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REVIEW ARTICLE

A review of the impact of pregnancy and childbirth on pelvic floor function as assessed by objective measurement techniques

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Abstract

The objective of this narrative review is to study the impact of pregnancy and childbirth on pelvic floor function as assessed by objective measurement techniques with quantitative data carried out during pregnancy and after childbirth. A literature search in MEDLINE and relevant and up-to-date journals from 1960 until April 2017 was performed for articles dealing with the impact of pregnancy and childbirth on pelvic floor function as assessed by objective measurement methods. Only studies describing objective measurement techniques. i.e., urodynamics, ultrasound (US), magnetic resonance imaging (MRI), Pelvic Organ Prolapse Quantification (POP-Q) system, and neurophysiologic tests carried out throughout pregnancy and after childbirth are included. Relevant studies presenting objective quantitative data are analyzed and briefly summarized. The number of studies meeting selection criteria was relatively few. Pregnancy, especially first pregnancy, is associated bladder neck lowering, increased bladder neck mobility, pelvic organ descent, decreased levator ani strength, and decreased urethral resistance. These changes are accentuated after vaginal delivery. Data on the impact of obstetrical and neonatal variables are transient and seem of less importance. Cesarean delivery is not completely protective. In most women, pelvic floor muscle function recovers in the year after delivery. Objective measurement techniques during pregnancy may allow identification of women susceptible to pelvic floor dysfunction later in life and offer the opportunity for counseling and preventive treatment strategies.

Keywords Pregnancy · Childbirth · Vaginal delivery · Cesarean delivery · Pelvic floor function · Objective measurement techniques

Introduction

Pelvic floor disorders (PFDs), including urinary incontinence (UI), stress urinary incontinence (SUI), overactive bladder syndrome (OAB), pelvic organ prolapse (POP), and anal incontinence (AI), represent a major health problem that affects about 25–30% of the adult female population. PFDs are associated with a negative impact on quality of life (QoL) and health-care expenditures. The impact of PFDs is likely to grow as the prevalence of these disorders increases with an aging population [1–6].

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Large, population-based epidemiological and cross-sectional observational studies have documented the relationship between parity, childbirth, and PFDs [6–14]. It is not clear to what extent pregnancy itself versus mode of delivery contributes to the development of PFDs in later life. Longitudinal studies with objective quantitative data to corroborate this association are relatively scarce. This narrative review focusses on prospective studies using objective measurement techniques during pregnancy and after childbirth to assess the impact of pregnancy and delivery methods on pelvic floor function. Measurement techniques include urodynamics, urethrocystography, ultrasound (US), magnetic resonance imaging (MRI), Pelvic Organ Prolpapse Quantification (POP-Q) system examinations, and neurophysiologic tests. Quantification data before and after childbirth are helpful in identifying the underlying pathophysiology and eventually may lead to improvement in treatment strategies. In this review, UI refers to all forms, and the review does not assess the association between pregnancy, childbirth, and AI.

Cystometry and urethrocystography

In 1960 Winifred Francis published the first prospective longitudinal study of a cohort of 400 unselected pregnant women [15]. All 268 women who had SUI (67%) had cystometry performed to confirm and assess symptom severity. Fifty-three percent of primigravid and 85% of multigravid women experienced SUI to some degree during pregnancy. Lateral urethrocystography was carried out in 83 women for obstetrical reasons during pregnancy: 33 women with SUI and 50 pregnant women with good bladder control. All 83 women consented to one further urethrocystography performed 2 days after delivery. Anatomic configurations of bladder and urethra returned to preparturition states during the first few postpartum days. Persistent severe incontinence after childbirth was observed in 9% of women, and 38% admitted to occasional incontinence during coughing or sneezing. Of the 20 women who were delivered by cesarean delivery, none continued to have UI after childbirth. Lateral urethrocystography revealed loss of posterior urethrovesical angle as the most striking feature in women with persistent symptoms of SUI. Results of this large prospective study led Francis to conclude that: "SUI, rarely, if ever, appears for the first time after childbirth, if it had not occurred before or during pregnancy. Symptoms tend to resolve in puerperium, but in a small percentage, UI persists or develops de novo after vaginal delivery. It recurs more severely with each subsequent pregnancy and ultimately may result in permanent disability. These observations suggest that pregnancy more readily predisposes to the onset of UI than vaginal delivery."

Loss of posterior urethrovesical angle, as observed in 90% of women with SUI, has long been considered the anatomical cause of SUI. Until the 1970s, plication of the pubovesical cervical fascia at the level of the bladder neck (Kelly stitches) to restore the anatomy of the urethrovesical junction was the procedure of choice for treating SUI.

