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Vaginal birth and *de novo* stress incontinence: Relative contributions of urethral dysfunction and mobility

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Abstract

Background—Vaginal birth increases the chance a woman will develop stress incontinence. This study evaluates the relative contributions of urethral mobility and urethral function to stress incontinence.

Methods—This is a case-control study with group matching. Eighty primiparous women with self-reported new stress incontinence 9–12 months postpartum were compared to 80 primiparous continent controls to identify impairments specific to stress incontinence. Eighty nulliparous continent controls were evaluated as a comparison group to allow us to determine birth-related changes not associated with stress incontinence. Urethral function was measured with urethral profilometry, and vesical neck mobility was assessed with ultrasound and Q-tip test. Urethral sphincter anatomy and mobility were evaluated using MRI. The association between urethral closure pressure, vesical neck movement, and incontinence were explored using logistic regression.

Results—Urethral closure pressure in primiparous incontinent women (62.9 $\pm$ 25.2 s.d. cm H₂O) was lower than in primiparous continent women (83.0 $\pm$ 21.0, $p < 0.001$; effect size $d = 0.91$) who were similar to nulliparous women (90.3 $\pm$ 25.0, $p = 0.09$). Vesical neck movement measured during cough with ultrasound was the mobility parameter most associated with stress incontinence; 15.6 $\pm$ 6.2 mm in incontinent women versus 10.9 $\pm$ 6.2 in primiparous continent women ($p < 0.0001$, $d = 0.75$) or nulliparas (9.9 $\pm$ 5.0, $p = 0.33$). Logistic regression disclosed the two-variable model (max-rescaled $R^2 = 0.37$, $p < 0.0001$) was more strongly associated with stress incontinence than either single variable models, urethral closure pressure ($R^2 = 0.25$, $p < 0.0001$) or vesical neck movement ($R^2 = 0.16$, $p < 0.0001$).

Conclusions—Lower maximal urethral closure pressure is the parameter most associated with *de novo* stress incontinence after first vaginal birth followed by vesical neck mobility.

INTRODUCTION

Urinary incontinence is the most prevalent pelvic floor dysfunction linked to birth (1) and is responsible for 16 billion dollars annual expense. (2) Stress urinary incontinence (SUI) affects 86% of incontinent women either alone (50%) or in conjunction with urge incontinence (36%). (3) Rortveit (4) found a relative risk of 2.4 for developing stress incontinence in a population-based study of 15,307 women with 14.7% of parous women having symptoms of stress incontinence compared with 4.7% for nulliparous women. Despite the association between vaginal delivery and stress incontinence, it is not clear exactly what structures are damaged by birth that result in an increased likelihood of stress incontinence.

Concern about birth-induced injury resulting in stress incontinence, and other pelvic floor dysfunction, has been an important factor in the debate over cesarean section on maternal request. (5) The recent State-of-the-Science Conference on Cesarean Delivery on Maternal Request has identified a critical knowledge gap concerning birth-induced injuries and the mechanisms responsible for pelvic floor dysfunction. (6) This lack of knowledge prevents rational decision-making concerning pelvic floor injury and birth.

The current paradigm for understanding the cause of stress incontinence suggests that it is attributable to loss of urethral mobility and abnormal sphincter function, but the relative strength of association between each of these 2 factors, and *de novo* stress incontinence, is not known. (7–9) Studies of the continence mechanism after birth have focused on vesical neck movement alone (10,11) or were conducted early in the postpartum period before full recovery had occurred. (12) The present study seeks to compare the relative contributions of urethral function and urethral mobility to stress incontinence at a point in time when the pelvic floor has fully recovered.

METHODS

Study Group

We enrolled 240 women from the University of Michigan Obstetrical Unit, and the surrounding Ann Arbor area, to constitute three groups of 80 women: nulliparous continent women, primiparous continent women and primiparous stress incontinent women. The comparison of nulliparous continent with primiparous continent women allows one to identify the postpartum changes not associated with stress incontinence; while comparison of primiparous continent and primiparous incontinent women allows postpartum changes that are associated with stress incontinence to be identified. A sample size of 80 women in each group was selected to have a power of 0.81 to detect moderate effect sizes of 0.45.

