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SPHINCTERIC URINARY INCONTINENCE: RELATIONSHIP OF VESICAL LEAK POINT PRESSURE, URETHRAL MOBILITY AND SEVERITY OF INCONTINENCE

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ABSTRACT

Purpose: We examined the relationships among urethral hypermobility, intrinsic sphincter deficiency and incontinence in women.

Materials and Methods: A total of 65 consecutive women with stress urinary incontinence and 28 with lower urinary tract symptoms not associated with stress urinary incontinence were evaluated with videourodynamics, 24-hour voiding diaries and pad tests, vesical leak point pressure measurement and the cotton swab test.

Results: A total of 93 patients with a mean age ± SD of 63 ± 13 years were studied, including 65 who presented with stress urinary incontinence and 28 who presented with lower urinary tract symptoms without stress urinary incontinence. The incidence of urethral hypermobility was 32% in the stress urinary incontinence group and 36% in the lower urinary tract symptoms group ($p = 0.46$). When stress urinary incontinence cases were stratified according to a vesical leak point pressure of 0 to 60, 60 to 90 and greater than 90 cm. H_2O, urethral hypermobility was noted in 25%, 31% and 41%, respectively, a difference that was not statistically significant ($p = 0.6$). Overall incontinent patients with and without urethral hypermobility had the same median number of incontinence episodes (5, range 1 to 13 versus 7, range 1 to 15, $p = 0.39$) and median pad weight (39.5 range 1 to 693 gm. versus 33.5, range 1 to 751, $p = 0.19$). When patients with intrinsic sphincter deficiency, defined as vesical leak point pressure less than 60 cm. H_2O, were divided into those with and without urethral hypermobility, there were no differences in the mean number of incontinence episodes (9.4 ± 3 versus 6 ± 3.6, $p = 0.17$) or median pad weight (90 gm., range 10 to 348 versus 86, range 30 to 81, $p = 0.76$). The degree of change in the urethral angle did not correlate with vesical leak point pressure ($r = 0.16$, $p = 0.24$) or with pad weight ($r = -0.23$, $p = 0.1$).

Conclusions: Urethral hypermobility was equally common in this group of women with lower urinary tract symptoms and stress urinary incontinence. Intrinsic sphincteric deficiency and urethral hypermobility may coexist and they do not define discrete classes of patients with stress urinary incontinence. Urethral hypermobility did not appear to have an independent effect on the frequency or severity of incontinence. Patients with stress urinary incontinence can still be characterized by vesical leak point pressure and change in the urethral angle, although these variables do not always define discrete classes.

KEY WORDS: urethra, urinary incontinence, bladder, urodynamics

Stress urinary incontinence is thought to arise from 2 separate mechanisms, namely anatomical incontinence, in which urethral hypermobility results in incontinence, and intrinsic sphincter deficiency, in which a defective closure mechanism causes failure of the urethra to coapt during increased intravesical pressure.[1,2] The evidence that anatomical incontinence and intrinsic sphincteric deficiency are optimally treated with widely different surgical approaches[3–5] suggests that evaluating these conditions and their relationship is clinically important.

In studies of urethral sphincter function abdominal leak point pressure was defined as the urodynamic measure of the lowest abdominal pressure required to overcome urethral resistance, resulting in urinary incontinence.[6,7] The Standardization Committee of the International Continence Society recently defined abdominal leak point pressure as "the intravesical pressure at which urine leakage occurs due to increased abdominal pressure in the absence of a detrusor contraction."[8] For clarity we prefer the term vesical leak point pressure to describe the intravesical pressure at which leakage occurs.

Urethral hypermobility has been measured by various methods, including imaging and clinical evaluation. Ultrasonography[9] and fluoroscopy[10] have been used with little agreement on a standardized method. The cotton swab test remains the gold standard for determining urethral hypermobility because it is simple, inexpensive and equivalent to more advanced imaging techniques.[11] A urethral angle change of 30 degrees or greater indicates urethral hypermobility.[12]

McGuire et al observed that women with an abdominal leak point pressure of less than 60 cm. H_2O are likely to have intrinsic sphincteric deficiency, whereas 80% with abdominal leak point pressure between 60 and 90 cm. H_2O have stress urinary incontinence due to urethral hypermobility.[6] We evaluated whether urethral hypermobility in fact inversely

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correlates with intrinsic sphincteric deficiency, as measured by vesical leak point pressure. We also determined the degree to which urethral hypermobility and intrinsic sphincteric deficiency interact to cause incontinence.

PATIENTS AND METHODS

We evaluated 65 consecutive women with stress urinary incontinence (group 1) and 28 with lower urinary tract symptoms not associated with stress urinary incontinence (group 2). All patients provided a history and underwent physical examination, a validated questionnaire, diary, pad test and videourodynamic study. Study inclusion criteria included female sex, lower urinary tract symptoms and age greater than 18 years. Study exclusion criteria included urinary tract infection, malignancy and age less than 18 years. Patients were enrolled between July 1, 2001 and January 1, 2002.

