelvic muscle strength six weeks after delivery [42].

It is important to note that there are no proven treatments for women who sustain levator avulsion with delivery. As an example, a small study of postpartum women found that those with and without levator avulsion (demonstrated on ultrasound) have a similar response to postpartum pelvic muscle exercises [43]. Therefore, it is not the standard of care to assess for levator avulsion in the postpartum period. Until effective secondary prevention measures are identified, assessment for obstetric levator avulsion does not have clinical value.

**Fascial injury** — Injury to other soft tissue structures may provide another mechanism by which childbirth may lead to PFDs, especially POP. One study described paravaginal defects as separation of the endopelvic fascia from its lateral attachment to pelvic side wall [44]. Paravaginal defects are associated with urethral hypermobility, SUI, and poor anterior vaginal support [45].

**Impaired connective tissue remodeling** — Collagen and elastin are the two primary components of extracellular matrix. Elastin contributes to distensibility, while collagen is associated with tensile strength. During pregnancy, increased synthesis of collagen and elastin contributes to changes in soft tissue biomechanics. Most notably, the vagina becomes more distensible [46]. Distension or stretching of the vagina promotes the release of collagenases from fibroblasts, leading to increased collagen degradation.

After delivery, there is an increase in synthesis of collagen and elastin, leading to substantial remodeling of pelvic floor and vaginal connective tissue. Defects in these biochemical processes have been postulated as a possible mechanism for development of PFDs. Genetic defects in connective tissue metabolism have been hypothesized as a mechanism for susceptibility to prolapse. Impaired elastin synthesis in animal models contributes to prolapse [47]. Alterations in collagen composition have been demonstrated in women with

prolapse [48]. Also, vaginal tissue from women with SUI has demonstrated differential expression of genes related to elastin metabolism [49]. However, in women with established prolapse, it is impossible to know whether the observed connective tissue properties are the cause of prolapse or a response to the vaginal wall distension associated with prolapse.

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## PREVALENCE IN PAROUS WOMEN

A causal role of pregnancy and childbirth is supported by the finding that pelvic floor disorders (PFDs) are more prevalent among women who have delivered at least one child [50-64]. Furthermore, the rate of PFDs increases with increasing parity. Among parous women, it has been estimated that 50 percent of incontinence and 75 percent of prolapse can be attributed to pregnancy and childbirth [65].

The effect of parity is prominent mainly in premenopausal women. Age is also a risk factor for PFDs and, in postmenopausal women, the impact of age appears to override the impact of parity [66,67].

In nulligravid women less than age 55 years, the prevalence of urinary incontinence is 6.5 to 18 percent and of prolapse is approximately 0.6 [53,68,69]. Representative studies that compared nulliparous with parous women and illustrate the effects of parity and age include:

- A national survey of nonpregnant women in the United States (n = 1961) suggested the following symptom prevalences: urinary incontinence (15.7 percent), fecal incontinence (9 percent), and POP (2.9 percent) [68]. The impact of parity on prevalent PFDs was notable:
  - Urinary incontinence: nulliparous (6.5 percent); one birth (9.7 percent); two births (16.3 percent); and three or more births (23.9 percent).
  - POP: nulliparous (0.6 percent); one birth (2.5 percent); two births (3.7 percent); and three or more births (3.8 percent).
  - Urinary incontinence: 20 to 39 years (6.9 percent) versus 60 to 69 (23.3 percent).
  - POP: 20 to 39 years (1.6 percent) versus 60 to 69 (4.1 percent).
- A study of 27,900 Norwegian women found that, among premenopausal women, parous women were significantly more likely than nulliparous women to report urinary incontinence (22 to 34 versus 14 percent) [53]. The impact was greatest with the first delivery. In younger women, odds for incontinence increased with increasing parity (20 to 34 years: 2.2-fold for one delivery versus 3.3-fold for four or more deliveries). In older

women, parity had little impact (65 to 74 years: ranging from 0.9-fold for one to 1.1-fold for four or more deliveries).