Chaliha et al. carried out a prospective longitudinal study including 286 nulliparae with a singleton pregnancy. Sitting and standing dual-channel subtracted cystometry was performed at 34 weeks of gestation and in 161 women who returned 12 weeks postpartum. Antenatally, the prevalence of urodynamic SUI and of detrusor overactivity was 9 and 8%, respectively, and 5% and 7%, respectively, postpartum. Comparison of antenatal and postpartum variables showed a statistically significant increase in all postnatal bladder measurements, i.e., first and strong desire to void and maximum cystometric capacity, both sitting and standing, and a decrease in maximum voiding pressure and peak flow rate. Analysis of urodynamic measurements showed no evidence of any difference between women who had a normal vaginal delivery, instrumental delivery, or cesarean section. There was no evidence of a relationship between cystometric measurements and obstetrical and neonatal variables. The authors concluded that the changes in bladder function were consistent with the pressure effect of the gravid uterus on the bladder and did not appear to be affected by the method of delivery or neonatal variables [16].

Simultaneous urethrocystometry

Simultaneous urethrocystometry during pregnancy and after childbirth, with the use of dual-channel microtransducer catheters (Millar Instruments, Inc. Houston, TX, USA) for simultaneous cystometry and urethral pressure profilometry, was reported by Iosif and Ulmsten (1981) and van Geelen et al. (1982). Simultaneous urethrocystometry with microtip transducers embedded in a thin semiflexible catheter allows accurate and reproducible measurements of the intravesical and intraurethral pressure under both static and dynamic conditions [17]. Iosif and Ulmsten performed urethral pressure profilometry early in pregnancy, in the 38th week, and 5–7 days after delivery in 12 women with symptomatic SUI. Compared with 14 continent pregnant women, women with SUI had a shorter urethra and lower resting urethral closure pressure, which did not increase sufficiently to compensate for the progressive increase in bladder pressure during pregnancy [18]. Van Geelen et al. studied 43 healthy primigravidae with ongoing pregnancy. Simultaneous urethrocystometry at rest and during stress (coughing) was performed at 8, 16, 28, and 36 weeks' gestation and 2 months postpartum. At each session, blood samples were taken to determine 17-beta-estradiol (E2), progesterone (P), and 17-alpha-hydroxyprogesterone (17-OH-P) [19]. Prepregnancy values were available for five women [20]. Functional urethral length, urethral closure pressure, and the urethral closure response to stress did not change significantly during the course of pregnancy and were only slightly below the mean values found in healthy nulligravid women. However, during pregnancy, an increase in anatomical urethral length by ~4 mm was measured, which correlated significantly with the rise in 17-beta-estradiol ($p < 0.001$). Alterations in hormone levels were not correlated with a change in urethral pressure variables. Urethral pressure and length parameters were significantly decreased 8 weeks postpartum in all women who delivered vaginally when compared with early pregnancy values. The postpartum changes were not significantly correlated with the duration of the second stage of labor, infant birth weight, or presence or absence of episiotomy. In women delivered before or early in labor by cesarean delivery ($n = 6$), the estimated difference in mean values at 8 weeks postpartum and at 8 weeks of pregnancy was minimal. Twelve women (28%) developed symptoms of SUI of varying degrees during pregnancy. SUI manifested itself in those women who demonstrated a low urethral closure pressure and defective transmission of intra-abdominal pressure rise to the urethra early in pregnancy. These conditions

worsened during pregnancy and after delivery. Post partum, two women had objective evidence of SUI, and symptoms were present in four women. In five of these six women, symptoms began during pregnancy. Postpartum changes seemed to be permanent: urethral pressure profile measurements recorded in six women >1 year after delivery were similar to those obtained 8 weeks postpartum. Meyer et al. performed urethral pressure profilometry (Gaeltec Microtip catheter) during one of the three trimesters of pregnancy in 149 nulliparas and again about 9 weeks after childbirth. Results were compared with those of volunteer age-matched nonpregnant nullipara controls ($N = 19$). Compared with nonpregnant controls, in pregnant women, there was a significant decrease of urethral closure pressure at rest/stress and of the area of continence at rest/stress in both the supine and standing positions. Pregnant women with SUI (29%) have a lower urethral sphincter function at rest/stress. The authors concluded that pregnancy induces diminished urethral sphincter function. After vaginal delivery functional urethral length was decreased, but other indices of urethral sphincter function were unchanged. There was a significant decrease in intravaginal and intra-anal pressure. In women who had cesarean delivery, only slight modifications of these measurements were observed [21, 22]. Results of these studies suggest that pregnancy itself and an inherent or acquired weakness of the urethral spincteric mechanism play a key role in the pathogenesis of SUI. Vaginal delivery further adversely affects the urethral sphincteric mechanism and pelvic floor supportive structures (Table 1).

Perineal ultrasound and the bladder neck

Perineal US is a simple, noninvasive, reproducible method of evaluating bladder-neck position and mobility. The technique is readily available, has good interobserver and interdisciplinary reliability, and allows for dynamic assessment of pelvic organs at rest and during straining, with visualization of the integrity of the pelvic floor supportive structures [23, 24]. Comparative studies found a good correlation between radiologic scanning of the bladder neck and perineal US [25, 26]. Both posterior rotational descent of the proximal urethra and bladder neck are strongly correlated with SUI [27–29]. However, bladder-neck mobility is common in asymptomatic nonpregnant nulliparous women and may vary from 4 to 32 mm during coughing and from 2 to 31 mm during Valsalva [30, 31]. Consequently, there is overlap between continent and incontinent women, and an internationally accepted US definition of bladder-neck hypermobility is lacking. Longitudinal studies with perineal US performed under standardized conditions during pregnancy and after childbirth can detect changes in pelvic floor function in individual women and may be helpful in identifying women with increased risk of postpartum pelvic floor disorders.