Primiparous women were recruited by reviewing delivery records for all primiparous women who gave birth between January 1, 1997 and March 1, 2000. Women were excluded if they delivered before 35 weeks, had a cesarean section, or delivered twins. Six months postpartum, a letter was sent to eligible women asking for their willingness to be contacted about participation. Those expressing interest were called to confirm eligibility and discuss participation. Nulliparous women were recruited from the Ann Arbor area through newspaper advertisements and recruitment posters during the same time frame.

To be included in the stress incontinent group, woman had to have been continent prior to pregnancy by self report and experience stress incontinence symptoms after delivery that persisted at the time of testing (between 9 and 12 months postpartum). No specific number of

incontinent episodes per week was required as long as the woman demonstrated stress incontinence on examination. Women with some urge incontinence symptoms were allowed to participate as long as they identified stress incontinence as the major symptom. In addition they had to demonstrate the physical finding of stress incontinence during examination. Evaluation at 9–12 months postpartum was selected because normal healing of muscle and connective tissue injuries has likely taken place, yet most women would not have conceived again. Women were considered continent if they denied any type of incontinence and if they did not demonstrate incontinence during a full bladder stress test. Women were excluded for a history of recurrent urinary tract infections, genital anomalies, known neurological disease, claustrophobia, or implanted foreign bodies that would preclude MRI examination. Because age (13) and race (14) affect continence, controls were matched to cases for age and race. To accomplish this, as women were enrolled as cases, control women were recruited to provide the groups of similar age and race. Current and previous continence experience was evaluated by written questionnaire along with basic demographic, contraceptive, and menstrual information. Obstetrical data were abstracted from the delivery record.

Testing was conducted by the Urogynecologist members of the team. Because of the obvious nature of stress urinary incontinence during the examination it was not possible to blind the testing personnel to continence status. Those investigators evaluating MRI scans and ultrasounds were blinded to parity and incontinence status.

Functional assessment: Urodynamic and pelvic floor testing

Urethral function was tested by measuring maximum urethral closure pressure using an 8 Fr. Gaeltec ® (Medical Measurements Incorporated, Hackensack N.J.) urodynamic catheter with the transducers directed laterally. The average of 3 profiles was taken. (15)

Urethral mobility was quantified in two ways: Q-tip and ultrasound testing. Standard assessment of urethral axis in the supine position was quantified by using a sterile cotton swab (“Q-tip”) placed in the urethra to measure the angle from the horizontal, using a protractor with 0 degrees at the horizontal (16) at rest, during Valsalva and during maximal pelvic muscle contraction. Positive numbers reflect deflection of the visible portion of the Q-tip above the horizontal reference line and indicate a change in urethral mobility.

Because Q-tip assessment cannot reliably be performed during the functionally relevant action of a cough or in the standing position, these tests were supplemented with translabial ultrasound in the standing posture at rest, during a cough, a Valsalva maneuver, and a volitional pelvic floor muscle contraction as previously described. (17) During her one year stay in our unit, a single examiner made ultrasound measurements of all subjects from recorded videotapes of ultrasound obtained during the clinical examination that had been completed at that time (57, 52, and 60 for the NC, PC, and PI groups respectively). Because significant experience is needed to obtain consistent results, and sample sizes available with this examiner had a power slightly over 0.80 when the effect size is 0.55 for both the NC vs. PC and PC vs. PI comparisons, we chose not to have a second individual analyze the scans performed after her departure. The number of ultrasounds analyzed is given in the Results section.

Strength of the levator ani muscles was quantified in the mid-sagittal plane using an instrumented strain-gauged vaginal speculum (US Patent #: 6,468,232 B1). Force measured at rest, and the amount of additional force generated during a maximal voluntary contraction above the resting value, was measured. (18,19) Such measurements have demonstrated good reliability. (20) Pelvic organ prolapse was quantified using the Baden-Walker (21) system because at study initiation we had extensive experience with this system and the standardized pelvic organ prolapse system had only recently been described. (22)

Structural assessment: MRI protocol and analysis

Scan technique—Multiplanar proton density 2D fast spin images of the pelvic organs were obtained with an echo time of 15 msec and a repetition time of 4 sec using a 1.5 Tesla superconducting magnet (Signa; General Electric Medical Systems, Milwaukee, WI) with version 5.4 software. The slice thickness was 4 mm with a slice spacing of 1 mm yielding an image spacing of 5 mm. A 160×160 mm field of view and an imaging matrix of 256×256 were used.

