DIFFERENCES IN INTRAVAGINAL PRESSURE GENERATED DURING VOLUNTARY PELVIC FLOOR MUSCLE CONTRACTIONS BETWEEN CONTINENT AND STRESS INCONTINENT WOMEN

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INTRODUCTION

The purpose of this study was to compare the patterns of abdominal and pelvic floor muscle (PFM) activity associated with intravaginal pressure generation in stress urinary incontinent (SUI) and continent women. This study builds upon our previous work in which vaginal pressure generation was described in continent women.[1-3]

Co-activation of the abdominal muscles with the PFM appears to contribute to the generation of intravaginal, and therefore intraurethral, pressure in urinary continent women.[4-6] In continent women we found that the initial intravaginal pressure rise generated with a voluntary PFM contraction resulted from activity of the PFM and the abdominal muscles. The middle phase of the intravaginal pressure rise where the rise is generated primarily by the PFM. The final phase of increase in intravaginal pressure is associated primarily with abdominal muscle contraction.[1] We found very similar patterns in a follow up study in both continent and stress incontinent women.[3] These two studies support DeLancey's Hammock Hypothesis which suggest that the primary role of the PFM is to provide a firm surface against which the urethra can be compressed by increased intra-abdominal pressure.[7] Understanding the nature of any abdominal and PFM synergies is particularly important given that increases in intra-abdominal pressure associated with abdominal muscle contraction appear on one hand to be responsible for, and on the other hand to prevent, the urine leakage seen in SUI.[8]

In a recent study that looked at PFM and abdominal muscle responses to unexpected trunk loading, Smith et al. found that women with more severe SUI displayed different muscle activation levels than either continent women or women with mild SUI.[9] As we had found no difference in muscle activation patterns between continent and stress incontinent women [3], we have analyzed our previously recorded data stratified by groups based leakage severity. The objectives of this study were: (1) to model the generation of intravaginal pressure and (2) to compare the activation patterns among women with no incontinence, mild incontinence and moderate incontinence.

MATERIALS AND METHODS

Ethical approval for this study was received from the Queen's University Human Research Ethics Board and all of the participants provided written consent prior to participating. Volunteers were recruited through newspaper advertisements in a Kingston newspaper. Women who were pregnant, had given birth in the previous six months, had neurological or rheumatological disorders, had undergone gynaecological surgery involving the pelvic floor, were taking medications that affect the lower urinary tract or who reported symptoms that were not consistent with SUI were excluded. The volunteers were then screened by a nurse for pelvic organ prolapse and the ability to correctly perform a PFM contraction. Women with prolapse past the hymenal ring or who were not able to contract the PFMs were excluded. Women with symptoms consistent with SUI were referred for urodynamic testing; the continent volunteers did not undergo urodynamic testing. Any women whose urodynamic test results were not consistent with SUI were excluded from the study.

The women with SUI were classified as mild, moderate or severe based on the frequency and severity of SUI episodes as recorded in a three-day bladder diary. Mild SUI was defined as two or fewer leakage episodes per day that were provoked by relatively intense physical activity, such as running or jumping. Moderate SUI was defined as between two and five leakage episodes per day that were provoked by sneezing or coughing. Severe SUI was defined as a more than five leakage episodes per day that were provoked by sneezing, coughing or low levels of physical activity, such as moving from sitting to standing.

Pelvic floor muscle electromyographic (EMG) and vaginal pressure data were acquired using a custom-modified Femiscan™ vaginal probe. (See Figure 1.) The probe had pairs of bipolar stainless steel electrodes mounted bilaterally, and was modified by mounting an air-filled pressure chamber through a hole in its posterior surface. The pressure chamber was coupled to a pressure transducer and did not require amplification (range –5 to +5V, resolution 0.04V= 1cmH_2O). Surface EMG data were recorded from three abdominal muscles: rectus abdominis (RA), external obliques (EO) and internal obliques (IO) using

Meditrace™ 133 surface Ag-AgCl electrodes. EMG data were amplified using Bortec AMT-8 amplifiers, and both EMG and pressure data were acquired at 1kHz using a 16-bit Analog to Digital Converter and Labview v. 6.1.