Pesschers et al. prospectively studied 25 primigravidas, 20 multiparas, and ten women who were to have elective cesarean delivery. Perineal US was performed at 36–42 weeks of pregnancy and 6–10 weeks after delivery. Results were compared with data obtained in 25 healthy nulligravid volunteers. In pregnancy, position of the bladder neck at rest was significantly more caudad and mobility significantly greater when compared with age-matched nulligravid volunteers. After vaginal delivery, the position of the bladder neck was significantly lower compared with those who had cesarean delivery and nulligravid volunteers. An increase in bladder-neck mobility during Valsalva was observed in most women delivering vaginally but not in those who delivered by elective cesarean delivery. The ability to elevate the bladder neck is restored in most women 6–10 weeks postpartum. In primipara, but not in multipara, levator ani muscle strength, measured by perineometry 6–10 weeks after vaginal delivery, was significantly decreased compared with antepartum values [32, 33].

King and Freeman performed antenatal and postnatal measurements of bladder-neck mobility in a cohort of 103 primigravid women with no pre-existing UI or neurological disorder. From the first visit at approximately 15–17 weeks, the women were seen every 10 weeks throughout their pregnancy and at 10–14 weeks postpartum. Antenatally, mild SUI was present in 47.6%, persistent occasional SUI after delivery in 18.4%, and de novo SUI postpartum in 3.9%. Antenatal SUI was associated with an increased risk of postpartum SUI [relative risk (RR = 3.3).] Women with postpartum SUI had significantly greater bladder-neck mobility antenatally than continent postpartum women (RR = 8.7). Cesarean delivery was protective for all measures of postpartum bladder-neck mobility. There was a greater incidence of SUI in mothers of women with postpartum SUI. There were no significant differences in any obstetrical variables, except for forceps delivery, between postpartum continent and incontinent women, which suggests that the mode of delivery is of minor importance. These observations support the urodynamic data that pregnancy itself and hereditary factors are of greater importance than parturition trauma to the development of SUI and pelvic organ prolapse in later life. Daily postpartum pelvic floor exercises were associated with a markedly reduced incidence of UI [27]. Similarly, Meyer et al. found that women with SUI during pregnancy had a significantly lower bladder-neck position, more bladder-neck hypermobility, and diminished urethral sphincteric function compared with continent pregnant women and nulliparous controls [21, 22].

Dietz and Bennett performed a prospective observational study of 169 nulliparous women at 6–18 weeks' and 32–37 weeks' duration and at 2–5 months after childbirth. Vaginal delivery was associated with significantly increased

Table 1 Urethral pressure profile (UPP) variables

Authors	Participants	Weeks	Pregnancy		After childbirth		Comments
			Early pregnancy		8 weeks postpartum		Supine position
			FUL	UCP	FUL	UCP	
Iosif, Ulmsten 1981 [18]	14 primi continent 12 primi SUI	12–16	29 ± 4	78 ± 9	27 ± 4 24 ± 3	72 ± 9 38 ± 11*	Women with SUI have shorter FUL and low UCP.
Van Geelen et al. 1982 [19, 20]	43 primi 5 controls	8–16–28–36	31 ± 5 32 ± 5	76 ± 14 84 ± 7	24 ± 2	70 ± 8	28% women SUI shorter FUL and low UCP: nonsignificant.
Meyer et al.1998 [21, 22]	149 primi	$N = 15$ in week 12–20 $N = 62$ in week 21–30 $N = 72$ in week 31–40	35 ± 9	78 ± 21**	30 ± 7	84 ± 27**	No significant differences in UPP during pregnancy.
	19 controls		32 ± 7	106 ± 21			

Primi nulliparous pregnant women, *FUL* functional urethral length (mm), *UCP* urethral closure pressure in (cm H_2O),

*UCP continent primi vs. UCP primi with SUI, $p < 0.001$

**UCP controls vs. UCP during pregnancy and after childbirth, $p < 0.0001$

mobility of the proximal urethra and bladder neck and descent of the anterior vaginal wall, cervix, and anterior rectal wall. The increase was most pronounced after forceps delivery and less so in those who had elective cesarean delivery. The study did not present data of changes in bladder-neck position or mobility during pregnancy and did not compare US parameters with those of nulliparous women. No significant associations were documented between symptoms of bladder dysfunction and US parameters of bladder-neck mobility [34].

