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URETHRAL CLOSURE PRESSURES AMONG PRIMIPAROUS WOMEN WITH AND WITHOUT LEVATOR ANI MUSCLE DEFECTS

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Abstract

Introduction and hypothesis—Vaginal birth is an established risk factor for levator ani (LA) defects and incontinence. We hypothesized an association between urethral pressure profiles and LA defects.

Methods—160 primiparous women, 9–12 months postpartum, including, MRI assessment for LA defects, urodynamic testing, and assessment of vaginal closure force by instrumented speculum. Urodynamic testing included resting maximal urethral closure pressure (MUCP) and urethral closure pressure with a pelvic floor contraction or Kegel (KUCP). We examined the relationships between MUCP, KUCP, LA defect status, and vaginal closure force.

Results—There was no significant association between MUCP or KUCP in women with and without LA defects ($p=0.94, p=0.95$). Additionally, there was no correlation between MUCP and vaginal closure force ($r=.06, p=.41$), and a weak correlation between KUCP and vaginal closure force ($r=.20, p=.01$).

Conclusions—In this population, urethral pressure profiles are unrelated to LA defect status after vaginal birth, indicating that the mechanism responsible for LA damage spares the urethra.

Keywords

Incontinence; Kegel; Levator ani; MRI; Pelvic floor muscles; Postpartum; Urodynamics; Urethral closure pressure

Introduction

It is well established that vaginal delivery leads to higher rates of stress urinary incontinence (SUI) as well as to levator ani (LA) muscle damage [1, 2]. Primiparous women with SUI have been shown to have lower maximal urethral closure pressure (MUCP) on resting

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urethral pressure profilometry and are twice as likely to have visible LA defects compared to continent primiparas [3].

The etiology of this lower MUCP and its relationship to LA defects has not been evaluated. In light of the aforementioned data, it is reasonable to expect that lower MUCP would be associated with LA defects. Thus, the primary objective of the study was to test the hypothesis that there is an association between maximal urethral closure pressure (MUCP) and LA defects when studied in primiparous women. Secondary objectives included investigating if: (1) a woman's ability to augment urethral closure pressure with a pelvic floor contraction (Kegel) differs by her LA defect status, and (2) if urethral functional measures correlate with vaginal closure force, a functional measure of the LA.

Materials and methods

This is a secondary analysis of an IRB approved study of 160 primiparous women recruited within the first year after vaginal birth. Participants included 80 women with *de novo* postpartum stress incontinence (SUI) and 80 continent women. The purpose of the parent study was to identify birth-related structural impairments or tissue damage post delivery specific to stress incontinence. Both groups of women had to have been continent prior to pregnancy by self report. In the SUI group the women had to experience symptoms of SUI that persisted at between 9 and 12 months postpartum. Postpartum stress continence/incontinence status was confirmed by standing stress test on examination. Study participants underwent magnetic resonance imaging as well as urodynamic testing [3].

Magnetic resonance imaging protocol and technique was completed as described in Chou, et al [5]. In brief, imaging was performed on a 1.5-Tesla system (Signa, General Electric, Milwaukee, WI) using multiplanar two-dimensional fast spin images with 4mm thick slices and slice spacing of 1mm, yielding an image spacing of 5mm. Axial, sagittal and coronal views were taken of all women. Each woman's axial and coronal images were analyzed independently by two examiners, who scored the integrity of the right and left sides of the LA muscle on a scale of 0–3. A score of 0 was assigned when no visible muscle damage was observed, a score of 1 when < 50% of the muscle was missing, a score of 2 when > 50% of the muscle was missing, and a score of 3 when complete loss of visible LA muscle was noted. Relevant images of specific defects are included in Kearney, et al [4]. If the patient had a grade 3 defect on at least one side or a score of 4 or more when both sides' scores were added, that was classified as a "major defect". Other LA defect scorings were classified as "minor defect" and no abnormalities noted were classified as "no LA defect" [1]. For the purposes of this analysis we reduced the data to "any LA defect," and "no LA defect." This reduction was performed to provide an adequate sample size to test the hypothesis. ANOVA analysis comparing the MUCP in the no defect, minor defect, and major defect was also performed.