- Among 271 pairs of identical twins (mean age of 47 years, range 15 to 85 years), the prevalence of urinary incontinence was higher in the parous twin (48 to 67 percent versus 24 percent) and increased with increasing parity (twofold for one birth, fourfold for two births) [55].
- The Oxford Family Planning study, a prospective cohort study of more than 17,000 women followed for 17 years, found that, compared with nulliparity, the risk of hospital admission for POP surgery increased markedly for the first (fourfold) and second (eightfold) birth, and then increased less rapidly for subsequent births (third: ninefold; fourth: 10-fold) [11].

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## ROLE OF OBSTETRIC FACTORS

Parity is comprised of several components (pregnancy, labor, delivery), each of which may contribute to the development of pelvic floor disorders (PFDs). The sections that follow will review the evidence for the role of each component.

**Pregnancy** — Urinary incontinence is more common during pregnancy than before pregnancy. Many women experience their first symptoms of incontinence during pregnancy. Urinary incontinence symptoms resolve in many women after delivery or the postpartum period, but many will have persistent symptoms.

During pregnancy, urinary incontinence is reported by 7 to 60 percent of women [67,70-74]. As an example, among over 5000 primiparous women who were continent before pregnancy, 46 percent leaked urine either during or after pregnancy, 7 percent leaked only during pregnancy, and 10 percent leaked only after delivery [73].

The prevalence and severity of incontinence increase during the course of pregnancy [75-77]. The highest incidence of incontinence is noted in the second trimester [74] and, based upon the cumulative rate of new incontinence symptoms, prevalence peaks in the third trimester [74-77].

The prognosis for women who develop urinary incontinence during pregnancy is generally favorable. For most women with incontinence during pregnancy, symptoms are likely to resolve after delivery. Seventy percent of women with onset of urinary incontinence during pregnancy experience spontaneous resolution of symptoms postpartum [70,75,78,79]. Specifically, within 12 months postpartum, the prevalence drops to 11 to 23 percent [70,75,78]. Also, among women with persistent incontinence, severity declines in the first

year after childbirth, with a substantial reduction in both the frequency of incontinent episodes and other measures of severity [56,80].

Nevertheless, women with incontinence during pregnancy appear to be at increased risk for postpartum urinary incontinence symptoms, compared with women who do not experience incontinence during pregnancy [74,75,81,82].

There are fewer data about POP among pregnant women; however, there is general agreement that Pelvic Organ Prolapse Quantification (POP-Q) stage increases during pregnancy [83-85]. It is not known whether prolapse that arises during or shortly after pregnancy is likely to resolve with time. Further study is needed to investigate the effects of pregnancy and type of delivery on pelvic support disorders. Common conditions unrelated to pregnancy and delivery, such as obesity, as well as aging, also affect the risk of developing prolapse. (See "[Pelvic organ prolapse in females: Epidemiology, risk factors, clinical manifestations, and management](#)" and "[Pelvic organ prolapse in women: Diagnostic evaluation](#)".)

**Labor** — Most of the available evidence suggests that labor, in the absence of vaginal delivery, has a negligible effect on the development of PFDs later in life. However, some studies have yielded inconsistent results [1,9,73,84,86-89].

Representative studies include:

- A population-based study of over 15,000 primiparous women surveyed within one year postpartum found no significant difference in the incidence of urinary incontinence in those who had elective cesarean delivery compared with cesarean after labor or cesarean after labor and pushing (6.1 versus 5.7 versus 6.4 percent) [73].
- A survey of 4458 women found no difference between women who did or did not labor prior to cesarean in terms of risk for stress urinary incontinence (SUI), overactive bladder, or anal incontinence symptoms. However, prolapse symptoms were more common among those who labored prior to cesarean (1 of 92 versus 13 of 198) [1].
- A study of 1011 women who were examined for objective evidence of prolapse reported no difference in prolapse between women who had undergone cesarean before or after labor [9].

## **Mode of delivery**

**Vaginal versus cesarean delivery** — Vaginal birth is associated with an increased risk of PFD development compared with cesarean delivery or nulliparity [63,64,90,91]. However, cesarean delivery does not completely prevent PFDs [1,70,92-96].