Wijma et al. performed a prospective longitudinal study of 117 primigravid women and 27 nulliparous age-matched asymptomatic controls. Bladder-neck position and mobility were related to intra-abdominal pressure changes simultaneously recorded by a microtip pressure transducer (Gaeltec) high in the posterior fornix. Serial investigations were done at 12–16, 28–32, and 36–38 weeks of pregnancy and at 6 weeks ($n = 117$) and 6 months after vaginal delivery in 62 women. At each visit, a questionnaire on symptoms of incontinence and a 24-h pad test were included. Perineal US at 12–16 weeks' gestation showed that the resting angle of the bladder neck was already significantly increased compared with the nonpregnant control group (51.5 and 44.5°, respectively) and further widened during pregnancy to 62° at 36–38 weeks ($p < 0.001$). Median displacement/pressure gradient, expressed as displacement of the bladder neck in degrees of abdominal pressure change during coughing showed, a significantly increasing trend during pregnancy, but no changes were seen during Valsalva. These observations indicate that the dynamic qualities of pelvic supportive structures are already affected early in pregnancy and hardly change as pregnancy progresses, whereas a significant decrease in pelvic floor contractility occurs [35]. Six months after vaginal delivery, the resting angle of the urethrovesical junction was still significantly greater than in the nulliparous control group. The number of women reporting incontinence at 38 weeks of gestation dropped from 35% to 10% at 6 months postpartum. The percentage of women with a positiv 24-h pad test decreased from 14% at 38 weeks to 5% at 6 months postpartum. No correlations were found between urethrovesical measurements, UI, and obstetrical variables [36].

Toozs-Hobson et al. studied 110 primigravid women recruited between 32 weeks and term and who completed the 6-month follow-up. Transvaginal US measurements of the levator hiatus and bladder-neck mobility were assessed at rest, maximum strain, and Valsalva. Urethral sphincter volume was calculated using a three-dimensional vaginal probe. Seventy-three women delivered vaginally and 37 by cesarean section. Compared with antenatal measurements, vaginal delivery was associated with a lower bladder-neck position, increased bladder-neck mobility and levator hiatus size, with a decrease of urethral sphincter volume. After cesarean delivery, bladder-neck position returned to its antenatal position, and the size of the levator hiatus and urethral sphincter volume were reduced. Comparison with nulliparous nonpregnant women was not possible without a control group. The most striking finding in that study was the difference antenatally between delivery methods: women delivered by cesarean had less distensible pelvic floors and less bladder-neck displacement antenatally compared with those delivered vaginally [37]. (Table 3).

Jundt et al. recruited 112 primigravid women between weeks 32 and 37 of their uncomplicated singleton pregnancy. Ninety-nine women returned for follow-up 6 months after childbirth. A detailed questionnaire concerning UI and AI was administered, pelvic examination using the POP-Q grading system, and perineal and endoanal US were performed at each visit. SUI and flatal incontinence significantly increased from before pregnancy (3 and 12%) to after vaginal delivery (21 and 28%, respectively), while no women with cesarean

delivery had new symptoms. There was no significant difference between bladder-neck position before and after delivery. Bladder-neck mobility was significantly greater after vaginal childbirth, especially after vacuum delivery, compared with cesarean delivery. Six-month postpartum primiparous women with SUI showed significantly increased bladder-neck mobility when compared with continent primiparous women. Apart from a decrease in perineal length after vaginal delivery, the quantitative parameters of the pelvic floor showed no differences before and after childbirth. However, the first POP-Q exam was performed late in pregnancy, so comparison with parameters in early pregnancy was not possible [29] (Table 2).

Imaging techniques and the pelvic floor:

During the last two decades, 2D and 3/4D translabial or transperineal US has been a highly valuable diagnostic tool in evaluating levator ani morphometry and function. Under standardized conditions, identical system settings, and in skilful hands, this investigational technique is reproducible and shows good correlation with MRI in detecting major levator ani defects [43–45]. However, there is a substantial learning curve in carrying out the procedure and interpreting images, as—even in asymptomatic nulliparous women—interindividual differences in levator ani morphometry exist [46]. Dietz and co-workers published a prospective study in 61 nulliparous women with translabial US performed at 36–40 weeks' gestation; 50 returned for their postpartum examination 2–6 months after delivery. Levator avulsion from its insertion on the arcus tendineus fascia pelvis was diagnosed in 36% who delivered vaginally [47]. In a second study, using the same standardized methodology in 367 primiparous women, levator avulsion was detected in 13% and overdistension (microtrauma), defined as >20% increase in hiatal area on Valsalva, occurred in 28.5%. Forceps delivery was associated with a three- to fourfold increase in levator avulsion and obstetrical anal sphincter injury. Prolonged second stage of labor was associated with overdistension, whereas epidural anesthesia had a protective effect [38]. The same research group reanalyzed US volume data of 393 nulliparous pregnant women seen in the late third trimester and again 4 months postpartum. Data was compared with that of 63 nulliparous nonpregnant volunteers. Hiatal dimensions and urethral mobility were determined as outcome measures. Comparison of third-trimester data with that of nonpregnant nulliparae revealed a 27 and 41% increase in hiatal area at rest and on Valsalva and an increase in urethral mobility by 64–91% in late pregnancy. The marked differences in hiatal dimensions and urethral mobility persist when comparing nonpregnant nulliparous women with women after prelabor/first-stage cesarean delivery, suggesting that hormonal and mechanical changes of pregnancy may have an irreversible effect on pelvic supportive structures [48]. DeLancey et al., using MRI in a case-controlled study of 80 nulliparous and 160 primiparous women, found that 20% of primiparous women had a visible defect in the levator ani muscle, whereas no levator ani defects were identified in nulliparous women. The authors concluded that active pushing during the second stage of labor led to distension and stretching of the pelvic floor muscles. In about one third of women delivering vaginally, this resulted in levator ani muscle injury, which most frequently involves the pubovisceral portion of the muscle [42].