Urethral function was tested by measuring MUCP at rest and during a pelvic muscle contraction effort (Kegel) using an 8F Gaeltec (Medical Measurements Incorporated, Hackensack, NJ) dual-tipped microtransducer urodynamic catheter, with the transducers directed laterally. The subject was asked to remain relaxed during a manual pull through of the catheter from the intravesical space to air, with maximal point determined as the highest urethral resting pressure. Then, with the transducer held at the point of highest resting urethral pressure, each woman was asked to perform a Kegel effort while an additional measure was made [6]. Measures were repeated, two or three times, based on the patient's comfort with the test, and then averaged. These measures were made on a bladder passively filled to 250cc as a part of the parent study protocol, which required full bladder leak point pressures. Pressure tracings were digitally stored for later data reduction.

Data reduction included subtracting the resting bladder pressure from each of the urethral measures, then averaging the three repeated measures, first for relaxed and then for the pelvic floor contraction or Kegel maneuvers. The difference between relaxed measures and Kegel effort measures was also computed and called “Augmentation UCP” (augmentation UCP = KUCP – MUCP). Although KUCP and augmentation UCP are not standard ICS abbreviations, and represent parameters not typically measured, yet these are illustrative of LA function for this investigation [7].

Strength of the LA muscles was quantified as vaginal closure force. This protocol and technique was completed as described in Miller, et al [8]. In brief, vaginal closure force was measured using an instrumented strain-gauged vaginal speculum. This speculum measured force in the mid-sagittal plane at rest. The amount of additional force generated during a maximal voluntary contraction above the resting value was also measured. Measurements were taken twice and averaged.

To determine the sample size required to address our hypothesis, we looked to the parent study. In this study, MUCP between the continent and incontinent primiparas differed significantly, and 23 of the 80 women in the SUI group were found to have LA muscle defects on MRI as compared to 9 of the 80 continent women [7]. We then performed a two tailed power calculation, assuming that the LA defect status was associated with MUCP between the continent women and incontinent women (83 ± 21.0 s.d. cm/H₂O and 62.9 ± 25.2 s.d. cm/H₂O respectively). In order to capture a comparable difference in MUCP we would need a sample size of 21 in each group with and without a LA defect, to have 80% power to detect this difference with an alpha of 0.05. With our sample size, we had a 98% power to detect this difference in MUCP between the groups with and without a LA defect.

Descriptive statistics were used to describe group mean scores on the continuous variables. One-way ANOVA or its nonparametric equivalent (Kruskal-Wallis when assumptions for parametric testing were not met) were used to test for differences by LA defect status in MUCP, KUCP and LA strength. We used graphical presentation to assess for linearity or other pattern of relationship and Pearson correlation R to estimate the strength of relationship between measures of urethral function and LA function. Definitions and units conform to the standards recommended by the ICS, except where specifically noted [8].

Results

One hundred and sixty women were included in the study, 80 with *de novo* postpartum incontinence and 80 with demonstrated continence at 6–9 months postpartum. Twenty nine of the 160 women (18%) had LA defects. As reported in the parent study, this LA defect group was older (32.8 ± 5.9 vs 29.3 ± 4.7 $p < .001$), had more anal sphincter ruptures (OR 8.1, 3.3–19.5), forceps deliveries (OR 14.7, 4.9–44.3), and more episiotomies (OR 3.1, 1.3–7.2) than the group without defects. Please see Kearney et al. for further comparison of defect status and birth variables [4]. Twenty of the 29 women (70%) with levator defects had SUI [4]. The remaining clinical and demographic variables are reported elsewhere [3, 4].

Table 1 presents the urodynamic variables stratified by the presence or absence of LA defects. It reveals that between these two groups, there is scant difference in MUCP, KUCP or UCP augmentation. Figure 1 is a graphical representation of this data, showing MUCP by continence groups and then within each group, by defect status. This figure demonstrates that although the continent group overall had a higher MUCP, within each group, those with and without LA defects showed similar MUCP scores. In the continent group, the MUCP cm H₂O mean $\pm$ sd was 83.3 ± 19.9 for those with no LA defect and 92.2 ± 29.5 for those

with LA defect. In the incontinent group, the MUCP cm H₂O mean $\pm$ sd 62.4 ± 24.0 for those with no LA defect and 64.1 ± 28.9 for those with LA defect.

Table 2 presents our analysis of vaginal closure force at rest, during a pelvic muscle contraction, and the difference in vaginal closure force. Women with LA defects showed less ability to augment their vaginal closure force with Kegel effort compared to women with no LA defects (1.3 ± 0.8 vs 2.28 ± 1.6 Newtons).