Representative studies include:

- **Increased risk of symptoms**

- **Pelvic organ prolapse** – A Swedish national cohort study including over 14,000 patients reported an increased risk of POP symptoms ("sensation of tissue protrusion, a vaginal bulge") in primiparous individuals with vaginal delivery compared with either nulliparous individuals or primiparous patients with cesarean delivery [97]. In addition, the risk accelerated over time. For patients with a single pregnancy and vaginal birth, the estimated probability of symptomatic POP increased fourfold, from approximately 4 to 13 percent, between the ages of 40 and 64 years. The prevalence of symptomatic POP remained relatively stable, at less than 5 percent, for patients in the nulliparous and cesarean delivery groups across the same age span.
- **Urinary incontinence** – A meta-analysis of 15 studies (both observational and trial data) reported an increased risk of SUI following vaginal birth compared with cesarean delivery (adjusted odds ratio [OR] 1.85, 95% CI 1.56-2.19, risk difference 8.2 percent) [91].

- **Both incontinence and POP**

- A Swedish national registry study including over 90,000 patients reported that those who had only vaginal births, compared with only cesarean deliveries, had significantly higher rates of urinary incontinence (1.2 versus 0.4 percent, hazard ratio [HR] 2.9, 95% CI 2.4-3.6) and prolapse surgery (2.2 versus 0.2 percent, HR 9.2, 95% CI 7.0-12.1) [98].
- A survey of patients who had one singleton delivery 20 years previously (n = 5236; mean age at delivery 29 to 32 years) reported that vaginal birth, compared with cesarean delivery, was associated with an increased risk of urinary incontinence (40.3 versus 28.8 percent, OR 1.7, 95% CI 1.5-1.9) and symptomatic POP (14.6 versus 6.3 percent, OR 2.6, 95% CI 2.0-3.3) [99,100].

- **Increased risk of pelvic surgery** – A Swedish national registry study reported that, compared with nulliparous patients  $\geq 45$  years old from the general population, patients experiencing vaginal birth were over-represented in both prolapse surgery (relative risk 1.23, 95% CI 1.22-1.24) and urinary incontinence surgery (relative risk 1.17, 95% CI 1.15-1.19) groups [64]. Further, the absolute risk of prolapse surgery increased with an increasing number of vaginal births (absolute risk 2.3 per 1000 nulliparous individuals increased to 38.7 per 1000 individuals with  $\geq 4$  vaginal births) while increasing numbers of cesarean deliveries was not associated with increased risk of prolapse surgery.

- **Worsened pelvic support** – A prospective cohort study that followed over 1200 parous patients for up to 9 years reported that, compared with patients who underwent

cesarean delivery, individuals who underwent vaginal birth had worse vaginal support five years following delivery as measured by the POP-Q system [101]. During longitudinal follow-up, patients with at least one vaginal birth had more rapid worsening of their apical vaginal support than those who had no vaginal deliveries. In addition, longitudinal analysis reported that, compared with spontaneous vaginal birth, cesarean delivery was associated with significant reduction of the risk of SUI (adjusted HR [aHR] 0.46, 95% CI 0.32-0.67), overactive bladder (aHR 0.51, 95% CI 0.34-0.76), and POP (aHR 0.28, 95% CI 0.19-0.42) [96].

**Operative vaginal delivery** — Operative vaginal delivery with forceps appears to increase the risk of developing POP and SUI [96,102,103]. Instrumented delivery also increases the risk of anal sphincter laceration, which increases the risk of fecal incontinence. (See "[Fecal and anal incontinence associated with pregnancy and childbirth: Counseling, evaluation, and management](#)", section on 'Role of obstetric factors' and "[Obstetric anal sphincter injury \(OASIS\)](#)", section on 'Epidemiology and risk factors'.)

In the early 1900s, it was hypothesized that outlet forceps delivery protected the pelvic floor by controlling delivery of the vertex, thereby allowing less force to be applied to the pelvic musculature [104]. There is no good evidence to support this hypothesis. Instead, forceps delivery appears to be associated with injury to the levator ani muscles [105] and has been associated with pudendal neuropathy [16]. (See '[Mechanisms of pelvic floor injury](#)' above.)