Videourodynamics were performed with the patient seated using a 7Fr dual lumen vesical catheter and a 9Fr rectal balloon catheter at medium filling, and radiographic contrast medium (200 ml. iothalamate meglumine 60% mixed with 800 ml. water). Vesical leak point pressure was defined as minimum vesical pressure in the absence of involuntary detrusor contraction at which fluid was visualized emanating from the urethral meatus during coughing or the Valsalva maneuver. Vesical leak point pressure was assessed at a bladder volume of 200 ml. and at 100 ml. increments thereafter until incontinence occurred or bladder capacity was attained. When no leakage was observed, the vesical catheter was removed and abdominal leak point pressure was recorded as the minimum abdominal pressure at which fluid was visualized emanating from the urethral meatus during efforts to increase abdominal pressure, that is during coughing or straining. The diagnosis of stress urinary incontinence was based on history, physical examination, pad testing and vesical leak point pressure.

Each group underwent urethral angle estimation at rest and during coughing or the Valsalva maneuver using the cotton swab test and a protractor. The maximum urethral angle was recorded. Urethral hypermobility was defined as a change in the urethral angle of 30 degrees or more from baseline.[12]

Statistical analysis of the data were performed with commercially available software. We used ANOVA and Student's t test to compare differences in means in the groups. Pearson's correlation test was done to correlate leak point with urethral angle. Fisher's exact test was used to compare the incidence of urethral hypermobility in women with and without stress urinary incontinence. The Mann-Whitney test was used to analyze nonparametric variables.

RESULTS

A total of 65 women with a mean age of 63 years (range 32 to 84) who had stress urinary incontinence and 28 with a mean age of 64 years (range 35 to 86) without stress urinary incontinence were included in the study (table 1). Of the patients with stress urinary incontinence 23 had associated urgency symptoms. Those without stress urinary incontinence had generalized lower urinary tract symptoms, including frequency in 7, urgency in 19 and dysuria in 2.

Relationship of urethral hypermobility to vesical leak point pressure. A linear regression model showed no correlation of vesical leak point pressure with urethral hypermobility in women with stress urinary incontinence (see figure). Analysis of patients according to a vesical leak point pressure of 0 to 60, 60 to 90 or greater than 90 cm. H_2O revealed concurrent urethral hypermobility in 24%, 31% and 41%, respectively, a difference that was not statistically significant (p = 0.6, table 2).

Urethral hypermobility and stress incontinence. Neither the number of incontinence episodes nor pad weight was normally distributed. Patients with and without urethral hypermobility had no significant difference in the median number of incontinence episodes (5, range 1 to 13 versus 7 range 1 to 15, p = 0.39) or median pad weight (39.5 gm., range 1 to 693 versus 33.5, range 1 to 751, p = 0.19).

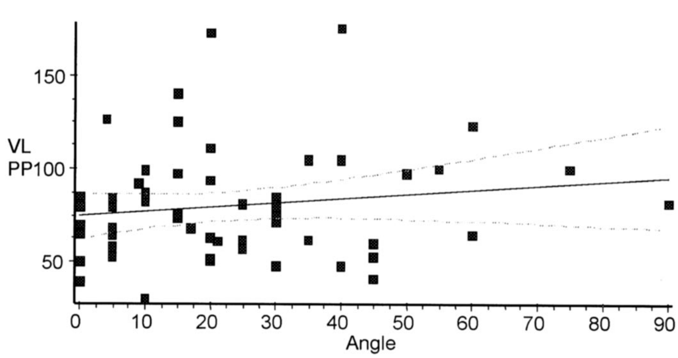
Intrinsic sphincteric deficiency with and without urethral hypermobility. When we compared women with a vesical leak point pressure of below 60 cm. H2O plus urethral hypermobility to those with a vesical leak point pressure of below 60 cm. H_2O without urethral hypermobility there was no significant difference in the mean number of incontinence episodes (9.4 $\pm$ 3 versus 6 $\pm$ 3.6, p = 0.17), or median pad weight (90 gm., range 10 to 348 versus 86, range 30 to 81, p = 0.76). Fisher's exact test showed no significant difference in the incidence of urethral hypermobility in groups 1 and 2 (table 3).

DISCUSSION

In 1968 Green characterized incontinence based on the anatomical relationship of the urethra to the bladder base.[13] In type I incontinence there was loss of the posterior urethrovesical angle. In type II inferior and rotational descent of the bladder base and urethra were also present. This system was able to predict which patients would respond well to simple anterior colporrhaphy, namely 90% and 50% with types I and II, respectively.[2,13] In contrast, patients with type II anatomy had a cure rate of as high as 90% after retropubic suspension.[14] In 1976 McGuire et al described type III stress urinary incontinence or intrinsic sphincteric deficiency.[1] In this type of incontinence the urethrovesical angle is not compromised but the bladder neck and proximal urethra fail to function as a competent sphincteric unit. A pubovaginal sling is effective in 95% of cases.[5] In 1988 Blaivas and Olsson defined types 0—stress incontinence not reproducible in the clinical setting and IIB—a urethra abnormally positioned at rest, such that incontinence ensues with little additional descent during stress.[2] In 1993 McGuire et al proposed that measuring the abdominal leak point pressure in women with stress urinary incontinence would suggest whether leakage was

TABLE 1. *Patient characteristics*

	Group 1	Group 2
No. pts.	65	28
No. chief complaint (%):		
Stress urinary incontinence	42 (65)	
Mixed urinary incontinence	23 (35)	
Urgency		19 (68)
Frequency		7 (25)
Dysuria		2 (0.7)
No. pad test (%)	56 (86)	16 (57)
No. voiding diary (%)	52 (80)	15 (54)



R = 0.17, r^2 = 0.029 and p = 0.2. *VL PP*, Valsalva leak point pressure.

