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Stress Urinary Incontinence: Relative Importance of Urethral Support and Urethral Closure Pressure

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Abstract

Purpose—Treatment strategies for stress incontinence are based on the concept that urethral mobility is the predominant causal factor with sphincter function a secondary contributor. The relative importance of these two factors has not been assessed in properly controlled studies.

Materials and Methods—The Research On Stress incontinence Etiology (ROSE) project is a case-control study that compared 103 women with stress incontinence and 108 asymptomatic controls in groups matched for age, race, parity and hysterectomy. Urethral closure pressure, urethral and pelvic organ support, levator ani muscle function, and intravesical pressure were measured and analyzed using logistic regression and multivariable modeling.

Results—Mean maximal urethral closure pressure was 42% lower in cases (40.8 ± 17.1 SD vs. 70.2 ± 22.4 cm H₂O) with an effect size (d) of 1.47. Lesser effect sizes were seen for support parameters: resting urethral axis (d=0.41) and urethro-vaginal support (d=0.50). Other pelvic floor parameters including genital hiatus size (d=0.60) and urethral axis during muscle contraction (d=0.58) differed but levator strength and levator defect status did not. Maximum cough pressure, an assessment of the mechanical stress on the continence mechanism, was also different (d=0.43). After adjusting for BMI, maximal urethral closure pressure alone correctly classified 50% of cases. Adding the best predictors for urethrovaginal support and cough strength to the model added 11% of predictive ability.

Conclusion—The finding that maximal urethral closure pressure, not urethral support, is the factor most strongly associated with stress incontinence implies that improving urethral function may have therapeutic promise.

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Keywords

stress urinary incontinence; urethral closure pressure; urethral support; pelvic floor disorders; female

INTRODUCTION

Urinary incontinence is the most prevalent pelvic floor disorder¹ and responsible for silent suffering and social ostracism. Treatment costs reach 16 billion dollars annually.² Stress urinary incontinence is the most common type of urinary incontinence affecting 86% of incontinent women either alone (50%) or in conjunction with urge incontinence (36%).³

Despite the prevalent nature of this troubling condition, important gaps in our understanding of causal mechanisms limit development of new treatments and prevention strategies. Most current interventions focus on the surgical improvement of urethral support as the primary therapeutic target. Less attention has been focused on understanding factors affecting urethral closure pressure and treatment strategies aimed at this therapeutic target. Although surgical improvement of urethral support is clearly effective, careful outcome analysis reveals that 20% of women treated in this way still have a positive pad test 2 years after surgery and still complain of stress incontinence.⁴

The hypothesis that urethral support is the predominant causal factor in stress incontinence has not been properly tested. Studies assessing both urethral support and sphincter function have been inconclusive in part because control subjects were mainly *symptomatic incontinent* women referred for urodynamic evaluation that did not demonstrate stress incontinence during testing.^{5–8}

The ROSE Study (Research On Stress Incontinence Etiology) sought to compare measures of each element of the stress continence mechanism in matched cases and asymptomatic controls.

MATERIALS AND METHODS

The study used a case-control design with group matching. Between February 2003 and October 2006, 103 women with daily stress urinary incontinence and 108 asymptomatic continent controls were recruited and gave consent to participate in a matched cohort IRB-approved study. Continent controls were recruited to match cases based on factors associated with stress incontinence: age, race, parity and hysterectomy status. Subjects were recruited through university-based gynecology and urology clinics and local advertisements. A sample size of 100 in each group was chosen to have an 80% power to detect effect sizes of 0.35 and greater.

To be included, cases had to report daily incontinence, have at least two episodes of stress incontinence on a 3-day voiding diary and demonstrate stress incontinence during clinical examination. Controls had to report fewer than 6 episodes of incontinence in the last 12 months, have no leakage on 3-day voiding diary and demonstrate a negative full bladder stress test. Women who had undergone hysterectomy were eligible if it was not done for prolapse and occurred at least one year before enrollment. Cases and controls were excluded for previous surgery for pelvic floor disorders or prolapse ≥ 1 centimeter below the hymen to avoid the confounding effects of prolapse on stress incontinence. Women who reported symptoms of urge incontinence were allowed to be cases if stress component was the dominant incontinence symptom. Frequency and severity of incontinence, as well as distress and pad use, were obtained by questionnaire. Women were excluded from either group if they were currently

being treated for cancer, chronically used steroids, were HIV positive, had sickle cell disease or neurological conditions, uncontrolled diabetes, stroke, or Alzheimer's disease.