Forceps delivery appears to increase the risk of development of POP and incontinence. In a study of nearly 450 women, women with at least one forceps delivery had double the relative odds of POP symptoms compared with women who did not have a forceps delivery [106]. In addition, the relative odds of having overactive bladder symptoms were increased almost threefold for the women who had at least one forceps delivery. A different study that assessed delivery route and subsequent prolapse surgery for women aged 15 to 44 years reported that, as the forceps rate declined during the period of 1989 to 2009, the rates of obstetric trauma and subsequent surgery for POP also declined [95]. Lastly, in a prospective cohort study that followed over 1500 women for a median of 5.1 years, operative vaginal delivery (either forceps or vacuum assisted) was associated with increased risk of both POP and anal incontinence compared with women having only vaginal births [96].

Vacuum-assisted vaginal delivery does not appear to have the same impact on risk of prolapse or urinary incontinence [102,103]. In the above study of nearly 450 women, vacuum delivery was not associated with urinary or fecal incontinence or POP [106]. Similarly, a survey study of over 13,000 women in Norway reported, for women younger than age 50, an increased risk of stress incontinence following forceps delivery, but not vacuum delivery, compared with spontaneous vaginal delivery [103]. Additionally, a survey study of over 5000

women reported no increase in the risk of prolapse 20 years after a single vacuum delivery [99].

**Episiotomy** — Episiotomy was first recommended almost 100 years ago as a strategy to prevent perineal lacerations and to protect the pelvic floor [107]. Observational studies in the 1930s suggested that postpartum "relaxation" was less common if episiotomy was performed [108,109].

However, the obstetrical community began to critically reappraise the relative harms and benefits of episiotomy in the last decades [110]. At this time, the impact on PFD of liberal episiotomy use is uncertain in part because of varied endpoints and study duration. Representative examples include:

- One Swedish study reported that mediolateral episiotomy was not associated with urinary incontinence six weeks after delivery [42].
- A systematic review of 26 prospective studies found no evidence that episiotomy prevents PFDs [111].
- A Cochrane review found no association between liberal versus restrictive use of episiotomy and incontinence or prolapse [112].

Episiotomy rates have been declining, given that evidence does not support routine use of episiotomy. (See "[Approach to episiotomy](#)", section on 'Lack of advantages for episiotomy use'.)

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## OTHER FACTORS

**Maternal age** — Increasing maternal age is associated with an increased risk of pelvic floor disorders (PFDs) [99,100]. As an example, in a retrospective cohort study with data from a national registry including over 90,000 women, increasing maternal age was associated with an increased risk of PFDs [10]. Among women who had a vaginal delivery, those who were 30 years or older at delivery had higher rates of surgery for stress urinary incontinence (4.9 versus 3.2 per 10,000 person-years) and POP (9.7 versus 3.4 per 10,000 person-years) compared with women less than 30 years of age.

**Birth weight** — Increasing infant birth weight appears to be associated with an increased risk of POP, but the relationship to urinary incontinence is unclear. As an example, a survey of women who had one singleton delivery 20 years previously (n = 5236) found that symptomatic POP increased 3 percent (odds ratio 1.03, 95% CI 1.02-1.05) for each 100 g increase of infant birth weight [99,100]. In contrast, no association was found between birth weight and urinary incontinence. In addition, a retrospective study of 1521 women found

that birth weight >4000 g was associated with a 50 percent increase in the risk of incontinence later in life [113].

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## SYMPTOM MANAGEMENT DURING PREGNANCY

Symptoms of urinary incontinence or POP may develop or be exacerbated during pregnancy. Conservative management with pelvic floor muscle training or pessary is preferred. (See 'Prophylactic pelvic floor muscle exercises' below.)

Management of urinary incontinence and POP are discussed in detail separately. (See "Pelvic organ prolapse in females: Epidemiology, risk factors, clinical manifestations, and management", section on 'Conservative management' and "Vaginal pessaries: Indications, devices, and approach to selection" and "Female urinary incontinence: Treatment", section on 'Pelvic floor muscle (Kegel) exercises'.)