Staer-Jensen et al. carried out a prospective longitudinal study in a cohort of 300 primiparous women using 3D/4D perineal US performed at 21 and 37 weeks of gestation and at 6 weeks and 6 and 12 months after delivery. Eighty-five percent had a vaginal delivery, and 15% underwent cesarean delivery. Assessment included measurement of the levator hiatal area, bladder-neck mobility, and rest-to-Valsalva hiatal area difference. At 6 weeks postpartum, major levator ani defects were diagnosed in 18.8% of women after vaginal delivery. Women with major levator ani defects at 6 weeks postpartum had a significantly smaller hiatal area at rest and during Valsalva both at 21 and 37 weeks of gestation and less shortening of levator ani muscle during contraction at 37 weeks [40, 41]. At 6 months postpartum, levator ani muscle defects were still present in 10.6%. Most recovery occurs during the first 6 months, but not all women recover to the 21 -week pregnancy level. Comparing the 1-year postpartum results with data at 21 weeks' gestation showed no difference for levator hiatus at rest and during contraction, whereas bladder-neck mobility and hiatal area during Valsalva were slightly increased. These observations are in agreement with results of a comparable prospective observational study by Van Veelen et al. of 280 nulliparous women. Perineal US was performed in early pregnancy and at 36 weeks. Between 12 and 36 weeks' gestation, there was a significant increase in absolute values of hiatal dimensions and in hiatus contractility and distensibility. Women who delivered vaginally showed a persistent significant increase in hiatal dimensions on Valsalva maneuver, whereas women who delivered by prelabor or first-stage cesarean delivery showed no significant changes on Valsalva maneuver and pelvic floor contraction but a significant decrease in hiatal area at rest [49]. In a secondary analysis of this observational cohort study, the authors measured mean echogenicity of the puborectalis muscle at rest, during pelvic floor contraction, and Valsalva in 254 women. Results revealed that women who delivered by cesarean because of failure to progress had a significantly lower mean echogenicity in pelvic floor contraction at 21 weeks' gestation than women who delivered vaginally or by elective cesarean delivery. There was no difference in mean echogenicity of the puborectalis muscle between women who had a spontaneous vaginal delivery versus operative vaginal delivery [50]. These observations support the hypothesis by Toozs-Hobson et al.

Table 2 Perineal ultrasound and the bladder neck

Authors	Participants	Weeks	Pregnancy		After childbirth		Comments
			BN		BN		
			Position	Mobility	Position	Mobility	
Pesschers et al. 1996 [33]	25 primi	36–42	–	++	–	+++	UI symptoms not assessed in this study
	20 multi		–	++	–	+++	
	11 elect. CD		–	+	–	+	
	25 controls		/	/	/	/	
King-Freeman 1998 [27]	103 primi	15–17	–	+	–	++	Increased antenatal BN mobility correlates significantly with SUI
	18 CD	25–27	–	+	–	++	
	No controls	35–37					
Meyer et al. 1998 [21, 22]	149 primi	12–38	+	–	+/–	+++	Low BN position and increased mobility correlates with SUI
	46 controls		/	/	/	/	
Dietz-Bennett 2003 [34]	169 primi	16–18	–	–	++–	++	BN changes post-partum not correlated with bladder dys-function
	45 primi CD	32–37	–	–	+	+	
	No controls						
Wyma et al. 2001–2003 [35, 36]	117 primi	12–16 28–32	+	+	++	+++	BN measurements not correlated with UI symptoms.
		36–38	++	++	++	+++	
	27 controls		/	/	/	/	
Toosz-Hobson et al. 2008 [37]	73 primi VD	32–38	–	++	–	+++	Antepartum BN mobility differs significantly between VD and CD groups
	37 primi CD		–	+	–	+	
	No controls						
Jundt et al. 2010 [29]	92 primi VD	32–37	–	+	–	++	BN mobility correlated significantly with SUI especially after Vacuum Delivery.
	20 primi CD		–	+	–	+	
	No controls						
Staer-Jensen et al. 2015 [41]	255 primi VD	21–37	+	++	+	++	BN mobility increased significantly during pregnancy. At 12 months postpartum no difference between VD and CD
			+	++	+	++	
	45 primi CD		+	++	+	++	
	no controls						

Primi: nulliparous pregnant women, *Multi*: multiparous pregnant women, *BN*: bladder-neck position and mobility, *VD*: vaginal delivery; *CD*: cesarean delivery, *UI* urinary incontinence, *SUI* stress urinary incontinence

+, ++, +++ level of increased descent/mobility

/ controls

suggesting there may be inherent differences in the biomechanical properties of the pelvic support system that predispose toward mode of delivery [37] (Table 3). Measuring mean echogenicity of pelvic floor muscles and mobility may become a valuable diagnostic tool in assessing the characteristics of pelvic floor function. The clinical value of these findings needs further investigation.

