Postpartum period: three distinct but continuous phases

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Summary

Postpartum period is distinct in three phases. The third phase is the delayed postpartum period, which can last up to 6 months. Some changes to the genitourinary system are much longer in resolving, and some may never fully revert to the prepregnant state. A burgeoning volume of literature on pelvic floor support implicates childbirth as the initiation of a whole host of conditions including stress urinary incontinence, incontinence of flatus or feces, uterine prolapse, cystocele, and rectocele. The duration and severity of these conditions affect many variables, including the patient's intrinsic collagen support, the size of the infant, the route of delivery, and the degree of perineal trauma occurring either naturally (lacerations) or iatrogenically (episiotomy). The initial or acute period involves the first 6–12 hours postpartum. This is a time of rapid change with a potential for immediate crises such as postpartum hemorrhage, uterine inversion, amniotic fluid embolism, and eclampsia.

The second phase is the subacute postpartum period, which lasts 2–6 weeks. During this phase, the body is undergoing major changes in terms of hemodynamics, genitourinary recovery, metabolism, and emotional status. Nonetheless, the changes are less rapid than in the acute postpartum phase and the patient is generally capable of self-identifying problems. These may run the gamut from ordinary concerns about perineal discomfort to periurinary cardiomyopathy or severe postpartum depression.

The third phase is the delayed postpartum period, which can last up to 6 months (1). Changes during this phase are extremely gradual, and pathology is rare. This is the time of restoration of muscle tone and connective tissue to the prepregnant state. Although change is subtle during this phase, it behooves caregivers to remember that a woman's body is nonetheless not fully restored to prepregnant physiology until about 6 months postdelivery.

Some changes to the genitourinary system are much longer in resolving, and some may never fully revert to the prepregnant state. A burgeoning volume of literature on pelvic floor support implicates childbirth as the initiation of a whole host of conditions including stress urinary incontinence, incontinence of flatus or feces, uterine prolapse, cystocele, and rectocele.

Many variables affect the duration and severity of these conditions, including the patient's intrinsic collagen support, the size of the infant, the route of delivery, and the degree of perineal trauma occurring either naturally (lacerations) or iatrogenically (episiotomy). Even when full recovery of pelvic floor integrity appears to be the case, menopause may elicit a return of many of these problems as the collagen support of estrogen is withdrawn (2). Although surgical intervention should not be considered until 6 months postpartum (the length of time for complete restoration of connective tissue support), an aggressive program of pelvic floor exercises may be prescribed at any time during the antenatal or postpartum course and has been shown to be of benefit in some series. Episiotomy and operative vaginal delivery increase the incidence of severe pelvic floor trauma and are proven risk factors for subsequent pelvic floor dysfunction, yet they were performed in 29% and 9% of vaginal births, respectively, in 2001 (3,4).

Genital tract lacerations are graded on a scale of one to four. First-degree lacerations involve only the vaginal mucosa or perineal skin.

Second-degree lacerations involve the muscles of the perineal body without transgressing the anal sphincter.
complex. Third-degree lacerations include any laceration of the external anal sphincter, and fourth-degree lacerations include laceration of the both the internal and external anal sphincter, and the rectal mucosa (5). Sphincter laceration is also associated with a 270% increase in sexual pain postpartum, when compared with women who deliver without perineal laceration (6). It is better to tear than to be cut; women who deliver with spontaneous lacerations report less pain with intercourse than women who undergo episiotomy.

The impact of less severe first- and second-degree laceration on pelvic floor function is unknown. The pudendal nerve is important to genitourinary change, too, as the pudendal nerve travels along the posterior wall of the pelvis and ultimately exits the pelvis to innervate the external genitalia, and because of its length and position, it is vulnerable to both compression and stretch injury, particularly during vaginal birth, when the fetal head is compressed pelvic floor (7,8).

Assessment of pudendal nerve terminal motor latencies (PNTML) before and after childbirth demonstrates alterations in women after vaginal birth or cesarean delivery after labor.

These changes do not appear during pregnancy (9), and often resolve by 6 months after birth (10). Women assessed 48 hours postpartum have significantly greater prolongation in PNTML when they have had a forceps-assisted vaginal delivery, compared with spontaneous vaginal birth (11).

Pelvic floor damage from childbirth can be measured objectively by the use of MRI of the levator ani complex. Nulliparous women do not have defects in levator ani musculature as evaluated by MRI. Up to 20% of primiparous women develop defects in the levator ani during vaginal birth (12). The defects occur most commonly in the pubovisceral portion of the levator ani.

The levator ani injuries have been shown to be associated with stress urinary incontinence, but it remains unclear if the defect is responsible for stress incontinence or is simply a marker of global pelvic floor injury (13). Stress or urge incontinence before pregnancy or childbirth is rare, occurring in less than 1% of women (14,15). Stress urinary incontinence that occurs during pregnancy is common, and affects up to 52% of primiparous women.

The causes of stress incontinence during pregnancy are thought to include maternal weight gain and increased mechanical pressure on the bladder from the enlarging uterus, and increased urine production from increased glomerular filtration rates. Although many women with stress incontinence during pregnancy report resolution of symptoms postpartum, the presence of incontinence during pregnancy may be predictive of postpartum incontinence (16,17).

Postpartum incontinence in the short term may be predictive of longer-term problems. Women with persistent stress urinary incontinence at 3 months postpartum have a 92% risk of having stress urinary incontinence at 5 years (18). Cross-sectional (19) and cohort studies (20,21) demonstrate a higher prevalence of stress incontinence in women who have had vaginal deliveries, compared with women only having cesarean deliveries. These differences are significant only in younger women; in older women, the stress incontinence risk factors of age and obesity outweigh the effect of childbirth and method of delivery (22).