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## APPROACH TO OBSTETRIC MANAGEMENT

Obstetric care interventions to reduce the long-term risk of pelvic floor disorders (PFDs), especially those that are modifiable, is an important area of investigation, given the association between pregnancy and childbirth and PFDs.

Various obstetric interventions have been proposed, but their impact is not certain. Clinical trials are challenging in this area of obstetrics and, therefore, most of the data are derived from observational studies. In addition, it is difficult to individually assess a particular intervention because obstetric characteristics may cluster (eg, episiotomy is associated with forceps delivery), making it difficult to assess the impact of any individual modification. Also, it is likely that our existing practices regarding clinical documentation of labor and delivery do not adequately describe obstetric "exposures" across a population (eg, how low did the presenting fetal part descend?, what was the position of the mother and fetus during the second stage?, did her symptoms progress or regress?, and how long was she followed postpartum?).

**Prophylactic pelvic floor muscle exercises** — Pelvic floor muscle exercises (PFME) performed during pregnancy help to decrease the short-term risk of urinary incontinence in women without prior incontinence, but a long-term benefit has not been established. This was illustrated in a meta-analysis that included six randomized trials that found that continent women (most trials included only nulliparous women) who were assigned to perform antenatal PFME compared with those who were not had a significantly decreased risk of urinary incontinence at three to six months postpartum (18 versus 25 percent, risk ratio 0.71, 95% CI 0.54-0.95) [114]. Among the few studies that evaluated women six or more

months postpartum, no significant effect was seen and the trend was toward a lower rate in urinary incontinence in the women who were not assigned to PFME.

In the same meta-analysis, data from seven randomized trials that included a mixed population of women (continent and incontinent; nulliparous and parous) showed that women assigned to antenatal PFME had a significant decrease in the rate of urinary incontinence at up to three months postpartum (risk ratio 0.77, 95% CI 0.59-1.01) [114]. This continued as a trend through 12 months postpartum, but did not reach statistical significance. It is important to note that study of a mixed population of continent and incontinent women results in study of use of PFME as both prophylaxis and treatment, so it is difficult to interpret the results.

In terms of postpartum PFME, there is increasing evidence that some women sustain injury to the levator ani muscle complex at childbirth [115,116]. If this is true, PFME in the immediate postpartum period may be harmful to some women because an exercise program may be contraindicated in the early phase of injury recovery. This is based on evidence from sports medicine that the preferred early treatment of muscle injury is rest or immobilization, with strength training only after initial healing is complete; however, there are no data regarding this issue following childbirth. (See "[Running injuries of the lower extremities: Risk factors and prevention](#)".)

A randomized trial found no decrease in urinary incontinence in postpartum women who performed supervised PFME for 16 weeks [43]. There was no significant effect in women with or without major levator ani defects.

**Cesarean delivery** — While cesarean delivery has been associated with a lower risk of PFDs compared with vaginal delivery, cesarean delivery does not eliminate the risk of a future PFD. As an example, a prospective cohort study of primiparous women (n = 124) who underwent cesarean delivery before labor found that 22.9 percent reported urinary incontinence at six months [117]. Less than 1 percent recalled urinary incontinence prior to pregnancy. (See '[Pregnancy](#)' above.)

The prophylactic benefit of cesarean delivery for the prevention of PFDs has not been established, and most studies are based on indirect comparisons [118]. Some insights can be gleaned from a randomized trial of women (n = 1159) with term breech fetuses who were assigned to planned cesarean or planned vaginal birth [119]. At two-year follow-up, an intent-to-treat analysis found that the rate of urinary incontinence did not differ significantly in the cesarean and vaginal delivery groups (17.8 versus 21.8 percent, relative risk 0.81, 95% CI 0.61-1.1); POP was not evaluated. However, many participants were multiparous, and had already delivered vaginally. Also, almost 50 percent of women assigned to the planned vaginal delivery group delivered by cesarean. Both of these factors limit the ability of the study to predict whether cesarean delivery only can prevent PFDs.