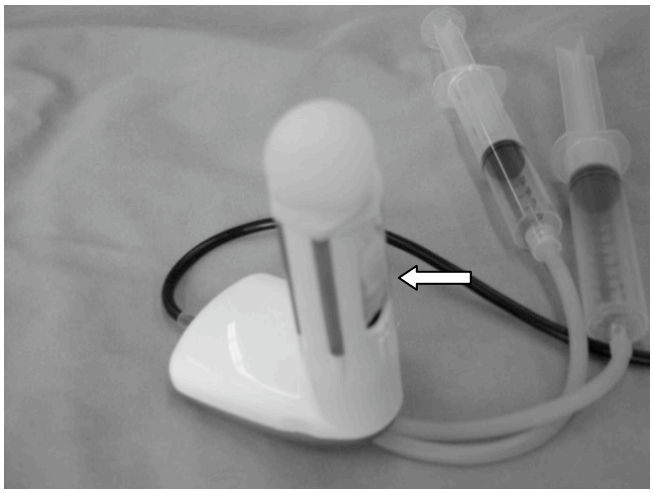

Figure 1. Modified Femiscan™ vaginal probe. The left pair of electrodes is visible in the figure. The arrow marks the location of the air-filled pressure chamber.

The volunteer was left in private to insert the probe and then the pressure chamber was inflated with 3cc of room air. Vaginal pressure and EMG data were acquired simultaneously at rest and during three maximum voluntary PFM contractions (PFM MVCs). Each volunteer was positioned in supine, with a pillow under her head and her thighs slightly abducted. The resting data were recorded first, while the volunteer were asked to relax all of her muscles. The volunteers were then instructed in the correct performance of a PFM contraction and were allowed to practice these contractions until the researchers were satisfied that the PFM contractions were being performed correctly through observation of the EMG and pressure data, and direct observation of the perineum for caudal movement. Then the volunteers performed three repetitions of a maximum voluntary PFM contraction. They were instructed to: “Pull up and in and squeeze around the probe.” Verbal encouragement was provided during each trial and at least two minutes rest was provided between trials.

All data were smoothed using a 20ms sliding window over which the root mean square (RMS) value was calculated across the contraction. The baseline RMS level, computed as the mean RMS of the resting data, was subtracted from all MVC values. Maximum amplitudes were determined as the highest 20ms RMS value minus the resting level achieved for each muscle and the intravaginal pressure during the PFM MVCs. In order to model the generation of intravaginal pressure, the data were normalized based on the maximum smoothed pressure or EMG amplitude achieved during each contraction. The normalized pressure vs. EMG curves were ensemble averaged, and the equations of these curves were computed for each muscle using Graph Pad Prism™ software. The fit of each equation was assured using a runs test (α=0.05).

The pressure versus EMG curves for each muscle were compared among the control and mild and moderate SUI groups using the initial and final percent EMG and the equations of the curves.

RESULTS

Twenty-four continent women and 24 women with mild SUI and four with moderate SUI participated. None of the volunteers was classified as having severe SUI. There were no differences in maximum EMG RMS generated during the PFM contractions among the groups; intravaginal pressure was not different between the continent women and the women with mild SUI, but it was lower in the women with moderate SUI. See Table 1.

Table 1. Maximum RMS Amplitudes during the Maximum Voluntary PFM Contractions, by Group. Mean (standard deviation), * indicates significant differences.

Site	Continent	Mild SUI	Moderate SUI
PFM (μV)	52 (41)	34 (34)	36 (31)
RA (μV)	10 (10)	11 (8)	15 (17)
EO (μV)	32 (25)	28 (15)	26 (28)
IO (μV)	25 (15)	22 (18)	19 (19)
Pressure (cmH_2O)	14 (14)	17 (12)	6 (4)*
			*P< 0.001

The PFM EMG vs. vaginal pressure curves were modeled using an exponential equation in each group:

$$\text{Pressure} = A*(1-e^{(-Kx)})$$

Where A is the percent maximum EMG when pressure is 100%, K is the rate constant and x is the pressure. See Figure 2 and Table 2 for the coefficients.