Electrophysiologic tests:

Neurogenic damage to pelvic floor muscles was first documented by Snooks et al., who showed that vaginal delivery but not elective cesarean delivery resulted in damage to innervation of pelvic floor muscles and was associated with an increased incidence of fecal (FI) and (UI) [51]. In 1986, those authors published a prospective study of 122 consecutively referred women. In 51 women, single-fiber electromyogram (EMG) was studied 6 months prior to delivery and 2 months post partum. Pudendal nerve terminal motor latency (PNTML) was performed 48–72 h after delivery and 2 months postnatally in 71 women studied after delivery and in 34 nulliparous controls. Analysis of all 122 women showed that multiparity, forceps delivery, increased duration of second stage, third-degree tear, and high birth weight were important factors leading to pudendal nerve damage. Epidural anesthesia had no effect on pudendal nerve function [52]. Allen et al. performed a comparable prospective study of 96 nulliparous women recruited at 36 weeks' gestation. The investigational techniques used were concentric needle EMG, assessment of pelvic floor muscle contraction using a perineometer, and PNTML within 5 days after delivery. At 2 months postpartum, 75 women consented to have the EMG and PNTML repeated. EMG changes in the duration of motor unit potentials (MUP) and polyphasia indicated that labor and vaginal delivery cause partial denervation of the pelvic floor in 80% of women delivering their first baby. There were no significant differences in mean PNTML measured in the immediate puerperium and at 2 months after delivery. The degree of denervation was slight, with EMG evidence of reinnervation in most women. As for pelvic floor contraction, there was a significant reduction immediately after delivery [5.4, standard deviation (SD) 4.9 cm H_2O] with some recovery at 2 months postpartum (10.1 SD 6.9 cm H_2O) but not to the original antenatal power (15.6 SD 9.2 cm H_2O). Factors associated with more severe denervation were duration of active second stage of labor and heavier than average baby, while forceps delivery and perineal tears did not significantly affect innervation [53]. Denervation may progress with future deliveries and aging and finally lead to symptomatic PFD. Sultan et al. carried out the first prospective study to investigate the effect of childbirth on PNTML and perineal plane [54]. PNTML studies, measured bilaterally, and perineometry were performed antenatally after 34 weeks' gestation and repeated 6–8 weeks after delivery. One hundred and five of the original 128 pregnant women returned for the postnatal investigation. Twenty-two women were restudied after 6 months. Vaginal delivery, particularly the first, was associated with a significant increase in PNTML at 8 weeks postpartum. In two thirds, abnormally prolonged PNTML was in the normal range when restudied after 6 months. The degree of perineal descent on straining is most marked after the first vaginal delivery, but the plane of the perineum at rest did not significantly change in multipara. A significant correlation was found between the incremental difference in perineal descent on straining and the incremental change in PNTML. In agreement with the studies by Snooks et al. [52] and Allen et al. [53], there was a significant association between PNTML and duration of the active second stage of labor and birthweight >4 kg, but no significant difference between PNTML increments in women delivered by forceps or vacuum and normal delivery. In women who had cesarean delivery after the onset of labor, an increase in PNTML was observed, but not following elective cesarean delivery. Patient characteristics and other obstetrical variables were not significantly associated with prolonged PNTML or abnormal perineal descent.

Tetzschner et al. performed PNTML measurement in a cohort of 146 healthy pregnant women between 30 and 40 weeks of pregnancy and 12 weeks after delivery. Their results confirmed the results of Sultan et al., showing an increase in PNTML after vaginal delivery and after cesarean delivery during labor. In that study, duration of the second stage of labor, infant's head circumference and weight, pudendal block, and epidural analgesia had no significant influence on PNTML measurements, whereas vacuum extraction was associated with a significant increase, of 0.2 ms ($p = 0.04$). Women with UI ($n = 25$) had a significantly increased PNTML: 2.20 ± 0.58 ms. [55]. In most women, pudendal nerve damage causes little or no clinical symptoms, since reinnervation and muscle hypertrophy compensate for prior denervation. In a small proportion of women, nerve damage was permanent, which may cause pelvic floor symptoms in later life. (Table 4).

POP-Q

Few authors have prospectively performed POP-Q during pregnancy and the first year after the first delivery. Elenskaia et al. performed a longitudinal cohort study in 182 nullipara women to prospectively evaluate the impact of childbirth on