Clinical examinations were performed with women in a semi-recumbent position in a urodynamics chair at a 45° angle. Assessment of vaginal and uterine support was conducted using the Pelvic Organ Prolapse Quantification System (POP-Q); a technique that assesses the downward displacement of specific points along the vagina and cervix at maximal Valsalva.⁹ Urethral axis inclination measurements were made from the horizontal with a cotton-tipped swab ("Q-tip").⁸

Urethral sphincter function was assessed with urethral profilometry. Three serial urethral pressure profile measurements were taken using an 8 Fr. Gaeltec® dual-microtip urodynamics catheter (Medical Measurements Incorporated, Hackensack N.J.) with the transducer laterally oriented and averaged. The bladder was filled to a volume of 300 mL. Cough and Valsalva leak point pressures were determined. A positive full bladder stress test was defined as urine leakage with cough or Valsalva seen during examination. Levator ani muscle function was assessed with an instrumented vaginal speculum specially designed to measure vaginal closure force both at rest and during maximum voluntary contraction.¹⁰

Subjects underwent multiplanar magnetic resonance imaging using a proton density technique (echo time: 15 ms, repetition time: 4000 ms) in the supine position. Scans were performed on a 1.5 Tesla superconducting magnet (Signa; General Electric Medical Systems, Milwaukee, WI). Slice thickness was 4 mm with a gap of 1 mm, yielding 5 mm image spacing. A 160 × 160 mm field of view and 256 × 256 imaging matrix were used. Levator muscle defects seen in the levator ani muscle's pubovisceral region were graded independently on MR scans by two examiners blinded to subject status using a system previously described for evaluating birth-associated damage and used in our study of pelvic organ prolapse.¹¹

Group comparisons for continuous measures were made using two-sample t-tests; group comparisons for binomial distributions were made using Fisher exact tests. Effect size (d) was calculated for continuous variables showing statistically significant differences. Logistic regression models were used to study the extent to which individual variables or combinations of variables predicted stress incontinence. The most predictive variable from each domain was selected for inclusion in the model based on the bivariate comparisons between cases and controls. These domains included the two major aspects of the continence mechanism; urethral sphincter function and urethral support as well as a measure relating to increased demands on the continence mechanism (intravesical pressure with maximum cough). The "Max-rescaled R-square" statistic (SAS Version 8, SAS Institute, Inc., Cary, NC, 1999) was calculated to characterize the explanatory power of the different statistical models. Computed in the context of general linear models such as logistic regression, the value of its coefficient ranges from 0 to 1, allowing the predictive ability of models using parameters of different units to be compared.

RESULTS

Clinical characteristics of the study population showed cohorts to be successfully matched according to age, vaginal parity, race and hysterectomy status (Table 1). Current menstrual status and hormonal therapy use were similar between cases and controls. Overall, this was a healthy population with 81.7% of women self-reporting "Excellent or Very Good" health. Medical conditions self-reported by subjects included hypertension (19.5%), diabetes mellitus (2.4%), lung disease (17%), heart disease (4.9%), arthritis (39%), and neurological disease (2.4%) with no differences between the groups.

The characteristics of women's incontinence occurrence and severity are as follows: Among stress incontinent women, the mean age at which they reported the onset of urinary incontinence was 38.4 ± 10.0 (SD) years of age. The mean number of self-reported urine loss episodes per month was 79.9 ± 75.9 . Protective pads were used daily by 81.1% of incontinent women (46.8%-minipads; 16.0%-regular pad, and 37.2%: maxi pads) with an average of 2.7 ± 2.8 pad changes per day. In response to the question, "Do you receive very little warning and suddenly find you are losing urine or are about to lose urine beyond control", 27.4% answered "often", 30.4% "sometimes", 19.6% "rarely", and 22.6% "never". In response to the question, "How much does losing urine interfere with your everyday life?" 74.5% of women responded "moderately" or "greatly". In women with stress incontinence, average cough leak point pressure was 127.8 ± 44.0 cm H₂O and Valsalva leak point was 92.3 ± 38.5 cm H₂O.