The abdominal EMG vs. vaginal pressure curves for the continent and mild SUI groups showed two-phase responses, where the abdominal muscles contributed significantly to the rise in vaginal pressure during the latter half of the contraction. See Figure 3 and Table 3 for the coefficients. The abdominal EMG vs. vaginal pressure curves for the moderate SUI group differed from the continent and mild SUI groups and were best described with linear regressions. They did not demonstrate the steeper rise in EMG activity in

the latter half of the contraction. In the moderate SUI group RA had an intercept of 66.13 (SE 1.40) and a slope of 0.1127 (SE 0.0240), for EO the intercept was 54.23 (SE 2.42) and the slope was 0.0756 (SE 0.0415) and for IO the intercept was 52.80 (SE 2.36) and the slope was 0.0596 (SE 0.0403).

Table 2. Coefficients for the exponential equations describing the PFM versus pressure curves. Values are given as the mean (standard error). Units for A are percent maximum EMG.

Group	A	K
Continent	69.64% (1.35)	0.0710 (0.0066)
Mild SUI	71.92% (1.25)	0.0891 (0.0086)
Moderate SUI	72.77% (2.11)	0.0833 (0.0129)

Table 3. Coefficients for the exponential equations describing the abdominal muscle versus pressure curves for the continent and mild SUI groups. Values are given as the mean (standard error). Units for A are percent maximum EMG.

Muscle, Group	A	K
RA Continent	66.83% (0.91)	0.5705 (0.2086)
RA Mild SUI	66.57% (0.93)	0.5083 (0.1557)
EO Continent	53.55% (0.87)	0.4169 (0.1123)
EO Mild SUI	53.29% (0.98)	0.3387 (0.0826)
IO Continent	54.43% (1.07)	0.1626 (0.0252)
IO Mild SUI	57.33% (1.17)	0.1614 (0.0259)

DISCUSSION

Although our sample of women with moderately severe SUI was small (n=4), the findings of this study suggest that women with moderate SUI use different motor control strategies when generating intravaginal pressure than do either continent women or women with mild SUI. The continent women and the women with mild SUI both demonstrated an increase in abdominal muscle activity during the latter half of vaginal pressure development whereas the women with moderate SUI did not. This is consistent with DeLancey's Hammock Hypothesis.[7] The women with moderate SUI might have learned to adapt their abdominal muscle activity to follow the PFM activity in order to avoid urine leakage. Further research needs to be undertaken to determine whether moderate to severe SUI has a different aetiology than mild SUI, or whether the severity of SUI depends upon the extent of the injury to the continence mechanism rather than the type of injury. Further research is indicated to determine if, since the abdominal muscle contributions differ between women with moderately severe SUI as compared to mild SUI, different rehabilitation treatment strategies are indicated for these women.

A major limitation of this study is that the women with SUI were stratified by the severity of leakage retrospectively, resulting in having a very small sample of women in the moderate SUI group. As the moderate group was very small, the pattern of abdominal muscle activity described may not be generalisable.