Table 3 Effect of pregnancy and childbirth on hiatal dimensions

Authors	0-parous	12 weeks	37 weeks		After Child birth	
Toozs-Hobson et al. 2008 [37]	–	$N = 110$	$N = 73$ VD	$N = 37$ CD	$N = 73$ VD 6 months	$N = 37$ CD 6 months
Hiatal area R			15.0	14.3	15.4	13.6
Hiatal area V			15.5	15.8	18.6	14.9
Hiatal area C			13.5	13.3	13.8	12.3
	0-parous	12 weeks	37 weeks		After Child birth	
Shek et al. 2012 [38, 39]	$N = 63$		$N = 393$		$N = 128$ VD subset:ref.39	$N = 105$ CD
Hiatal area R (with avulsion)	11.93		15.11		16.09 ** (20.31)	14.17 ***($p = 0.01$)
Hiatal area V (with avulsion)	15.31		21.55		22.61 ** (25.46)	19.56 ***($p = 0.03$)
**hiatal area VD vs 0-parous $p < 0.001$						
***hiatal area after CD vs o-parous $p = 0.01$/ $p = 0.03$						
	0-parous	21 weeks	37 weeks		Normal VD	Instrumental VD
Staer Jensen et al. 2013/14 [40, 41]			Mean		hiatal dimensions at 37 weeks	
	–	$N = 300$	$N = 274$		$N = 184$	$N = 47$
Hiatal area R		11.70	13.70		13.92*	12.70**
Hiatal area V		15.40	18.70		18.91*	17.47
Hiatal area C		09.20	10.40		10.61*	09.77**
* vs 21 weeks $p < 0.01$						
**instrumental vs normal VD, $p < 0.01$						
	0-parous	12 weeks	36 weeks		After child birth	
Van Veelen et al. 2014 [49]	–	$N = 231$	$N = 231$		$N = 193$ VD	$N = 30$ CD: prelabor
Hiatal area R		14.73	16.06*		15.23*	13.16*
Hiatal area V		17.68	20.08*		23.00*	18.50
Hiatal area C		11.75	12.64*		12.68*	10.71
*VS0 12 weeks $p < 0.001$						

CD Cesarean delivery, *VD* Vaginal Delivery

Hiatal area: *R* at rest, *V* Valsalva, *C* contraction *vs* versus

pelvic organ support, pelvic floor symptoms, and QoL. Pelvic organ support was objectively assessed by POP-Q and symptoms subjectively evaluated using the validated electronic Personal Assessment Questionnaire for the Pelvic Floor (ePAQ-PF) in the second trimester and at 14 weeks, 1 year, and 5 years after delivery ($n = 97$). There was a significant worsening of POP and subjective prolapse symptom scores at 14 weeks and 1 year after vaginal delivery without a corresponding change in condition-specific QoL. After cesarean birth, there was a significant increase in POP stage at 14 weeks but not at 1 and 5 years. The size of the genital hiatus increased significantly, and the perineal body decreased significantly at 5 years' follow-up after both vaginal and cesarean delivery. The other POP variables did not significantly change between 1 and 5 years after delivery [56]. These observations concur with results of other longitudinal studies, all of which report an increase in POP during pregnancy in 30–50% of nulliparous women. At 6 weeks postpartum, POP-Q stage was higher than in the first trimester and significantly higher after vaginal compared with cesarean delivery [57–60]. Cesarean delivery is only partially protective, as POP to or beyond the hymen was also found in 5% of women who had undergone elective cesarean [60, 61]. Nygaard et al. analyzed ten studies that measured pelvic floor support in the first postpartum year. In their studies, maximum vaginal descent (MVD) at or below the hymen ranged from 0 to 41%. In an unpublished pilot study, 18% of primiparous women had MVD > 0 at 1 year postpartum [62]. These findings are at variance with results of a prospective study carried out in Norway of 300 nulliparous women. In that study, the prevalence of POP during pregnancy was low and ranged from 0 to 10%. At 6 weeks after childbirth, pelvic organ descent was present after both vaginal and cesarean deliveries but was more pronounced after vaginal delivery. The number of women having an enlarged hiatus increased during pregnancy and at 12 months postpartum. No difference between delivery groups was found. At 12 months postpartum, all POP-Q points, except the cervix, had recovered to baseline [63].

Table 4 Electrophysiologic tests

Authors	Participants	Weeks	Pregnancy		After childbirth		Comments
			PNTML (ms)	EMG	PNTML (ms)	EMG	
Snooks et al. [51, 52]	50 primi	± 16	–	1.34 ± 0.05	2.0 ± 0.2	1.58 ± 0.12*	Single fiber
	52 multi		–	1.44 ± 0.16	2.1 ± 0.3	1.67 ± 0.18*	EMG increase after childbirth
	34 controls				1.9 ± 0.2	1.37 ± 0.09	* $P < 0.01$
Allen et al. [53]	96 primi	± 36	–	3.3 ± 0.6	2.0 ± 0.2	5.2 ± 1.5**	Significant increase MUP; Concentric needle EMG ** $p < 0.00001$
Sultan et al. [54]	105	± 34	Bilateral	Perineum	Bilateral	Perineal descent	
	57 primi		R. 1.91 L. 1.96	1.3 cm	R. 2.00 L. 2.06**	1.7 cm***	Increase PNTML after childbirth **$p < 0.0001$
	32 multi		R. 1.93 L. 1. 96	1.6	R. 2.01 L. 2.09**	1.8 cm*	Increase perineal descent ***$p < 0.0001$ *$p < 0.006$
	16 CD						
	74 controls				R. 1.91 L.1.95		
Tetzschner et al. [55]	146 68 primi	30–40	PNTML 1.7		PNTML 2.0**		PNTML increase after childbirth **$P < 0.001$
	78 multi		1.8		2.1**		
	9 CD						
	Controls				1.90 ± 0.5		

Primi: nulliparous pregnant women, *Multi* multiparous pregnant women, *CD* cesarean delivery, *VD* vaginal delivery, *PNTML* pudendal nerve motor latency (ms), *EMG* electromyography, *MUP* motor unit potentials

Discussion

The aim of this narrative review was to describe changes during pregnancy and after childbirth in anatomic and functional variables of pelvic floor supportive structures as assessed by objective measuring techniques. Clinical implications were determined as the second outcome measure.