In considering continence mechanism parameters maximum urethral closure pressure (cm H₂O) was 42% lower in incontinent subjects than continent controls and had the largest effect size in differentiating stress incontinent women from continent controls (Table 2). Incontinent subjects had greater urethral axis of inclination at rest. Although there was a trend towards a greater urethral axis at maximal Valsalva among incontinent women, this did not reach statistical significance. The urethro-vaginal point (Aa) descended more in cases than controls, but apical support and posterior vaginal wall did not differ. The genital hiatus during Valsalva was larger in incontinent women. Cases and controls were similar in the occurrence of minor and major defects in the levator ani muscle's pubovisceral region, and there were no differences in vaginal closure force occurring during a maximal pelvic muscle contraction or at rest. Cases demonstrated greater elevation of the proximal urethra during a pelvic muscle contraction. Analysis of these variables was also carried out controlling for BMI. This analysis did not demonstrate any differences in statistical significance; therefore the unadjusted values are shown.

In considering factors that place increased demands on the continence mechanism, stress incontinent women generated higher intravesical pressures both during cough and Valsalva but resting intravesical pressure did not differ between cases and controls.

We used logistic regression to evaluate the role of the three mechanistic domains (urethral support, urethral function, and intravesical pressure) on continence (Table 3). In each of these domains, we chose the variable with the largest effect size for differentiating between cases and controls to include in the model and controlled for BMI. Maximal urethral closure pressure had the largest Max-rescaled R² followed by urethrovesical support and maximal cough strength. Models that combined two and all three continence parameters showed only modest improvements in predictive ability above that for maximal urethral closure pressure alone as assessed by the Max-rescaled R².

DISCUSSION

Of the three factors involved in the mechanism of stress continence, 1) urethral function, 2) urethrovaginal support, and 3) a measure relating to increased demands on the continence mechanism (increased vesical pressure during cough), we found that urethral function (maximum urethral closure pressure) was the parameter most strongly associated with stress urinary incontinence. In regression models, urethral closure pressure alone predicted half of the occurrence of stress incontinence when comparing these women to continent volunteers of similar age, race, parity, and history of prior hysterectomy. Variables concerning urethro-vaginal support had moderate effect sizes (0.41 to 0.58) and the most predictive variable; point Aa, explained approximately 16% of stress incontinence. Earlier observational studies have considered one or two of the three continence parameters but not all.^{7, 12} In addition, controls in those studies were women undergoing urodynamic testing because of clinical problems such

as urge incontinence, who had a negative stress test, or had different parity and age⁷; factors known to influence continence status compared with cases. There is clearly overlap in urethral function between continent and incontinent women. In addition there are instances such as intrinsic sphincter deficiency or a scarred urethra where maximal urethral closure pressure and the degree of incontinence do not correlate. However the large effect size difference between the groups indicates that MUCP plays an important role in stress incontinence.

The finding that maximal urethral closure pressure is the predominant factor associated with stress incontinence has important clinical implications. At present, the majority of current and newly developed treatments focus on affecting urethral support. This strategy has been effective, however one of five women treated with operations intended to affect urethral support still have symptoms of stress incontinence.^{4, 13} Research into the function and dysfunction of other sphincters (e.g. lower esophageal sphincter) has led to important and effective additions to our pharmacologic armamentarium and scientific investigation.

The current belief that urethral support is the primary factor in stress incontinence has been plausible due to the success of widely used operations for stress incontinence¹³ that improve urethral support yet do not typically change urethral closure pressure.¹⁴ Surgical success, however, does not determine cause as is demonstrated by the effectiveness of bariatric operations for obesity. The fact that making the stomach smaller with surgery successfully causes weight loss does not imply that obesity is caused by a large stomach. Of the various aspects of urethrovaginal support measured, several were associated with stress incontinence; however their effect sizes (0.41 to 0.58) were considerably smaller than those seen with urethral closure pressure (1.47). These studies are consistent with the findings of other investigators who have demonstrated decreased urethrovaginal support associated with stress incontinence.^{5–8, 15} It has been difficult to assess the relative contribution of urethral support measured in these investigations because they used incontinent women without stress incontinence as controls or did not control for differences in age.

It is still unknown why some women have a lower resting urethral pressure than others. Discovering the answer to this question could lead to development of novel treatment strategies and opportunities for prevention. In broad perspective, urethral closure pressure is determined by the action of striated muscle, smooth muscle, and turgidity of the mucosal-vascular core with each element contributing approximately 1/3 of maximal urethral closure pressure.¹⁶ Advancing age is associated with a 10% to 15% decline per decade.¹⁶ Striated muscle loss has been demonstrated with advancing age¹⁷ and could be a target for future study. Recent investigations are clarifying the types of cellular mechanisms and neural transmitters.^{18, 19} These newly defined mechanisms may provide novel targets for intervention development, perhaps including regenerative tissue techniques in this small and easily accessible organ.