Structure of the urethra and striated urogenital sphincter muscle—The length of the muscular urethra was measured as the distance from the internal urinary meatus to the distal end of the perineal membrane where the urethra becomes fibrous (23,24), and the length of the striated urogenital sphincter was assessed from this landmark to the top of the visible sphincter. Examiners were blinded to the continence status and parity of subjects. The outer and inner diameters of the striated sphincter were measured at the transverse equator and thickness was calculated as half the difference in the diameters. One nulliparous continent woman, five primiparous continent women and eight primiparous incontinent women did not show up for their MRI scans or did not complete the scans due to claustrophobia. Inadequate visibility of the urethral layers combined with suboptimal scan quality resulted in an inability to make urethral measurements in one primiparous continent woman and 11 stress incontinent women. The resulting sample sizes are tabulated in the Results section.

Levator ani muscle integrity—Technique and findings of levator integrity in this population have been previously reported (25) and are included here in order to be reviewed with the other variables.

Statistical Analysis

For continuous outcomes, we made the pair-wise group comparisons in the context of one-way analysis of variance models. Because the pair-wise comparisons were pre-planned, the significance levels are unadjusted. For continuous variables related to pregnancy or delivery, two-sample t-tests were used to compare the two primiparous groups. Chi-square statistics from two-way contingency tables were used to make two or three group comparisons. The Fisher exact test was used to make two-group comparisons when the assumptions for the chi-square test were not met. A conservative p value of 0.01 was considered statistically significant in view of multiple comparisons. Cohen's effect size (d), was used to assess the degree to which differences in continuous variables (e.g., MUCP or Q-Tip angle) were associated with group status (primiparous continent versus incontinent and nulliparous continent versus parous continent) by expressing the difference between the means of the two groups in units of standard deviation (Cohen's $d = M_1 - M_2 / \sigma_{\text{pooled}}$ where $\sigma_{\text{pooled}} = \sqrt{[(\sigma_1^2 + \sigma_2^2) / 2]}$

Logistic regression models were used to study the extent to which individual variables or combinations of variables predicted continence status among primiparous women. We studied a battery of variables selected conceptually as potential key contributors to the continence mechanisms. We also explored demographic/descriptive sample variables found to be significantly different between groups.

The statistics used to compare models included the significance levels associated with individual regression coefficients, the area under the receiver operator curve, the percent of cases correctly predicted for an optimally chosen cut-point, and the Max-rescaled R-square statistic, an indication of the degree to which one variable explains another. (SAS/STAT User's guide, Version 8, SAS Institute, Inc., Cary, NC, 1999)

RESULTS

Study Population and Symptoms (Table 1)

Study population demographics are reported in Table 1. Women with stress incontinence were heavier, but not taller, resulting in a greater BMI. Among the incontinent women, 50.0% had incontinence at least once a week, with 12.8% having daily incontinence. 'Moderate bother' was noted by 49% and 11.3% reported restricting their activities because of the incontinence. There were 66.6% of incontinent women who were unable to defer urination to a convenient time and 42.5% indicated that this restricted their activities. There were no differences between the primiparous groups with respect to contraceptive use, breastfeeding status, or menstrual history.

Urethral Sphincter Function and Structure (Table 2)

Maximum urethral closure pressure was 25% lower in stress incontinent women compared with primiparous continent women (Table 2) with a Cohen's d effect size for predicting stress incontinence of 0.91. The nulliparous women and the primiparous continent women had similar values indicating that birth, in the absence of stress incontinence, was not associated with altered urethral function.

MRI assessment of urethral structure revealed that the striated urogenital sphincter muscle was 18% shorter in women with stress incontinence compared with continent primiparas while the overall urethral length was 5% shorter in the stress incontinent group. Although there were differences in urethral diameter and striated urethral sphincter thickness, these did not reach statistical significance.

Standing Assessment of Vesical Neck Mobility Under Stress (Ultrasound) (Table 3)

During a cough, the vesical neck descended 50% more in the stress incontinent group yielding an effect size of 0.75. This cough-specific change in vesical neck movement in the upright position was limited to incontinent women and did not occur in continent nulliparas or primiparas. Of note, vesical neck movement during the Valsalva maneuver was similar among the three groups.