Effects of childbirth on urge urinary incontinence are less well described. Urge incontinence may occur as commonly as stress symptoms after childbirth, affecting approximately 30% of postpartum women (23).

Forceps delivery and episiotomy have been associated with increased reports of urge incontinence, whereas cesarean delivery has been protective (23).

Fetal macrosomia has been associated with the development of both stress and urge symptoms. Not all incontinence is troublesome to patients. Most studies evaluating the incidence and impact of postpartum urinary incontinence compare women with any urinary incontinence with women with no incontinence, and do not include descriptions of the severity of incontinence. This omission underlies the importance of using reliable methods of obtaining information regarding functional outcomes. Injury to the bladder and ureters during cesarean delivery is an uncommon cause of urolologic injury. The bladder may be lacerated during cesarean delivery at the time of peritoneal entry, development of the bladder flap, incision of the uterus, or lysis of adhesions. Prior cesarean delivery is associated with increased risk of cesarean after onset of labor (24). Women having a fourth cesarean had a 1.2% risk of bladder injury, compared with 0.13% in a first cesarean delivery (25). Fortunately, most bladder injuries involve the dome of the bladder, with minimal long-term morbidity if recognized intraoperatively.

When bladder laceration is suspected but is not grossly apparent, further evaluation is done by filling the bladder with methylene blue, indigo carmine, or sterile milk, which is readily available on labor and delivery units. The ureters are rarely transected or occluded during cesarean delivery. Episiotomy and operative vaginal delivery are known risk factors in the development of anal incontinence symptoms. A meta-analysis of six randomized trials compared restrictive to liberal use of episiotomy in 4850 women, and concluded that liberal use of episiotomies conferred no benefit (26).

Operative vaginal delivery was reviewed similarly in 2582 women and it was found that vacuum delivery was associated with a much lower risk of anal sphincter laceration than delivery with forceps (relative risk 0.41, 95% CIs 0.33 to 0.50) (27). The incidence of anal incontinence following sphincter laceration increases over time. For example, in a cohort of 72 women who had anal sphincter laceration, 4% had stool incontinence at 3 months after delivery, and this increased to 17% 2 to 4 years later (28). Prevention of anal sphincter laceration and subsequent development of anal incontinence partly lies in decreasing the use of episiotomy and forceps delivery at the time of delivery.

Surprisingly, cesarean delivery does not necessarily prevent postpartum anal incontinence symptoms. Most women resume sexual activity by 8 weeks postpartum, and nearly all have some sexual complaints (29). Only 14% of women and 12% of men report no sexual problems whatsoever postpartum (30).

Six months postpartum, 35% of primiparous women complain of decreased sexual sensation and 24% of decreased sexual satisfaction, compared with function before childbirth.

In the same retrospective cohort, 22% also complained of dyspareunia (6). Intercourse-related problems can persist 12 to 18 months following delivery (31), and are
more common in women who underwent operative vaginal delivery (32).

Women who have episiotomies or spontaneous perineal lacerations complain of increased perineal pain, decreased sexual satisfaction postpartum, and delayed return of sexual activity, compared with women who give birth with an intact perineum (32,34). Women with severe perineal lacerations of the anal sphincter are more likely to report dyspareunia than women with an intact perineum. It is unclear whether or not cesarean delivery is protective of postpartum sexual complaints. Because cesarean delivery avoids genital tract trauma, it has often been assumed to protect sexual function postpartum (35).

Some investigators have found that increased reports of pain are limited to the immediate postpartum timeframe, with differences between cesarean and vaginal delivery groups resolving by 6 months postpartum. Nearly all studies that have examined the effect of mode of delivery on postpartum sexual complaints conclude that sexual dysfunction is highest in women undergoing operative vaginal delivery with forceps or vacuum (35). Does elective cesarean delivery prevent the development of pelvic floor disorders?

The National Institute of Health held a consensus conference titled “Cesarean Delivery on Maternal Request” in March 2006 to address this question (36). Although some pelvic floor disorders seem to be reduced in women who undergo elective cesarean delivery, the quality of the evidence supporting this was assessed as weak.

Of particular concern in the consensus statement was how long differences between vaginal and cesarean delivery persisted, and the potential impact of multiple cesarean deliveries on the development of pelvic floor disorders. For example, a comparison of women who underwent three cesarean deliveries with women after three vaginal births showed comparable rates of stress urinary incontinence, perhaps because of the cumulative effect of pregnancy itself or denervation injury during cesarean delivery (37).

When comparing elective cesarean to vaginal delivery, it is important to appreciate that all vaginal deliveries may not be equivalent in their impact on the pelvic floor. A style of actively coached pushing was associated with increased urge incontinence at 3 months, when compared with women who were not actively coached (38).

Much can be done to prevent pelvic floor disorders by limiting the use of episiotomy and operative delivery using forceps, known risk factors for pelvic floor dysfunction. One can learn from past mistakes. Episiotomy was adopted in the 1920s to protect the pelvic floor, without clear evidence to support its use (39). Years later, one third of women still undergo episiotomy, despite lack of evidence of benefit, and risk of significant harm (40). What is needed before making broad public health changes in delivery method is data comparing the effects of pregnancy alone, cesarean delivery (labored and unlabored), and vaginal birth. Perhaps then, physicians will have better information to help advise patients about the postpartum genitourinary tract changes they might expect.

References

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