We found that visible defects in the levator ani muscle's pubovisceral region occurred with similar frequency in cases and controls. This observation is in contrast to findings in an earlier study of women that developed new stress incontinence after their first vaginal birth where defects were seen twice as often in stress incontinent women.²⁰ We hypothesize that in younger women who have higher urethral closure pressures¹⁶ it takes significant damage to the support system to result in stress incontinence while in older women, such as those seen in this study, loss of sphincter function plays a greater role.

Several factors must be considered when interpreting the results of this study. Women were only included if they had daily stress incontinence; the findings may differ in women with lesser degrees of stress incontinence as found in population-based investigations. Our study population was constituted primarily of Caucasian women and similar data in more diverse populations are needed to assess the degree to which these findings can be generalized to other

groups. In performing our statistical modeling, we chose to evaluate standard measures in our three domains and did not include analysis of hiatus size and urethral axis during pelvic muscle contraction as it is unclear at present how these parameters fit into the conceptual framework for stress incontinence. In quantifying pelvic floor function, we acknowledge the limited ability of current techniques to quantify all aspects of this highly complex neuromuscular unit whose control during daily life is largely involuntary. Specifically, because of its invasive nature, the ability of a microtip transducer to accurately reflect urethral resistance to urine flow is limited. In addition it is also possible that problems such as neuropathy may affect several aspects of continence system function by, for example, affecting both urethral function and support factors, so these that should be kept in mind when interpreting our results.

If the finding that urethral function is the factor that best differentiates stress incontinent women from continent women is corroborated by others, future treatment paradigms may prioritize improving urethral function, or preventing its damage, rather than just focusing on urethral support. With the rapid progress being made in clarification of disease mechanisms through cellular and molecular techniques, and the therapeutic possibilities possible through regenerative medicine, this type of effort would seem worthwhile.

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Abbreviations

BMI

Body mass index

MUCP

Maximum urethral closure pressure

References

1. Lukacz ES, Lawrence JM, Contreras R, Nager CW, Lubner KM. Parity, mode of delivery, and pelvic floor disorders. *Obstet Gynecol* 2006;107:1253–60. [PubMed: 16738149]
2. Wilson L, Brown JS, Shin GP, Luc KO, Subak LL. Annual direct cost of urinary incontinence. *Obstet Gynecol* 2001;98:398–406. [PubMed: 11530119]
3. Hannestad YS, Rortveit G, Sandvik H, Hunskaar S. Norwegian EPINCONT study. Epidemiology of Incontinence in the County of Nord-Trøndelag. A community-based epidemiological survey of female urinary incontinence: the Norwegian EPINCONT study. Epidemiology of Incontinence in the County of Nord-Trøndelag. *J Clin Epidemiol* 2000;53:1150–7. [PubMed: 11106889]
4. Ward KL, Hilton P. UK and Ireland TVT Trial Group. A prospective multicenter randomized trial of tension-free vaginal tape and colposuspension for primary urodynamic stress incontinence: two-year follow-up. *Am J Obstet Gynecol* 2004;190:324–31. [PubMed: 14981369]
5. Bai SW, Kang JY, Rha KH, Lee MS, Kim JY, Park KH. Relationship of urodynamic parameters and obesity in women with stress urinary incontinence. *J Reprod Med* 2002;47:559–63. [PubMed: 12170533]
6. Dietz HP, Clarke B, Herbison P. Bladder neck mobility and urethral closure pressure as predictors of genuine stress incontinence. *Int Urogynecol J Pelvic Floor Dysfunct* 2002;13:289–93. [PubMed: 12355287]
7. Schick E, Dupont C, Bertrand PE, Jolivet-Tremblay M, Tessier J. Predictive value of maximum urethral closure pressure, urethral hypermobility and urethral incompetence in the diagnosis of clinically significant female genuine stress incontinence. *J Urol* 2004;171:1871–5. [PubMed: 15076296]