Supine Assessment of Vesical Neck Mobility under Stress (Urethral axis: "Q-tip") (Table 3)

Urethral axis at rest in the supine position was greatest in incontinent primiparas (five degrees greater than nullipara), and did not deflect downward as much during pelvic muscle contraction when compared with either nulliparous or primiparous continent controls. Increased mobility (change in axis from rest) during Valsalva maneuver was 40% greater in all primiparous women compared with nulliparous women, and greatest in stress incontinent women (51% greater than nulliparous controls) with a Cohen's d effect size of 0.68.

Levator Ani Muscle Function (Table 3)

Women who had given birth vaginally had a lower resting vaginal closure force than nulliparous women. At rest, primiparous continent women had 33% lower closure force and primiparous incontinent women had 26% lower closure force compared to nulliparous women. During maximal voluntary contraction the additional vaginal closure force generated above the resting state was 41% lower in continent primiparas and 46% lower in incontinent primiparas than in nulliparas. Both findings indicate reduced vaginal closure force following vaginal delivery; however, this finding is not specific to stress incontinence. These values were not statistically different between the two primiparous groups.

Levator Ani Muscle Morphology (Table 3)

All nulliparous women had normal appearing muscles on MRI. Primiparous women were more likely to have a visible muscle defect, as previously reported. (26) Primiparous women with stress incontinence were twice as likely to have visible defects in the levator ani muscle as continent primiparas.

Vaginal Wall Mobility During Valsalva (Table 4)

The position of the anterior vaginal wall, at or below the hymen, during Valsalva was altered in both primiparous groups (when compared with the nulliparas), and was twice as common in the incontinent as in the continent primiparas. Descent of the posterior wall was more common among primiparas, but did not differ between continent and incontinent primiparas.

Relative Association of Urethral Sphincter Function and Urethral Mobility with Continence

Variables explored for generating the logistic regression model included: maximal urethral closure pressure; augmentation of MUCP with Kegel effort; urethral length and tri-laminar length on MRI; ultrasound measures (vesical neck movement associated with cough, Kegel, and Valsalva and the difference between the Valsalva and cough measure on ultrasound); Q tip measures at rest, strain and Kegel; levator ani defect status on MRI; speculum measures of resting and maximal voluntary contraction force; neonatal head circumference; and operative delivery. Of all of these measures the only ones that were significant when included in a simple univariate logistic model were: MUCP; urethral length; Q-tip measures of rest, strain, and Kegel; and vesical neck movement with cough on ultrasound evaluation. A number of these measures were significant when combined with MUCP in 2 variable models, but little was gained when a third variable was added.

MUCP and the ultrasound cough measures were the strongest measure predictors as judged by effect size. Considering this, and because urethral length and tri-laminar length are both urethral measures, and because the Q-tip and the cough on ultrasound are both measures of structure, we elected to focus on MUCP and the cough ultrasound measures as the strongest and most parsimonious bivariate model.

Data for maximal urethral closure pressure and vesical neck movement during cough are displayed in Figure 1. Logistic regression analyses confirmed that maximal urethral closure pressure was more strongly associated with continence status than assessment of urethral mobility. The one-variable model using maximal urethral closure pressure had a better ability to predict incontinence (max-rescaled $R^2 = 0.25$, $p < 0.0001$) than the one-variable model using only vesical neck movement (max-rescaled $R^2 = 0.16$, $p = 0.0002$). For BMI the max-rescaled R^2 was 0.114, $p = 0.0006$. The two-variable model using maximal urethral closure pressure and vesical neck movement increased the ability to predict the occurrence of stress incontinence (max-rescaled $R^2 = 0.37$, $p < 0.0001$) compared with either of the one-variable models, demonstrating the importance of impairments in these two different systems in describing the pathophysiology of stress incontinence.

When we explored demographic/descriptive information about the sample groups, the only significant difference found between groups was BMI. When the parameters listed in Table 2 were calculated adjusting for BMI the mean values and significance levels remained the same with the exception of striated muscle length whose significance level changed from slightly significant to below the level of statistical significance ($p = 0.08$). We also explored adding BMI to each of the above models. The results after adding BMI were as follows: MUCP, Max-rescaled $R^2 = 0.32$, $p < 0.0001$; vesical neck movement, Max-rescaled $R^2 = 0.21$, $p < 0.0005$; MUCP plus vesical neck movement, Max-rescaled $R^2 = 0.41$, $p < 0.0001$.

Obstetrical Variables

Obstetrical variables for the two groups are listed in Table 5 for descriptive purposes. At this sample size none of these differences were statistically significant.