Table 3 presents MUCP, KUCP and its relationship to LA vaginal closure force at rest as well as to vaginal closure force with Kegel effort. KUCP and vaginal closure force with a Kegel effort were weakly correlated ($r=.20$, $p=.01$). However, r values for other urethral measures and vaginal closure forces were closer to zero, with $p>0.05$.

DISCUSSION

In this study, urethral function (measured as MUCP, KUCP, or UCP augmentation), observed within the first year after delivery, does not differ in women with and without levator ani muscle injury. The group with LA defects were able to increase their intraurethral pressure during a Kegel effort similarly to the group without a LA defect, by about 12–13 cm H₂O. Not surprisingly, the group showing injured LA muscles was not able to generate as much vaginal closure force with a Kegel effort as the group without LA muscle injury, but the urethral pressures did not seem to differ in like manner. The hypothesis, that there is an association between MUCP and LA defects is not supported.

Our findings suggest that we cannot assume the vaginal childbirth related mechanisms responsible for LA muscle injury are also responsible for observed differences in urethral pressure between, incontinent and primiparous women [1]. This is consistent with Shek's observational study that demonstrated another postpartum functional change, urethral mobility, is not directly related to continence status [10].

The potential implications of this are as follows. First, it is important to evaluate the two components of the continence mechanism, urethral closure pressure and levator ani support, separately after birth as MUCP change may not necessarily accompany LA change or other anatomical change. The two factors and their relationship to each other should continue to be studied, since changes to both are associated with clinical symptoms, yet potential interaction effects and degree of effects are still largely unknown, and existing studies have been underpowered to evaluate this. Second, birth events that injure the LA do not necessarily limit a woman's ability to augment MUCP with a Kegel effort. This latter finding, that some women with known LA defects can still improve urethral closure pressure on Kegel effort, is similar to the data reported by Miller et al. in a sample of older women with and without LA defects [6]. Plausibly, even with less LA force generated, there can still be augmentation of urethral closure pressures on Kegel effort, presumably through contraction of the striated muscular components of the urethra; pointing to some utility in pelvic muscle training for targeting the striated muscle of the urethra in conjunction with LA activation in carefully selected women.

It is also important to acknowledge that urethral pressure measures are subject to artifact. The measure itself can be subject to artifact, because the urethral wall comes into contact with the transducer and this may cause directional differences. We use flexible catheters and orient the transducer laterally to minimize these effects. Additionally, there are several ways to assess the increase in urethral pressure that occurs during a pelvic muscle contraction and they can likewise be subject to artifact. We have chosen to find the point of maximal pressure and to carefully hold the catheter at this position rather than having it in a catheter puller where the catheter cannot follow the movement of the urethra. In addition, we sought

to assure that the pressure before and after the contraction were similar. There is certainly a possibility that some catheter movement occurs in certain patients and can bias the measures, yet at the time of the study, newer strategies such as sleeve catheters were not in use.

Secondary analysis limits some of the findings of this study. Our work is powered only to detect a large difference in MUCP such as that observed in the parent study between continent and stress incontinent women. Similarly, it is possible that our finding of a weak but statistically significant association between LA strength and KUCP might be stronger if the sample size of those with LA defects was larger. Though we encourage our findings to be viewed with caution, we present this data as a consequence of new understandings about continence mechanisms and birth injury. In our prior work, MUCP was found to be significantly lower in women with postpartum SUI and these women were found to have more LA defects [3]. These observations led to the question about the relationship between urethral pressure measures, LA defect status and LA function. This new analysis offers increased clarity of our original findings insofar as neither LA defect status nor functional status directly relate to urethral functional measures. To our knowledge we are the first to offer any analysis of these potential inter-relationships.

In summary, birth-related changes to the levator ani muscle do not explain prior findings of MUCP differences by continence status after first delivery, nor do these changes fully explain differences in ability to augment MUCP through a Kegel effort. We did not find an association between two measurements that would be expected to be associated, since both are associated with SUI (MUCP and LA anatomy). This suggests that they are independent of one another. Further investigation of postpartum injury and function is warranted.