TABLE 2. *Vesical leak point pressure and hypermobility*

Vesical Leak Point Pressure (cm. H_2O)	No. Urethral Hypermobility (%)		
	Yes	No	Totals
0–60	4 (25)	12 (75)	16 (100)
60–90	10 (31)	22 (69)	32 (100)
Greater than 90	7 (41)	10 (67)	17 (100)
Totals	21 (32)	44 (67)	65 (100)

p = 0.6.

TABLE 3. *Urethral mobility and incontinence*

	No. Group 1 (%)	No. Group 2 (%)	Total No.
Hypermobility	21 (32)	10 (36)	31, p = 0.46
No hypermobility	44 (68)	18 (64)	62
Totals	65	28	93

caused by intrinsic sphincteric deficiency (abdominal leak point pressure below 60 cm. H_2O) or urethral hypermobility.[6] Patients with abdominal leak point pressure between 60 and 90 cm. H_2O were considered to have mixed incontinence. Although this system is widely accepted and clinically applicable, it is sometimes erroneously interpreted to suggest that incontinence associated with high leak point pressure implies urethral hypermobility. Our study partially confirmed this relationship, that is high vesical leak point pressure is related to urethral hypermobility.

We noted that urethral hypermobility does not predict stress urinary incontinence. There was an equal incidence of urethral hypermobility in the lower urinary tract symptom and stress urinary incontinence groups, confirming the findings of Bergman et al, who observed urethral hypermobility in 50% of continent women and a well supported urethra in 10% to 40% of women with stress urinary incontinence.[12] In our incontinent patients with intrinsic sphincteric deficiency concomitant urethral hypermobility did not worsen the frequency or degree of incontinence.

Notably there are several differences in our study and that of McGuire et al.[6] McGuire et al used a 10Fr catheter, filled the bladder to 150 ml., measured abdominal leak point pressure and estimated it based on radiographically detected leakage. These methods are valid but compared with our methodology they would result in higher average leak point pressure. Our sample of patients had relatively severe incontinence with a mean of 5.5 episodes and a loss of 113 gm. urine daily, and there was a relatively low incidence of urethral hypermobility (33%). It is possible that in patients with less severe incontinence there may be a different association of leak point pressure with urethral hypermobility. Furthermore, McGuire et al estimated urethral angle radiographically and not with the cotton swab test. Neither our study nor that of McGuire et al addressed the potential impact of pelvic organ prolapse on the results.

Our findings in this investigation support the vast body of research that describes urinary incontinence as a multifactorial problem. We do not see cause to abandon tests such as those for leak point pressure or the urethral angle and our results are not an indictment of these individual tests. For example, the lack of a correlation of low vesical leak point pressure with pad weight does not indicate that leak point pressure is not a useful way to characterize incontinence. Clinically patients with leakage at lower pressure who may be expected to have more episodes of more severe incontinence may compensate by voiding more frequently or drinking less, which does not undermine the validity of assessing leak points. We cannot ignore the importance of evaluating urethral hypermobility because it has been shown to affect the surgical outcome, including that of bladder neck suspension, pubovaginal sling surgery, and collagen injection,[3–5, 15] although recent research also suggests that urethral hypermobility does not affect the outcome of collagen injection.[16, 17] The latter finding is consistent with our observation that urethral hypermobility does not exacerbate incontinence due to intrinsic sphincteric deficiency.

Further investigation is needed to explain why the cotton swab test can predict certain surgical outcomes and yet fails to correlate with the presence or severity of stress urinary incontinence. Urethral hypermobility may cause stress urinary incontinence only when there is unequal movement of the anterior and posterior urethral walls, when the urethra is pulled open by the movement of the posterior wall alone.[18, 19] Alternatively there may be several types of urethral descent with various degrees of clinical relevance that are not distinguished by available diagnostic tests. It is certain that only by continued research into various contributing factors may we achieve a better understanding of urinary incontinence and through this understanding develop more effective and durable treatments for it.

CONCLUSIONS

Urethral hypermobility and intrinsic sphincteric deficiency often coexist and they are independent variables. Urethral hypermobility is not predictive of stress urinary incontinence and it appears not to worsen stress incontinence, as measured by voiding diaries and the pad test in patients with concurrent intrinsic sphincteric deficiency. Measurements of urethral hypermobility are clinically useful when planning the surgical approach. Currently we recommend that stress urinary incontinence should be characterized rather than classified by 2 parameters, namely leak point pressure and change in the urethral angle.

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