DISCUSSION

This study provides new data relevant to the 2.4-fold increase in the risk of developing stress incontinence after vaginal birth. (6) Contrary to clinical opinion that suggests that urethral mobility is the primary factor in stress incontinence, we found that maximal urethral closure pressure was the variable most strongly associated with *de novo* stress incontinence in primiparas. The findings of this study, which includes multiple assessments of urethral mobility in continent and incontinent women, suggest that urethral mobility is not the dominant factor affecting continence. Both parameters (urethral mobility and sphincter function) are important and must be considered together. These findings persist after controlling for BMI which also is associated with stress incontinence. We used multiple assessments of urethral mobility in an attempt to best capture this parameter. Differences between ultrasound and Q-tip measures probably relate to differences in position (standing versus supine) that impose different loads on the pelvic floor and maneuver (cough versus Valsalva) that differ in speed of pressure development or muscle activity. These results clinically validate earlier theoretical work that could only describe, in mathematical terms, the interaction between resting urethral closure function and the dynamic events occurring during a cough that are related to urethral mobility (27). Recognizing this, the fact that only 37% of incontinence is predicted by these two parameters means that there is more work to be done to fully understand the mechanism of stress incontinence.

The clinical importance of these findings lies in their ability to direct us in seeking rational ways to decrease pelvic floor injury during vaginal birth. The recent State-of-the-Science Conference on Cesarean Section on Maternal Request (6) has highlighted the need to identify the relationship between birth and pelvic floor disorders so that targeted strategies can prevent injury or hasten recovery. In the absence of reliable data about which structures are injured, or what obstetrical factors are associated with these injuries, liberalized elective cesarean section has been advocated by some. But, since approximately 1 in 10 women will develop pelvic floor dysfunction, 9 women would be subjected to the risks of the preventive intervention (cesarean birth) for every one woman whose future pelvic floor dysfunction would be prevented. (28) This research, by identifying impairments in urethral function and mobility, can lead to further studies designed to evaluate the effect of delivery mode and obstetrical factors on urethral closure pressure and urethral mobility. Eventually we hope to identify risk factors that may direct targeted injury prevention strategies.

This study has demonstrated that there are substantial differences in urethral sphincter function between primiparous incontinent and primiparous continent groups, notably decreased closure pressure, shorter striated muscle length and shorter overall urethral length in the incontinent group. While this is consistent with earlier longitudinal studies showing that urethral pressure and functional length decrease following vaginal delivery (29), it further extends these observations by comparing groups of both stress continent and incontinent women. Our findings add to the growing body of knowledge that women with stress incontinence have decreased urethral length (30) and urethral impairment. (15) Neither urethral closure pressure nor urethral length differed between nulliparous and primiparous continent women, indicating that these factors are specific to primiparous women *with* stress incontinence, but do not necessarily occur in all primiparous women. These findings highlight the importance of urethral sphincter function in the continence mechanism.

The occurrence of inadequate urethral sphincter closure and altered vesical neck mobility does not necessarily mean these impairments arose entirely following vaginal birth or that they are solely responsible for the occurrence of stress incontinence. Birth induces changes in urethral mobility and levator function that do not result in incontinence, as evidenced by the comparison of nulliparous and primiparous continent women. This study reveals wide variation in urethral function and mobility even among nulliparous women and specifically demonstrates (Figure 1) that a number of nulliparous women have either poor urethral function or altered mobility in the absence of vaginal birth. These women may represent a vulnerable population who might develop stress incontinence with relatively mild birth-induced damage; other women with better functional sphincter capacity who deliver vaginally may not experience a sufficient loss in this capacity so as to reach their threshold for incontinence. To move forward in answering these complex causal questions, longitudinal data will be needed to investigate the degree to which birth changes these parameters and identify which changes and combinations of changes are specific to stress incontinence. This will require a large prospective longitudinal study that contains sufficient numbers of women that can be expected to develop persistent clinically significant incontinence for multivariable analysis.

In an earlier study from this population, we reported on the occurrence of levator ani muscle defects in 32 of the 160 primiparous women.(26) Women with these defects had evidence of difficult deliveries with an odds ratio for forceps delivery of 14.7 and for anal sphincter rupture of 8.1 and a 78 minute longer second stage of labor.(25) We have also recently seen that these levator defects are more common in women with prolapse than normal controls.(31) Fifty-five percent of women with prolapse had major defects compared with 16% of women with normal support (adjusted odds ratio of 7.3). The lack of differences in obstetrical variables at this same sample size between women with and without stress incontinence suggests that obstetrical variables and levator injury may be more strongly associated with prolapse than with stress incontinence. Further research using methodologies designed to answer these questions will be needed to confirm these suspicions.