8. Tapp K, Connolly A, Visco AG. Evaluation of Aa point and cotton-tipped swab test as predictors of urodynamic stress incontinence. *Obstet Gynecol* 2005;105:115–9. [PubMed: 15625151]
9. Bump RC, Mattiasson A, Bo K, Brubaker LP, DeLancey JO, Klarskov P, et al. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. *Am J Obstet Gynecol* 1996;175:10–7. [PubMed: 8694033]
10. Morgan DM, Kaur G, Hsu Y, Fenner DE, Guire K, Miller JM, et al. Does vaginal closure force differ in the supine and standing positions? *Am J Obstet Gynecol* 2005;192:1722–8. [PubMed: 15902185]
11. DeLancey JO, Morgan DM, Fenner DE, Kearney R, Guire K, Miller JM, et al. Comparison of levator ani muscle defects and function in women with and without pelvic organ prolapse. *Obstet Gynecol* 2007;109:295–302. [PubMed: 17267827]
12. Hilton P, Stanton SL. Urethral pressure measurement by microtransducer: the results in symptom-free women and in those with genuine stress incontinence. *Br J Obstet Gynaecol* 1983;90:919–33. [PubMed: 6684949]
13. Lapitan MC, Cody DJ, Grant AM. Open retropubic colposuspension for urinary incontinence in women. *Cochrane Database Syst Rev* 2005;(3):CD002912. [PubMed: 16034879]
14. Koelle D, Windisch J, Doerfler D, Marth C, Kropshofer S. Effect of tension-free vaginal tape operation on urethral closure function. *Urology* 2006;67:524–8. [PubMed: 16527571] discussion 528–9
15. Minardi D, Piloni V, Amadi A, El Asmar Z, Milanese G, Muzzonigro G. Correlation between urodynamics and perineal ultrasound in female patients with urinary incontinence. *Neurourol Urodyn* 2007;26:176–82. [PubMed: 17016799]
16. Rud T. Urethral pressure profile in continent women from childhood to old age. *Acta Obstet Gynecol Scand* 1980;59:331–5. [PubMed: 7192471]
17. Perucchini D, DeLancey JO, Ashton-Miller JA, Galecki A, Schaer GN. Age effects on urethral striated muscle. II Anatomic location of muscle loss. *Am J Obstet Gynecol* 2002;186:356–60. [PubMed: 11904591]
18. Ho KM, Borja MC, Persson K, Brading AF, Andersson KE. Expression of nitric oxide synthase immunoreactivity in the human female intramural striated urethral sphincter. *J Urol* 2003;169:2407–11. [PubMed: 12771807]
19. Brading AF. Spontaneous activity of lower urinary tract smooth muscles: correlation between ion channels and tissue function. *J Physiol* 2006;570:13–22. [PubMed: 16210349]
20. DeLancey JOL, 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 support loss. *Obstet Gynecol* 2007;110:354–62. [PubMed: 17666611]

Table 1

Comparison of Characteristics between Stress Urinary Incontinent Women and Continent Volunteers.

	Stress Incontinent (N=103)	Continent (N=108)	<i>P</i>
* Age (in years)	47.7 ± 9.3	47.7 ± 11.4	.99
* Parity	2.3 ± 1.3	2.0 ± 1.1	.55
* Caucasian Race % (N)	94.1 (95)	95.3 (102)	.76
* Prior Hysterectomy % (N)	11.7 (12)	8.3 (9)	.49
Body mass index (kg/m ²)	30.4 ± 6.6	27.6 ± 5.6	.001
Current Menstrual Cycles % (N)	66.7 (68)	61.1 (66)	.47
Hormone Replacement Therapy Use % (N)	9.8 (10)	9.3 (10)	.89

Values expressed as mean ± standard deviation or % (N, number).

* Subjects were recruited to constitute groups who were similar for these variables.

Table 2
Comparison of Clinical Pelvic Floor Measures Between Stress Urinary Incontinent Women and Continent Volunteers. Data are reported as mean $\pm$ standard deviation or % (N).