Some studies have calculated that 7 to 12 women would have to deliver only by cesarean to prevent one woman from having a PFD later in life, assuming that the observed associations are causal [1,99,100]. A 2006 United States National Institutes of Health (NIH) expert panel concluded that there is only weak evidence to support a preventive role for elective cesarean delivery, and that the existing data do not adequately answer the question of whether elective cesarean delivery can reduce the incidence of PFDs. Even if a reduction in PFDs could be demonstrated, other harms and benefits of elective cesarean delivery need to be weighed against this benefit [120]. (See "[Cesarean birth on patient request](#)", section on '[Potential disadvantages and risks of planned cesarean birth](#)'.)

Thus, pregnant women who request cesarean delivery should be advised that there is insufficient evidence that elective cesarean delivery, or cesarean delivery on maternal request, prevents the development or exacerbation of PFDs [118]. Antepartum maternal characteristics are poor predictors of PFDs. At this point, there are no maternal characteristics that can be used to reliably guide specific women to the option of planned cesarean delivery.

- (See '[Vaginal versus cesarean delivery](#)' above.)
- (See "[Cesarean birth on patient request](#)".)

**Vaginal delivery management** — For women who plan vaginal birth, options to minimize the risk of PFDs include:

- **Prolonged labor** – Avoidance of protracted active labor or second stage of labor have not been proven to decrease the risk of PFDs. More study of this issue is needed. (See '[Labor](#)' above.)
- **Interventions in the second stage of labor** – To date, research has not identified opportunities to reduce PFDs by modifying the management of the second stage of labor. For example, no difference in PFD later in life has been shown for coached versus uncoached maternal pushing [121,122]. The impact of other second stage management strategies (including perineal massage during labor and maternal position) have not been critically evaluated.
- **Selective use of operative vaginal delivery** – Based upon the studies cited above, avoidance of forceps delivery appears to be a promising intervention to reduce the risk of damage to the pelvic floor [9,106]. (See '[Operative vaginal delivery](#)' above.)
- **Selective use of episiotomy** – As previously noted, routine versus restrictive episiotomy also does not appear to influence PFD later in life [112,123]. (See '[Episiotomy](#)' above.)

For operative vaginal delivery or episiotomy, studies could have been confounded by other factors that influence the development of PFDs, including cephalopelvic disproportion, maternal race, and the use of other obstetric interventions. Thus, there is insufficient evidence to establish whether an episiotomy or operative delivery is the cause of postpartum incontinence or simply a marker for a more difficult birth. The risks of performing these procedures should be weighed against the potential benefits in specific clinical situations.

The question has been raised regarding whether vacuum delivery should be used rather than forceps, as a measure to decrease the risk of PFDs. A variety of endpoints, including neonatal morbidity, should be considered in the decision to perform either vacuum or forceps delivery. If both are felt to be safe and viable options, the existing evidence suggests that PFDs are more strongly associated with forceps delivery. Vacuum delivery may be less traumatic to the pelvic floor.

**Limiting parity** — Obstetric providers may be asked by parous women about the impact of additional deliveries on the risk of PFDs. The evidence suggests that the biggest increase in the prevalence of PFDs is associated with the first birth [51,54,55]. As an example, among women over 50 years of age, the odds of uterine prolapse double after a first birth and then increase by only 10 percent with each additional birth [51]. There may be a measurable increase in incontinence with additional births after the first [51,54,55,124,125], although some studies show no increase with additional births [126,127].

Women considering additional pregnancies may be counseled that there may be an increasing risk of PFDs. Nevertheless, considerations other than the risk of PFDs will influence decisions regarding family size for the majority of women.

**Other strategies** — As providers of women's health care, obstetric care providers should also look beyond obstetric factors to identify other prevention opportunities. Parity and childbirth are important factors in the development of incontinence and prolapse, but not the only factors. Nulliparous women can experience PFDs [53,128] and, even among parous women, obstetric history is estimated to account for only 50 percent of incontinence [65].