There are significant challenges to studying pelvic floor function in incontinent women. This study represents a specific point in time (nine to twelve months after delivery) in women after a single birth. The effects of subsequent delivery and aging also need to be studied. Although examiners of the ultrasounds and MRIs were blinded to subject status, incontinence is present during examination and evidence of prior vaginal delivery is visible at the time of evaluation so it is not possible to blind the examiners to continence status. Furthermore, currently available evaluation tools are not perfect. Measurement of urethral and pelvic floor function is influenced by limitations in equipment currently available. Continuing research into improved methods is ongoing and future studies may shed new light in these areas. Some women did not complete the MRI scan. This was less common in the control women because they received compensation for scan completion while the women with stress incontinence did not. This decision was made because the stress incontinent women received the benefit of a free urodynamic evaluation that benefited them personally. Poor layer definition in urethral images, especially in scans of marginal quality, precluded measurements more often in the stress incontinent group than the controls. It is possible that this is caused by such significant muscle loss that the band is no longer visible as a well-defined structure. Our data may have been influenced by these factors and are, therefore, a conservative estimate of reduced muscle mass. Despite these shortcomings, we believe that the findings do represent real biological phenomena and that our findings are biologically plausible.

We have demonstrated maximal urethral closure pressure to be the factor most strongly associated with development of *de novo* stress urinary incontinence post-vaginal delivery, as well as the importance of the combined effects of decreased urethral closure pressure and altered urethral mobility. The identification of these specific physiological parameters will

guide our future attempts to study the pathophysiology of stress urinary incontinence. We hope that this work will eventually lead toward rational strategies to reduce the risk of pelvic floor dysfunction.

Précis

Low urethral closure pressure is the factor most strongly associated with *de novo* stress urinary incontinence followed by altered vesical neck mobility.

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Figure 1. Scatterplot showing the relationship between Vesical Neck Movement with Cough (in mm of movement), on the X-axis, and Maximal Urethral Closure Pressure (in cm H₂O), on the Y-axis, among Nulliparous Continent (▲), Primiparous Incontinent (●), and Primiparous Continent (○) subjects. Note that when either axis is evaluated alone, separation of the incontinent women is not as clear as when both axes are displayed. Also note the substantial overlap in values between the stress continent and stress incontinent women.

Table 1

Subject Demographics expressed as mean values (standard deviation)

	Nulliparous Continent (NC) (n=80)	Primiparous Continent (PC) (n=80)	Primiparous Incontinent (PI) (n=80)	"Birth" NC v PC p value	"SUT" PC v PI p value
Age (years)	29.2 (± 5.5)	29.9 (± 4.5)	30.0 (± 5.7)	0.40	0.94
Height (m)	1.66 (± 0.07)	1.66 (± 0.07)	1.66 (± 0.07)	0.79	0.92
Weight (kg)	66.9 (± 13.3)	63.9 (± 11.4)	71.8 (± 16.8)	0.16	0.001
BMI (kg/m^2)	24.4 (± 4.4)	23.3 (± 3.9)	26.2 (± 5.6)	0.16	<0.001
Race				.59	.47
Euro-American	91.2%	88.8%	90.0%		
African-American	3.8%	2.5%	5.0%		
Other	5.0%	8.7%	5.0%		

Table 2

Urethral sphincter mechanism structure and function expressed as mean values (standard deviation)

	Nulliparous Continent (NC) (n=80)	Primiparous Continent (PC) (n=80)	Primiparous Incontinent (PI) (n=80)	"Birth," NC vs PC p value (d)	"SUI," PC vs PI p value (d) *
Urethral Closure Pressure					
Max. urethral closure pressure (cm H ₂ O)	90.3 (±25.0) n=77	83.9 (±21.0) n=72	62.9 (±25.2) n=61	0.091	.000 (0.91)
MRI of Urethral Sphincter					
Length of striated urethral sphincter (mm)	9.8 (±3.8) n=77	10.2 (±3.9) n=72	8.4 (±4.6) n=61	0.555	0.011 (0.42)
Length of urethra (mm)	29.4 (±3.3)	30.2 (±3.7)	28.7 (±5.7)	0.226	0.046 (0.31)
Maximal diameter of urethra (mm)	14.5 (±1.7)	14.1 (±1.67)	13.6 (±1.9)	0.360	0.109
Maximal thickness of striated urethral sphincter (mm)	1.9 (±0.6)	1.9 (±0.5)	1.7 (±0.6)	0.360	0.124