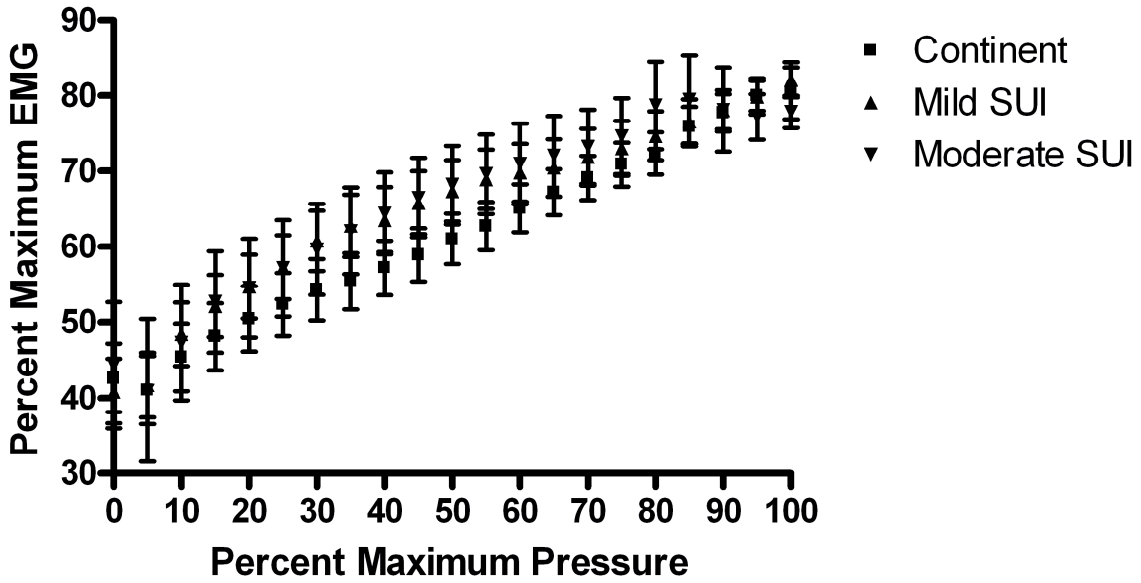

Figure 2. PFM EMG versus Pressure curves. All three groups demonstrate inverse exponential relationships.

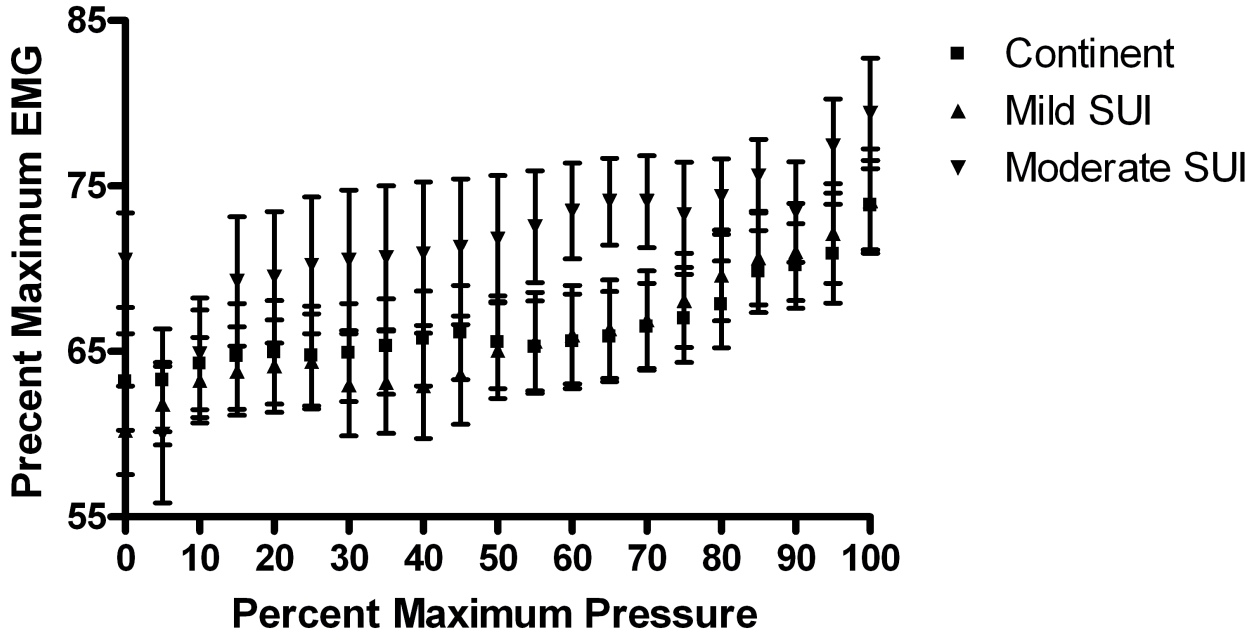

Figure 3. Rectus Abdominis EMG versus Pressure curves. Rectus abdominis is representative of the patterns found with all of the abdominal muscles tested. In the continent and mild stress incontinent women the initial rise in EMG amplitude was more gradual than the later rise where abdominal muscle contraction was associated with a late rise in intravaginal pressure. In the four women with moderate SUI the pattern was reversed.

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