Other strong risk factors for PFDs include age and race, but these factors are not modifiable. To minimize the incidence and impact of PFDs, women's health care providers should focus on modifiable risk factors, such as obesity [11,51,129,130]. Obesity has consistently been identified as a risk factor for prevalence and/or severity of PFDs and represents an important prevention opportunity.

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## OBSTETRIC CARE OF SPECIAL POPULATIONS

**Patients with urinary incontinence before or during pregnancy** — Women with a history or urinary incontinence prior to pregnancy or who have new or recurrent incontinence symptoms during pregnancy are a management challenge. Some data suggest that incontinence during pregnancy is a risk factor for persistent incontinence, as discussed above [75,81,82,131]. (See '[Pregnancy](#)' above.)

There is no evidence that elective cesarean delivery prevents persistent urinary incontinence in women with incontinence symptoms before or during pregnancy. In a study of over 1200 primigravid women, including 192 with urinary incontinence during pregnancy and 1040 without incontinence during pregnancy; the reduction in postpartum urinary incontinence associated with cesarean delivery was greater in the women who were continent during pregnancy (4.7 versus 20.8 percent). The impact of cesarean on postpartum incontinence was not significant among women who experienced incontinence during pregnancy (odds ratio 2.0, 95% CI 0.8-4.9) [81]. However, the study lacked sufficient statistical power and further study is needed.

**Patients who have undergone surgical repair** — Typically, surgical treatment of incontinence or prolapse are deferred until childbirth is complete. A review by the American Urogynecologic Society (AUGS) Guidelines and Statements Committee concluded that there are insufficient data to adequately counsel patients about either (1) the effect of future pregnancies on recurrence of PFDs after prior surgical treatment or (2) the impact of surgery for pelvic floor disorders on future pregnancies [132]. With respect to the former question, it is uncertain whether cesarean delivery is indicated for women who have had prior surgical treatment of prolapse or urinary incontinence. The question of prolapse recurrence after surgical treatment is becoming more relevant as uterine-sparing procedures, such as hysteropexy, gain popularity. With respect to the latter question, isolated cases of urinary retention have been reported among women who underwent surgery for stress incontinence, attributed to tension on mesh implants with anatomic changes of advanced pregnancy [133,134]. At this point, women considering surgery for prolapse or incontinence should be advised that the impact on future pregnancy remains uncertain. (See "[Female stress urinary incontinence: Choosing a primary surgical procedure](#)", section on '[Women finished with childbearing](#)' and "[Pelvic organ prolapse in women: Choosing a primary surgical procedure](#)", section on '[Patients finished with childbearing](#)'.)

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## **SOCIETY GUIDELINE LINKS**

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "[Society guideline links: Pelvic organ prolapse](#)".)

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## SUMMARY AND RECOMMENDATIONS

- **Impact of pregnancy and birth on pelvic floor disorders** – Pregnancy and childbirth appear to be associated with an increased risk of developing pelvic floor disorders (PFDs). Observational studies preclude definitive conclusions regarding the effect of pregnancy and childbirth on the later incidence of these disorders and whether any changes in obstetric management can reduce the risk. However, most of the accumulated evidence suggests that PFDs, especially stress urinary incontinence and pelvic organ prolapse (POP), are significantly more common after vaginal versus cesarean birth. Also, most evidence suggests that forceps delivery further increases the incidence of PFDs later in life. (See ['Prevalence in parous women'](#) above and ['Pregnancy'](#) above.)
- **Mechanisms of pelvic floor injury** – Pregnancy and delivery likely contribute to pelvic floor injury due to compression, stretching, or tearing of nerve, muscle, and connective tissue. (See ['Mechanisms of pelvic floor injury'](#) above.)
- **Pelvic floor muscle exercises** – Pelvic floor muscle exercises performed during pregnancy help to decrease the short-term risk of urinary incontinence in women without prior incontinence, but a long-term benefit has not been established. (See ['Prophylactic pelvic floor muscle exercises'](#) above.)
- **Cesarean delivery** – Pregnant individuals who request cesarean delivery should be advised that there is insufficient evidence that cesarean delivery prevents the development or exacerbation of PFDs. Clinicians should explore patient concerns about vaginal delivery and educate patients about the risks associated with repeat cesarean delivery. (See ['Vaginal versus cesarean delivery'](#) above and ["Cesarean birth on patient request"](#).)
- **Midline episiotomy** – Midline episiotomy increases the risk of anal sphincter tears, which are associated with anal incontinence. (See ["Fecal and anal incontinence associated with pregnancy and childbirth: Counseling, evaluation, and management"](#) and ['Vaginal delivery management'](#) above.)

