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CONTROVERSIES-FOR

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Should women be offered elective cesarean section in the hope of preserving pelvic floor function?

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Pregnancy and vaginal delivery have long been known to be associated with an increased incidence of urinary incontinence, anal incontinence and genital prolapse. The development of stress urinary incontinence (SUI) appears to involve a genetic predisposition to the development of myofascial weakness following injury of the pelvic floor. Women who develop urogenital prolapse and incontinence heal with a higher proportion of type 3 than type 1 collagen which leads to weakened pelvic floor support. These genetic changes are likely multifactorial and are poorly understood.

Better understood is the association of SUI and anal incontinence with injury to the pudendal and perineal nerves. Several investigators have shown delayed pudendal nerve conduction in women who develop urogenital prolapse, anal incontinence and SUI. Delayed conduction decreases the strength, speed and duration of the reflex levator ani contraction and allows for posterior displacement of the urogenital tract with stress on its connective tissue supports, which eventually may result in urethral hypermobility and genital prolapse. This delayed pudendal and perineal nerve conduction also adversely affects anal sphincter function resulting in decreased resting and anal squeeze pressures.

Snooks et al. [1] reported on the 5-year follow-up of their original 1984 study in which they found that 80% of the 122 women sustained occult, but reversible pudendal nerve damage and had reduced anal pressures compared to controls. These changes were not seen in the women who delivered by cesarean section. Parity, forceps delivery and increased length of the second stage of labor were associated with more severe injury. Five years later, 36% had persistent increased anal sphincter fiber density and pudendal nerve terminal motor latencies compared to their antepartum measurements. All of these women developed symptomatic SUI and 60% developed flatal incontinence. Allen et al. [2] prospectively studied 96 nulliparas recruited at 36 weeks and found persistent decreased pelvic floor strength ($p = 0.0006$) and persistent EMG and pudendal conduction abnormalities in 80% 2 months after their first vaginal delivery. None of these changes occurred following elective cesarean section.

In a prospective trial, Sultan et al. [3] found that 28 of 79 primiparas (35%) had occult anal sphincter tears after vaginal delivery on ultrasonography, which persisted in all 22 women studied at 6 months postpartum. They found sphincter defects in eight of the ten women who underwent forceps delivery. None of the 23 women who had cesareans developed sphincter defects, anal incontinence or SUI, whereas 5% of primiparas and 4% of multiparas developed anal incontinence and 3% of primiparas and 21% of multiparas developed SUI 6 weeks following vaginal delivery. They also found significant decreases in resting and squeeze pressures on anal manometry in primiparas ($p < 0.001$) and multiparas ($p < 0.004$) and prolonged pudendal latencies in primiparas ($p < 0.001$) and multiparas ($p < 0.002$) 6 weeks after vaginal delivery but not following cesarean section.

Chaliha et al. [4] evaluated anal symptoms, sensation and manometric function in the third trimester and 3 months postpartum in 286 nulliparas, of whom 38% developed anal sphincter tears after vaginal delivery and 3% after cesarean. Vaginal delivery led to significant decreases in resting and squeeze anal pressures, while cesarean did not.

In the first prospective study of the effects of pregnancy on urinary function, Winifred Francis [5] studied 400 women (222 nulliparas and 178 multiparas) in the first trimester and carefully questioned them about urinary symptoms before pregnancy, during each trimester and postpartum. Antepartum SUI on cystometry occurred in 53% of nulliparas and 85% of multiparas.

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Mild SUI persisted in 38% and severe SUI in 9% of these women but in none of the 20 antepartum incontinent women who delivered by cesarean. Stanton et al. [6] prospectively studied 181 women in the third trimester and pueperium. Of the 83 nulliparas, 38% had SUI in the third trimester and 6% had persistent postpartum. Of the 98 multiparas, 10% had SUI prior to pregnancy, 42% in the third trimester and 11% had persistent postpartum SUI. Viktrup et al. [7] prospectively studied 305 primiparas about their urinary incontinence symptoms before, during, and after pregnancy. In those without antepartum SUI, 21 of 167 women (13%) developed it postpartum compared to the 35 delivered by cesarean ($p < 0.05$). After the first year, only 3% still had stress incontinence; but 5 years later, they questioned 91% of their original subjects and found a 30% prevalence of SUI but cesarean was again protective [8]. Hannah et al. [9] reported on the only prospective RCT of planned cesarean versus vaginal delivery in the International Randomized Term Breech Trial. They found cesarean section to be protective against urinary incontinence with a relative risk of 0.62 without any adverse effects of cesarean compared to vaginal delivery 3 months later.

These data show that vaginal delivery is associated with injury to the pudendal nerve, and an increased risk of urinary and anal incontinence that can be avoided by cesarean delivery. More randomized data is needed with long-term follow-up to clearly answer this debate.

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