	Stress Incontinent (N=103)	Continent (N=108)	P	Effect Size (d)
MEASURE OF URETHRAL FUNCTION				
Maximum Urethral Closure Pressure (cm H ₂ O)	40.8 $\pm$ 17.1	70.2 $\pm$ 22.4	<.0001	1.47
MEASURES OF URETHROVAGINAL SUPPORT & PELVIC FLOOR STATUS				
Urethral Axis (degrees from horizontal)				
Cotton-tipped swab-rest	-0.8 $\pm$ 11.8	-6.3 $\pm$ 15.1	.004	.41
Cotton-tipped swab-strain	29.5 $\pm$ 20.3	25.0 $\pm$ 19.2	.10	-
Pelvic Organ Prolapse (cm relative to the hymen with + denoting locations below)				
Point Aa *	-0.6 $\pm$ 0.8	-1.0 $\pm$ 0.8	<.0001	.50
Point C	-6.3 $\pm$ 1.7	-6.3 $\pm$ 1.4	.74	-
Point Ap *	-1.4 $\pm$ 1.0	-1.4 $\pm$ 1.0	.95	-
Genital hiatus	4.0 $\pm$ 1.0	3.4 $\pm$ 1.0	<.0001	.60
Perineal body	4.0 $\pm$ 1.4	3.8 $\pm$ 1.3	.147	-
Levator Function				
Vaginal Closure Force, Rest (Newtons)	4.5 $\pm$ 2.7	4.5 $\pm$ 1.2	.89	-
Vaginal Closure Force, Augmented (Newtons)	2.3 $\pm$ 1.7	2.8 $\pm$ 1.9	.10	-
Cotton-tipped swab axis-Muscle Contraction (degrees)	-11.6 $\pm$ 14.9	-21.0 $\pm$ 17.4	<.0001	.58
Levator Muscle Defects				
	(n=99)	(n=102)	.31	
No defects % (N)	60.8 (62)	63.0 (68)		-
Minor % (N)	26.5 (27)	19.4 (21)		-
Major % (N)	12.7 (13)	17.6 (19)		-
MEASURES RELATING TO INCREASED DEMANDS ON THE CONTINENCE SYSTEM				
Intravesical Pressure (cmH ₂ O)				
At rest	21.2 $\pm$ 5.9	19.6 $\pm$ 9.3	.15	-
With maximum cough	143.2 $\pm$ 43.4	126.4 $\pm$ 34.3	.002	.43
Body mass index (kg/m ²) **	30.4 $\pm$ 6.6	27.6 $\pm$ 5.6	.001	.46

* Because Aa and Ba and Ap and Bp were similar in these populations, only the values for Aa and Ap are reported.

** Repeated from Table 1 for reference in this table.

Table 3
One, two and three variable logistic regression analyses to predict stress incontinence controlling for BMI.

Model	Variable	Coefficient R	t-stat	Significance	Odds Ratio	Confidence Interval
ONE VARIABLE MODELS (Controlled for BMI)						
MUCP Model: Max-Rescaled $R^2=0.50$, Sensitivity=0.78, Specificity=0.77						
	MUCP	-.075	-7.0	<0.001	.93	.91-.95
	BMI	.101	3.3	.002	1.1	1.0-1.2
Point Aa Model: Max-Rescaled $R^2=0.16$, Sensitivity=0.54, Specificity=0.71						
	Aa	.688	3.8	.0001	2.0	1.4-2.8
	BMI	.075	3.4	.003	1.1	1.02-1.13
Maximal Cough: Max-Rescaled $R^2=0.01$, Sensitivity=0.42, Specificity=0.81						
	Maximal Cough	.009	2.3	.02	1.0	1.00-1.02
	BMI	.046	1.8	0.08	1.1	0.01-1.10
TWO-VARIABLE MODELS						
MUCP, Aa Model: Max-Rescaled $R^2=0.59$, Sensitivity=0.79, Specificity=0.81						
	MUCP	-.0859	-7.0	<0.0001	.9	0.9-0.94
	Aa	1.0637	4.4	<0.0001	3.0	1.8-4.64
	BMI	.1084	3.0	.003	1.1	1.04-1.19
MUCP, Maximal Cough: Max-Rescaled $R^2=0.53$, Sensitivity=0.76, Specificity=0.81						
	MUCP	-.0790	-7.1	<0.0001	.9	.9-.94
	Maximal Cough	.0143	2.8	.005	1.0	1.00-1.03
	BMI	.0543	1.5	.14	1.1	.98-1.14
THREE VARIABLE MODEL						
MUCP, AA, Max Cough: Max-Rescaled $R^2=0.61$, Sensitivity=0.83, Specificity=0.83						
	MUCP	-.0892	-7.0	<0.0001	.9	0.89-0.94
	Aa	1.0581	4.3	<0.0001	2.8	1.77-4.68
	Maximal Cough	.0141	2.6	.01	1.0	1.00-1.03
	BMI	.0627	1.6	.12	1.1	0.98-1.15

Maximal Urethral Closure Pressure (MUCP), Urethrovaginal Support (Aa), Maximal Cough Strength (Maximal Cough), controlled for Body Mass Index (BMI) alone and in combination.

** Max-rescaled R^2 indicating the models ability to predict stress incontinence status shown for each model along with sensitivity and specificity.

*** The R^2 statistic is the Maximum rescaled R-square produced by PROC LOGISTIC in SAS. The P-values test the null hypothesis that the coefficient associated with the predictor is equal to zero.