* Cohen's d listed when p < 0.05

Table 3

Vesical neck mobility and structure expressed as mean values (standard deviation) showing comparisons between changes associated with birth; nulliparous controls (NC) versus primiparous continent (PC), and those associated with stress incontinence; primiparous continent (PC) versus primiparous incontinent (PI)

	Nulliparous Continent (NC) (n=80)	Primiparous Continent (PC) (n=80)	Primiparous Incontinent (PI) (n=80)	"Birth" NC vs PC p value (d)	*"SUR" PC vs PI p value (d)
Urethral Mobility: Supine Posture (Q-tip)					
Rest (degrees)	-3.0 (±8.9)	-0.2 (±9.5)	5.4 (±13.3)	0.105	0.001 (0.48)
Valsalva (degrees)	27.2 (±17.8)	39.1 (±17.5)	51.0 (±17.3)	<0.001 (0.67)	<0.001 (0.68)
Kegel (degrees)	-16.1 (±12.1)	-14.6 (±13.0)	-8.2 (±17.2)	0.509	0.006 (0.42)
Standing Posture (ultrasound)	n=57	n=52	n=60		
Cough (mm)	9.9 (±5.0)	10.9 (±6.2)	15.6 (±6.2)	0.322	<0.001 (0.76)
Valsalva (mm)	13.6 (±7.1)	15.3 (±7.3)	16.2 (±7.0)	0.200	0.458
Kegel (mm)	5.9 (±5.8)	5.7 (±4.9)	4.8 (±6.4)	0.831	0.376
Levator ani muscle function (Vaginal closure force)					
Rest (Newtons)	2.7 (±1.17)	1.8 (±0.89)	2.0 (±1.1)	<0.001 (0.87)	0.161
MVC above resting value (Newtons)	3.7 (±2.27)	2.2 (±1.62)	2.0 (±1.36)	<0.001 (0.76)	0.378
Levator Ani Muscle Anatomy	n=79	n=75	n=70		
LA defect (% affected)	0%	12.0%	28.6%	<0.001	0.023

* Cohen's d listed when $p < 0.05$

**

Sample size for ultrasound measures smaller as mentioned in methods section

As previously reported (25)

Pelvic organ support evaluated with the Baden Walker reporting the percent of each group that had prolapse at or below the level of the hymenal ring ($\geq$ Baden Walker stage 2)

	Nulliparous Continent (NC) (n=80)	Primiparous Continent (PC) (n=80)	Primiparous Incontinent (PI) (n=80)	"Birth," NC vs PC p value	"SUI," PC vs PI p value
Urethrovesical	1.3%	17.7%	37.2%	<0.001	0.006
Bladder	0.0%	11.4%	26.9%	<0.001	0.012
Cervix	0.0%	0.0%	1.3%	NA	0.234
Upper Posterior	1.3%	7.7%	9.0%	0.039	0.772
Lower Posterior	1.3%	10.3%	11.5%	0.010	0.797
Any Site	2.5%	21.5%	47.4%	<0.001	0.001

Table 5

Obstetrical Variables expressed as mean (standard deviation) or percentage.

	Primiparous Continent (PC) n=80	Primiparous Incontinent (PI) n=80	PC vs PI
Gestational Age (weeks)	39.0 (± 1.5)	39.4 (± 1.4)	0.08
Birth Weight (kg)	3.42 (± 0.54)	3.53 (± 0.49)	0.14
Head Circumference (cm)	35.0 (± 1.6)	35.1 (± 1.3)	0.47
Length of second stage (minutes)	100.7 (± 76.0)	111.5 (± 91.0)	0.40
Oxytocin Use	60.0%	46.3%	0.08
Episiotomy	39.4%	40.0%	0.87
Anal Sphincter Laceration (includes partial 3 rd degree)	22.5%	27.5%	0.46
Epidural Anesthesia	63.8%	72.5%	0.23
Operative delivery	13.8%	23.8%	0.10
Vacuum	3.8%	11.3%	0.06
Forceps	10%	12.5%	0.62