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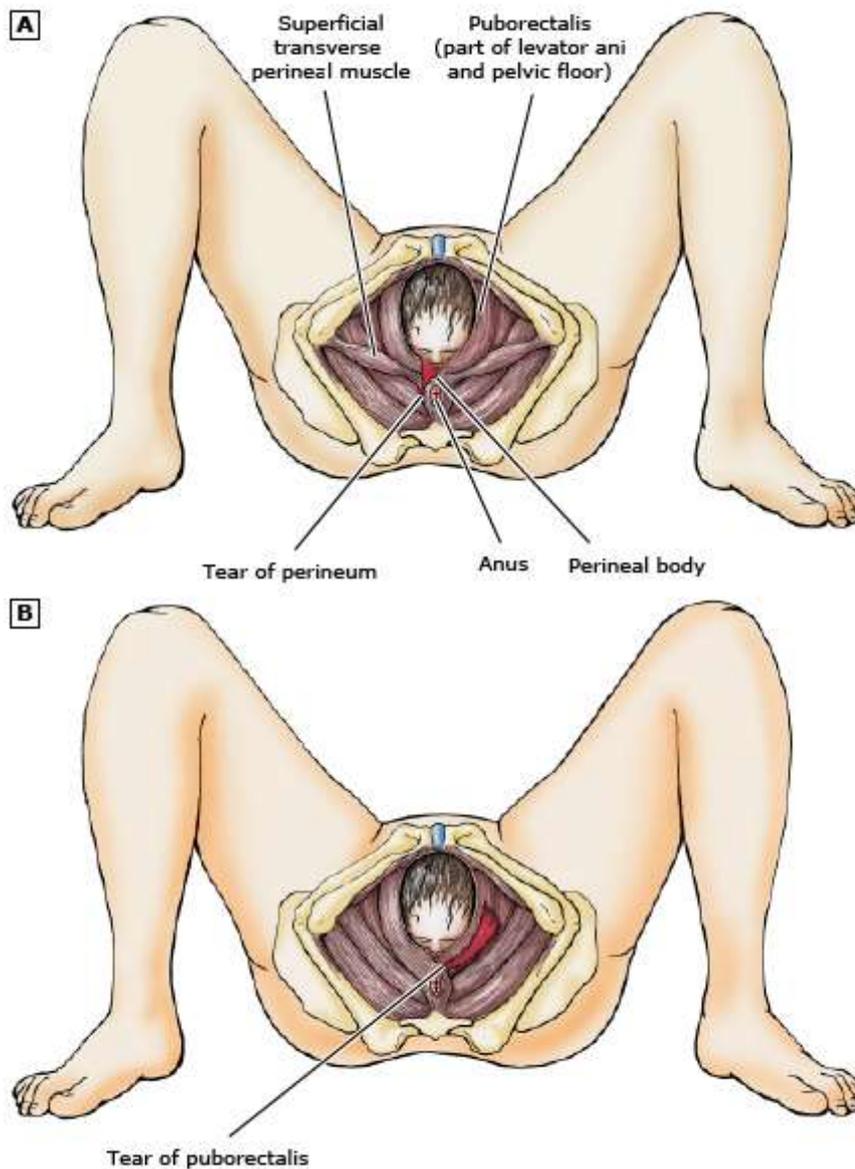
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Topic 8079 Version 42.0

## GRAPHICS

### Pelvic floor anatomy during childbirth



Injury to the pelvic floor.

(A) The perineum, levator ani, and pelvic fascia may be injured during childbirth.

(B) It is the puborectalis, the main part of the levator ani, that is usually torn.

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## Contributor Disclosures

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