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References

1. DeLancey JO, Kearney R, Chou Q, Speights S, Binno S. The appearance of levator ani muscle abnormalities in magnetic resonance images after vaginal delivery. *Obstet Gynecol.* 2003; 1:46–53. [PubMed: 12517644]
2. Rortveit G, Hannestad YS, Daltveit AK, Hunskaar S. Age- and type-dependent effects of parity on urinary incontinence: The Norwegian EPINCONT study. *Obstet Gynecol.* 2001; 6:1004–10. [PubMed: 11755545]
3. DeLancey JO, Miller JM, Kearney R, Howard D, Reddy P, Umek W, et al. Vaginal birth and de novo stress incontinence: Relative contributions of urethral dysfunction and mobility. *Obstet Gynecol.* 2007; 2:354–62. [PubMed: 17666611]
4. Kearney R, Miller JM, Ashton-Miller JA, DeLancey JO. Obstetric factors associated with levator ani muscle injury after vaginal birth. *Obstet Gynecol.* 2006; 1:144–9. [PubMed: 16394052]
5. Chou Q, DeLancey JO. A structured system to evaluate urethral support anatomy in magnetic resonance images. *Am J Obstet Gynecol.* 2001; 185:44–50. [PubMed: 11483902]
6. Miller JM, Umek WH, Delancey JO, Ashton-Miller JA. Can women without visible pubococcygeal muscle in MR images still increase urethral closure pressures? *Am J Obstet Gynecol.* 2004; 191:171–5. [PubMed: 15295360]
7. DeLancey JO, Kearney R, Chou Q, Speights S, Binno S. The Appearance of Levator Ani Muscle Abnormalities in Magnetic Resonance Images After Vaginal Delivery. *Obstet Gynecol.* 2003 Jan; 101(1):46–53. [PubMed: 12517644]

8. Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, et al. The standardization of terminology of lower urinary tract function: report of the terminology subcommittee of the International Continence Society. *Neurourol Urodyn*. 2002; 21:167–178. [PubMed: 11857671]
9. Miller JM, Ashton-Miller JA, Perruchini D, DeLancey JOL. Test-retest reliability of an instrumented speculum for measuring vaginal closure force. *Neurourol Urodyn*. 2007; 26:858–863. [PubMed: 17357114]
10. Shek K, Dietz H, Kirby A. The effect of childbirth on urethral mobility: a prospective observational study. *J Urol*. 2010; 184(2):629–34. [PubMed: 20639028]

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Figure 1. Resting MUCP portrayed by continence status and LA defect status

This figure depicts the resting mean urethral closure pressure (MUCP) in women with and without levator ani (LA) defects. They are divided by continence status as well as by levator defect status.

Table I

Urodynamic parameters by defect status.

Closure Pressure cm H ₂ O	Levator defect		Levator defect		p	Levator defect		p
	NONE	ANY	NONE	ANY		MINOR	MAJOR	
	n=128	n=29	n=9	n=20				
MUCP	73.8 (24.0)	72.2 (31.3)	73.2 (25.7)	71.7 (34.3)	0.75			0.94
KUCP	87.4 (30.8 SD)	85.0 (21.6)	85.4 (37.6)	85.1 (44.6)	0.69			0.95
Augmentation	13.2 (18.9)	12.8 (21.6)	12.1 (31.6)	13.2 (16.0)	0.93			1.00

Standard deviations (SD) are in parenthesis. **KUCP** signifies the urethral closure pressure during a pelvic floor contraction, or Kegel. **Augmentation** is the difference between MUCP and KUCP.

* ANOVA analysis was performed comparing the values of the none, minor defect and major defect groups.

Table II

Levator strength by LA muscle defect status.

	Levator defect NONE n=128	Levator defect ANY n=29	p v NONE ANY
LA Force (Newtons)			
Rest	2.04 (1.0)	1.6 (0.2)	.03
Kegel	5.7 (2.99)	4.39 (3.22)	.050
Increase with Kegel	2.28 (1.6)	1.3 (0.8)	<.0001

Standard deviations (SD) are in parenthesis. **Rest** signifies the resting vaginal closure force at rest while Kegel signifies the vaginal closure force during a pelvic floor contraction, . Increase with Kegel is the increase in vaginal closure force.

Table III

Associations between LA Muscles and Urethral Closure Pressure.

Variables (n=156)	r	p
MUCP v LA Strength	0.07	0.41
MUCP v LA Rest	−0.05	0.50
KUCP v LA Strength	0.20	0.01
KUCP v LA Rest	0.09	0.27

Correlations between MUCP and LA muscle strength (LA Strength) measured during a Kegel effort as well as at rest, and urethral closure pressure during a Kegel effort (KUCP) correlated with LA muscle strength and LA muscle at rest.