Pregnancy, especially the first pregnancy, reveals inherent weakness of the pelvic supportive structures and is predictive for symptoms of pelvic floor dysfunction in later life. Studies on the composition of connective tissue in continent and stress incontinent women have shown defective connective tissue in women with SUI [64–66]. These observations, results of investigative measurement studies, and a positive family history for PFDs suggest that a genetic predisposition plays an important role in the etiology of pelvic floor dysfunction in later life. Collagen is the main component of the endopelvic fascia. Landon et al. elegantly demonstrated that during pregnancy, the mechanical properties of connective tissue alter. The pregnant fascia stretches to a much greater length before tissue failure occurs but is also much weaker. Less stress is required to produce tissue failure due to reduced tensile strength [67]. The correlation between the increase in urethral length in pregnancy and the increase in estradiol levels and marked differences in urethral mobility seen in early pregnancy lead one to suggest that the rise in hormone levels may affect qualitative properties of pelvic floor supportive tissues [20]. The mostly temporary incontinence in pregnancy must be interpreted as the result of predisposing hereditary factors, hormonal alterations, and increased pressure on the urethrovesical junction [15, 16, 18, 19, 27, 64, 68, 69]. This may also explain why cesarean delivery, elective or after onset of labor, is not totally protective against the development of pelvic floor dysfunction. Stretching of the connective tissues beyond their physiological limits during labor, with subsequent injury to the levator ani muscle and/or neurogenic damage, occurs in most women delivering vaginally, but in most of them, reinnervation, wound healing, and muscle hypertrophy compensate for that denervation and muscle trauma. In a minority (5–20%) of women, major levator ani defects and neurogenic injury may lead to irreversible changes in pelvic floor function. Subsequent pregnancies, hereditary factors, aging, and obesity are major factors that contribute to the development of pelvic floor disorders later in life [1, 10, 12–14, 68–74]. Data on the impact of other obstetrical and neonatal variables on pelvic floor function (e.g., duration of second stage of labor, vacuum extraction, episiotomy, perineal laceration, birth weight, epidural anesthesia, and use of oxytocin are not consistent and seem of minor importance [7, 13, 14, 19, 70, 75–82]. Forceps delivery is associated with increased risk of PFDs, whereas cesarean

delivery is relatively protective [7, 8, 13, 14, 38, 57, 70, 75–78, 83–85]. The efficiency of perineal muscle function, as measured with a perineometer, is not related to the degree of perineal trauma but significantly related to the extent that women undertook regular exercise [86]. During the last decade, several randomized controlled trials have clearly shown that intensive and supervised PFMT, whether or not combined with biofeedback, is effective for improving prolapse symptoms and in reducing POP-Q stage in some women [87–90].

Conclusion

Pregnancy is associated with bladder-neck descent, increased bladder-neck mobility, increased POP, decreased urethral resistance, and loss of pelvic floor contractility. These changes are compatible with changes in mechanical properties of fascial tissue and can be considered a physiological adaptation to mechanical and hormonal alterations in pregnancy. Generally, these changes cause minor restrictions in lifestyle and do not affect condition-specific QoL [56, 91]. The first vaginal delivery is when most women are likely to sustain pelvic floor damage, such as, neurogenic injury, change in bladder-neck position and mobility, levator ani trauma, increase in levator hiatus, and anal sphincter disruption. In most women, pelvic floor muscle function recovers during the year following delivery. In a minority, symptoms persist and may finally lead to PFD in later life.

Proper history taking [the Incontinence Impact Questionnaire (IIQ)}, perineal US at rest and during straining, screening for bladder-neck mobility, and digital control of pelvic floor contractility at midpregnancy, will allow the detection of women susceptible to pelvic floor dysfunction. It also offers the opportunity to initiate preventive treatment strategies, such as supervised pelvic PFMT and/or pessary placement. There is circumstantial evidence that antenatal and postpartum PFMT is effective in decreasing the prevalence and/or severity of UI and POP in the postpartum period. PFMT and lifestyle interventions (weight loss, no smoking) are the first treatments of choice for PFDs. The added value of biofeedback and temporary pessary treatment needs further evaluation. The effectiveness of PFMT in preventing PFDs in later life requires long-term longitudinal studies, which may be difficult to perform.

Compliance with ethical standards

Conflict of interest Hans van Geelen: nothing to disclose. Don ostergard: nothing to disclose. Peter Sand reports being consultant and/or speaker for Allergan, Astellas, Boston scientific, Medtronic, Cook Myosite, Roivant, Valencia technologies, Outpost Medical.

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